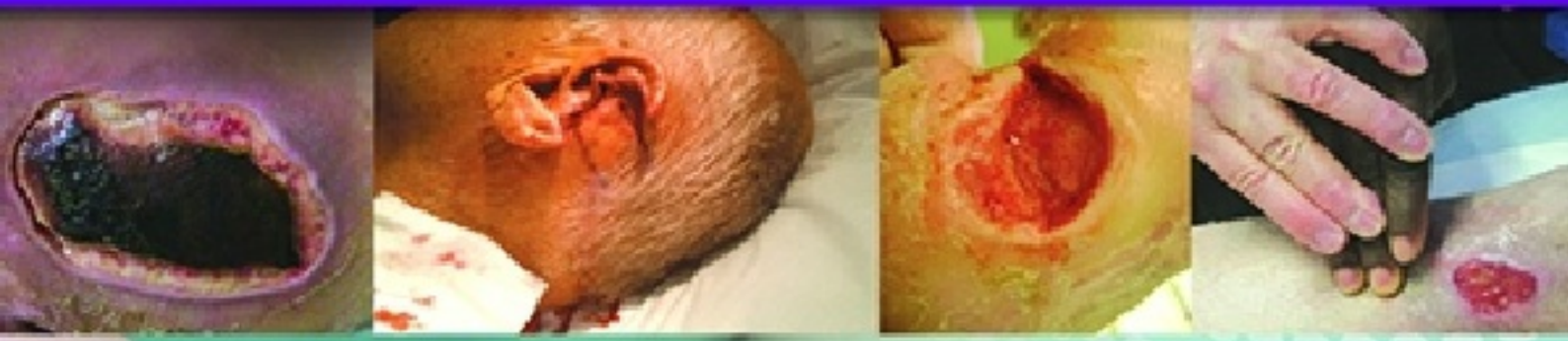
Wound Care Essentials

Practice Principles



Sharon Baranoski
Elizabeth A. Ayello

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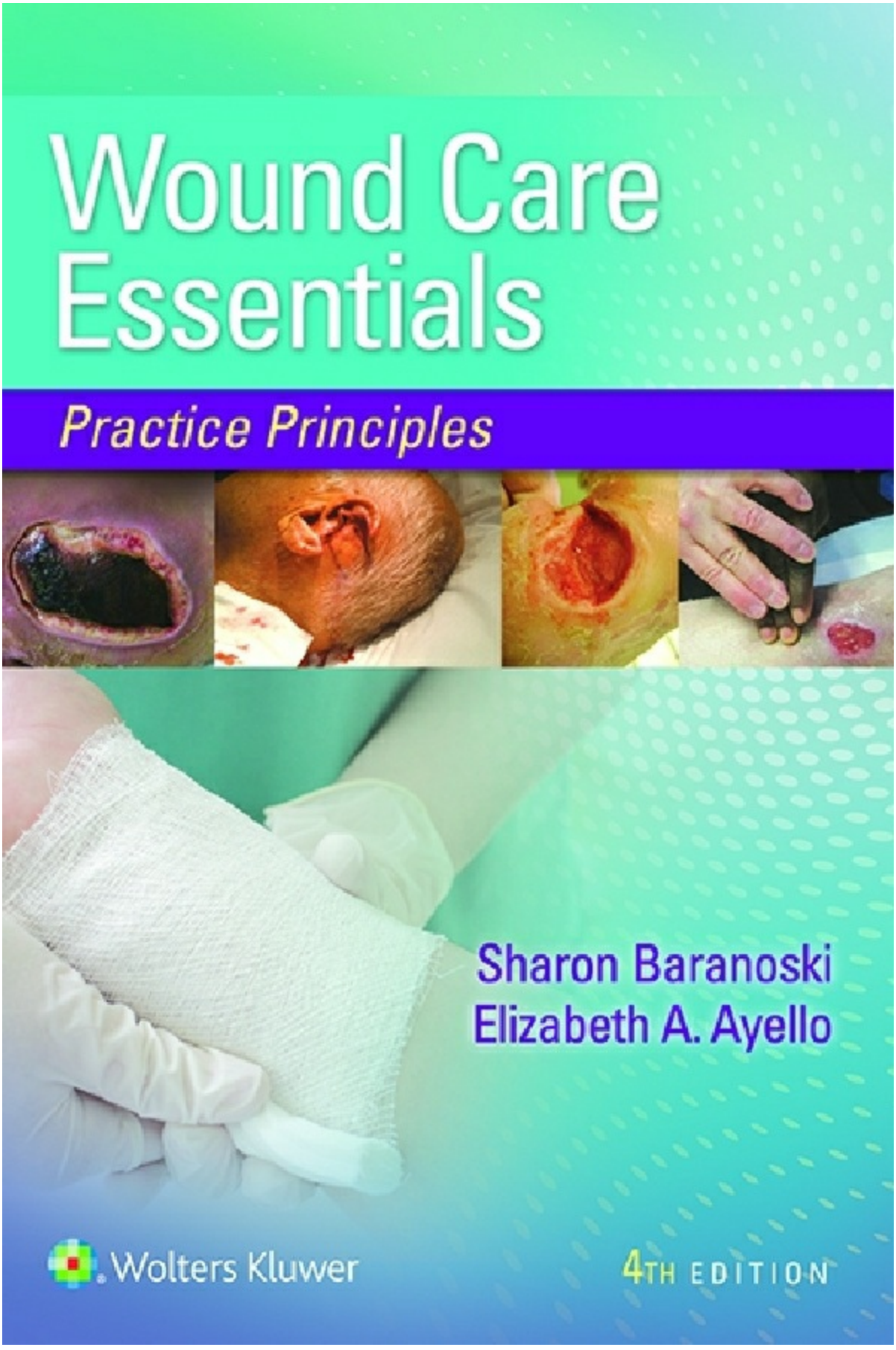
4TH EDITION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Practice Principles
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Practice Principles

Fourth Edition

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Dedication

This fourth edition is dedicated with love and gratitude to:

My husband—Jim "Bear" Baranoski—*For the support, encouragement, and devotion you have given me for half century. I couldn't love you more.*

The Baranoski offspring and their significant others—*Jim Jr.; Deborah and Mark Prosise; Jeffrey and Kari Baranoski; and JR (John Robert) and Carissa Baranoski, thank you for your love and laughter and for bringing joy and craziness to my life. I am so blessed to have wonderful children.—Love you all, Mom.*

The Baranoski grandchildren—*Maddie Gombosi, Morgan Prosise, Lexi, and Lanie Baranoski; Brek and Brooklyn Baranoski; Alia and Tyler (TJ) Baranoski; and any future ones to come. I love you all from the deepest part of my heart. You brighten my life and certainly make me laugh and very proud.—I love you much, Gramma.*

To all the nurses, physicians, healthcare practitioners, and healers *who have honestly acknowledged how much we don't know and how much we have to learn. Never stop wanting more.*

With Love, Sharon

"Family is everything."

That's what my parents, Phyllis and Tony, and brothers, Bob and Ron, taught me.

So thank you to my families—biological and professional—friends, and patients who have shared their experiences, supported, and nurtured me through this educational journey. There are too many of you to mention individually; it would take more than what could be contained on this page. Hopefully I have done a good job of expressing my appreciation and gratitude to you each and every day.

To:

Katie, who taught me the psychosocial meaning of healing

Sarah, who taught me the true meaning of healing

Wendy, who taught me the meaning of hope for healing and the promise of tomorrow

A. Scott, who taught me to never stop learning and who brings the art to balance my science. Thank you for being there, so I can dance like nobody is watching.

Love, E.A.A.

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Foreword

It is my pleasure to write the foreword for the fourth edition of *Wound Care Essentials: Practice Principles*, edited by two of the most respected people in the field of wound care, Dr. Elizabeth Ayello and Sharon Baranoski. As always, these editors have done a superb job of making improvements to a text that is already outstanding. The changes they have made are relevant and timely, and they have brought together an inimitable team of experts to write each chapter. Over half of the chapters in this edition have either entirely new authors or additional experts added to a team of authors to assure the most up-to-date, accurate, and pertinent content.

This edition follows rapidly on the heels of the recent release of the 2014 NPUAP/EPUAP/PPPIA clinical practice guidelines and systematically incorporates these into all appropriate chapters. Readers can be assured that clinical recommendations incorporated from the guidelines have been subjected to structured and deliberate scientific scrutiny to evaluate the strength of the evidence. In addition, clinicians can depend on the expertise of the authors to deliver the best in clinical decision making when evidence is lacking but action is necessary.

High-resolution color photos, so important to understanding the nature of various types of wounds and treatments, are no longer confined to the center pages of the book but are integrated throughout the text and placed in proximity to the content that is germane to the type of wound or type of treatment under discussion. In addition, Chapter 24 has a wound photo gallery of 39 different types of wounds with questions for readers to answer and consider.

Several topics have expanded coverage in this edition. There is an entirely new chapter on Quality of Life issues for patients with wounds that lays a sound foundation of theoretical issues before moving on to clinical concerns related to quality of life. Skin tears have been more thoroughly addressed, and the update includes the work of International Skin Tear Advisory Panel (ISTAP). Also, in this new and improved edition, the reader will find the chapter on wound bioburden has been rewritten and expanded and fistula management has been added to the chapter on tubes and drains. These are but a few examples, but because of the many chapters that have new authors or coauthors, many other changes will be evident to those

familiar with the third edition.

As in the previous edition, a general emphasis on knowledge management remains. Knowledge management involves gathering data from a variety of sources, organizing it so that patterns become apparent, providing context that is relevant to understanding the principles related to action (in this case, professional practice) and delivering all of these things to the end user in timely fashion and in a format that expedites rapid assimilation. These are the "bones" around which the third edition of *Wound Care Essentials: Practice Principles* was built and the fourth edition retains this structure. New authors, new content, and new sources of evidence have been added. Context is provided with patient scenarios and case discussions as well as an expanded use of color photos within the text. Questions are provided at the end of each chapter as a self-assessment with answers provided at the end of the book. All are in a format that supports rapid assimilation of content.

Congratulations to the editors and the authors on producing a book that will help clinicians, whether novices or experts, to manage the complex knowledge surrounding wound care in a truly substantive way.

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Preface

"May there never develop in me the notion that my education is complete, but give me the strength and leisure and zeal continually to enlarge my knowledge"

We agree with the above statement by Maimonides. That's one of the reasons that we have decided to update and expand this fourth edition of *Wound Care Essentials: Practice Principles*. Over the years, you, our colleagues, have told us the role our book has played in your continuing professional education. Whether it was for studying for certification, updating policies and procedures, or serving as a concise compendium of evidence for informed practice. Your feedback is valuable to us; you spoke and we have listened! You asked for some new features and we have delivered. The first obvious change is we have increased the number of color photos, and they are now within each chapter rather than just in one color section. Each chapter has been reviewed, revised, and updated. Your enthusiastic support of the "Show what you know" feature of our book will remain at the end of each chapter. As you requested, we have moved the answers to the back of the book so you don't immediately see the answer below each question. No peeking now! You told us that you wanted to be challenged with more pictures to strengthen your ability to assess and identify wounds. New to this edition of *Wound Care Essentials* is the Wound Gallery. This chapter will give you the opportunity to apply and test your knowledge gained from the fourth edition. See how well you do with this new photo quiz feature of our book. Of course, we have retained all the other components of our book that you love including the patient scenarios at the end of each chapter.

Thank you to all who have come up to us at clinical symposiums and congresses to show us your well used copy! We are thrilled to know that our book hasn't sat on a shelf unused. Essential to our book has been the interprofessional approach to both the content and authorship of the chapters. We are delighted that so many of our previous authors could continue to share their expertise with you as well as to welcome new authors to this fourth edition. Thank you to all of our authors for your contributions. We are deeply grateful to Barbara Braden for once again writing the foreword to this our fourth edition.

Our dream of a book that contained the essentials of wound care was inspired by our mentor and dear late friend, Roberta Abruzzese. We hope we have lived up to her words "keep the words succinct, and clear so to help the clinician by compiling the latest and best information they need in practice."

Bringing a book to publication is no easy task, but we have been supported by a phenomenal team from Wolters Kluwer. Thank you to Shannon Magee, Maria McAvey, Emilie Moyer, Karen Doyle, Tom Conville, and all those who worked to produce our book.

To our readers, we hope the knowledge you acquire from our book will positively impact, and change outcomes for your patients and perhaps even the care delivery systems within your work place. For we believe what Nelson Mandela said "*Education is the most powerful weapon which you can use to change the world*."

Sharon and Elizabeth

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PART I

Wound Care Concepts

Quality of Life and Chronic Wound Care

1

Kevin Y. Woo, RN, PhD, FAPWCA
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We gratefully acknowledge the contributions of Mona Baharestani, PhD, ANP, CWON, CWS, for her work on previous editions of this chapter.

Objectives

After completing this chapter, you'll be able to:

- describe how wounds and those afflicted by wounds are viewed
- identify the impact of quality of life on patients with wounds and their caregivers
- describe ethical dilemmas confronted in wound care
- identify issues and challenges faced by caregivers of patients with wounds
- describe strategies aimed at meeting the needs of patients with wounds and their caregivers.

Wound healing involves complex biochemical and cellular events. Chronic wounds do not follow a predictable or expected healing pathway, and they may persist for months or years.[1] The exact mechanisms that contribute to poor wound healing remain elusive; an intricate interplay of systemic and local factors is likely involved. With an aging population and increased prevalence of chronic diseases, the majority of wounds are becoming recalcitrant to healing, placing a significant burden on the health system and individuals living with wounds and their caregivers. Although

complete healing may seem to be the desirable objective for most patients and clinicians, some wounds do not have the potential to heal due to factors such as inadequate vasculature, coexisting medical conditions (terminal disease, end-stage organ failure, and other life-threatening health conditions), and medications that interfere with the healing process.[2] Whether healing is achievable or not, holistic wound care should always include measures that promote comfort and dignity, relieve suffering, and improve quality of life (QoL).

Case Study

Margaret is an 86-year-old woman who resides in a long-term care facility. With progressive dementia in the last 5 years, she has become incontinent and experienced significant weight loss due to poor oral intake. Margaret developed a stage IV pressure ulcer (PrU) in the sacral area after a recent hospitalization for exacerbation of heart failure. She continues to exhibit symptoms of dyspnea and prefers to sit in a high Fowler position (head of bed above 45 degrees) in bed to help breathing. She gets agitated when she is repositioned, especially in a side-lying position. Her only daughter is distraught over her mother's agitation, and she wonders if the constant repositioning is necessary. During a family meeting, the daughter asked the following questions regarding her mother's care, "If this is not something she likes, are we doing the right thing for her? Is this quality of life?"

Quality of Life and Person-Centered Concerns

What is quality of life (QoL)? Generally, QoL is defined as a general perception of well-being by an individual. It is a subjective but dynamic construct that is influenced by emotions, beliefs and values, social context, and interpersonal relationships, which together account for its variability.[3] Health-related quality of life (HRQoL) refers to the sense of well-being that is specifically affected by health and illness along with other related efforts to promote health, manage disease, and prevent recurrence.[4] According to the model proposed by Wilson and Cleary,[5] multiple overlapping dimensions (e.g., biological and physiological factors, symptoms, functioning, general health perceptions, and overall QoL) are ascribed to the underlying structure of HRQoL. Each component may carry more

importance at a given time based on the context of health and illness. Among people with chronic wounds, there is very little dispute that their QoL is severely diminished.[6,7]

Quality of Life Instruments

There are a number of validated instruments to measure QoL. The generic instruments most commonly used are the Medical Outcomes Study Short Form 36 (SF-36) and adaptations, Research and Development 36-item Form, Sickness Impact Profile, Quality of Life Ladder, Barthel Index, the Nottingham Health Profile, and EuroQol EQ-5D. Specific instruments that are used to evaluate HRQoL for patients with diabetic foot ulcers include Cardiff Wound Impact Schedule Diabetes 39, Norfolk Quality of Life in Diabetes Peripheral Neuropathy Questionnaire, Neuro-QoL, the Manchester-Oxford Foot Questionnaire, and Diabetic Foot Ulcer Scale. For the leg ulcer patient population, the Hyland Leg and Foot Ulcer Questionnaire, Charing Cross Venous Leg Ulcer Questionnaire, and Sheffield Preference-based Venous Leg Ulcer 5D could be considered.[8,9]

Quality of Life and Chronic Wounds

The three major chronic wound types are PrUs, diabetic foot ulcers, and venous leg ulcers. A PrU is an area of skin breakdown due to prolonged exposure to pressure, shear, and friction leading to tissue ischemia and cell death. PrUs remain a significant problem across the continuum of healthcare services; prevalence estimates range from 3.7% to over 27% depending on the setting of care.[10,11] PrUs are linked to a number of adverse patient outcomes including prolonged hospital stay, decline in physical functioning, and death. In fact, patients with a PrU have been reported to have a 3.6-fold increased risk of dying within 21 months, as compared with those without a PrU.[12] Gorecki and colleagues[12] reviewed and summarized 31 studies (10 qualitative studies and 21 quantitative studies) that examined issues related to QoL in people with PrUs. Common concerns and salient issues were synthesized and categorized into the following themes:

1. Physical restrictions resulting in lifestyle changes and the need for environmental adaptations
2. Social isolation and restricted social life
3. Negative emotions and psychological responses to changes in body

image and self-concept, and loss of independence
4. PrU symptoms: management of pain, odor, and wound exudate
5. Health deterioration caused by PrU
6. Burden on others
7. Financial hardship
8. Wound dressings, treatment, and other interventions
9. Interaction with healthcare providers
10. Perception of the cause of PrU
11. Need for education about PrU development, treatment, and prevention

Diabetes is one of the leading chronic diseases worldwide.[13] Persons with diabetes have a 25% lifetime risk of developing foot ulcers that precede over 80% of lower extremity amputations in this patient population.[14,15] The 5-year mortality rates have been reported to be as high as 55% and 74% for new-onset diabetic foot ulcers and after amputation, respectively; the number of deaths surpasses that associated with prostate cancer, breast cancer, or Hodgkin's disease.[15] Individuals with unhealed diabetic foot ulcers share some unique challenges. Due to problems using the foot and ankle, patients with foot ulcers suffer from poor mobility limiting their ability to participate in physical activities.[16] Mobility issues may also interfere with their performance at work resulting in loss of employment and financial hardship. Increased dependence can lead to caregiver stress and unresolved family tension. High levels of anxiety, depression, and psychological maladjustment may affect patients' abilities to participate in self-management and foot care.[16,17]

It is estimated that approximately 1.5 to 3.0 per 1,000 adults in North America have active leg ulcers, and the prevalence continues to increase due to an aging population, sedentary lifestyle, and the growing prevalence of obesity.[18] Chronic leg ulcers involve an array of pathologies: 60% to 70% of all cases are related to venous disease, 10% due to arterial insufficiency, and 20% to 30% due to a combination of both.[19] Although venous leg ulcers are more common in the elderly, 22% of individuals develop their first ulcer by age 40 and 13% before age 30, hindering their ability to work and participate in social activities.[20,21] To understand the experience of living with leg ulceration, Briggs et al.[22] reviewed findings from 12 qualitative studies. Results were synthesized into five categories, similar to those identified above in individuals with PrUs:

1. Physical effects including pain, odor, itch, leakage, and infection

2. Understanding and learning to provide care for leg ulcers
3. The benefits and disappointment in a patient–professional relationship
4. Social, physical, and financial cost of a leg ulcer
5. Psychological impact and difficult emotions (fear, anger, anxiety)

In two other reviews examining the impact of wounds on QoL, a total of 22[23] and 24 studies[24] were identified. Both qualitative and quantitative studies were included in the reviews. Pain was identified as the most common and disabling symptom leading to problems with mobility, sleep disorders, and loss of employment. Other symptoms associated with leg ulcers, including pruritus, swelling, discharge, and odor, are equally distressing but often overlooked by caregivers. In the studies, patients discussed the impact of leg ulcerations on their ability to work, carry out housework, perform personal hygiene, and participate in social/recreational activities. Patients feel depressed, powerless, being controlled by the ulcer, and ashamed of their body. Efforts were taken to conceal the bandages/dressings with clothing or shoes; however, the latter were often considered less attractive than what would normally be worn. Both reviews identified the need to address patient engagement and patient knowledge deficits to promote treatment adherence.[23,24]

Chronic Wound–Related Quality of Life (CW-QoL) Framework

Based on the above review of the literature, common themes were identified to create a conceptual framework for the concept of QoL as it relates to patients with chronic wounds (Fig. 1-1). Included in the framework are two concentric circles and a center representing the individual coping with a chronic wound. The outer circle represents the social, political, and healthcare systems within which QoL is realized and lived. The inner circle outlines six key stressors encountered by people living with chronic wounds:

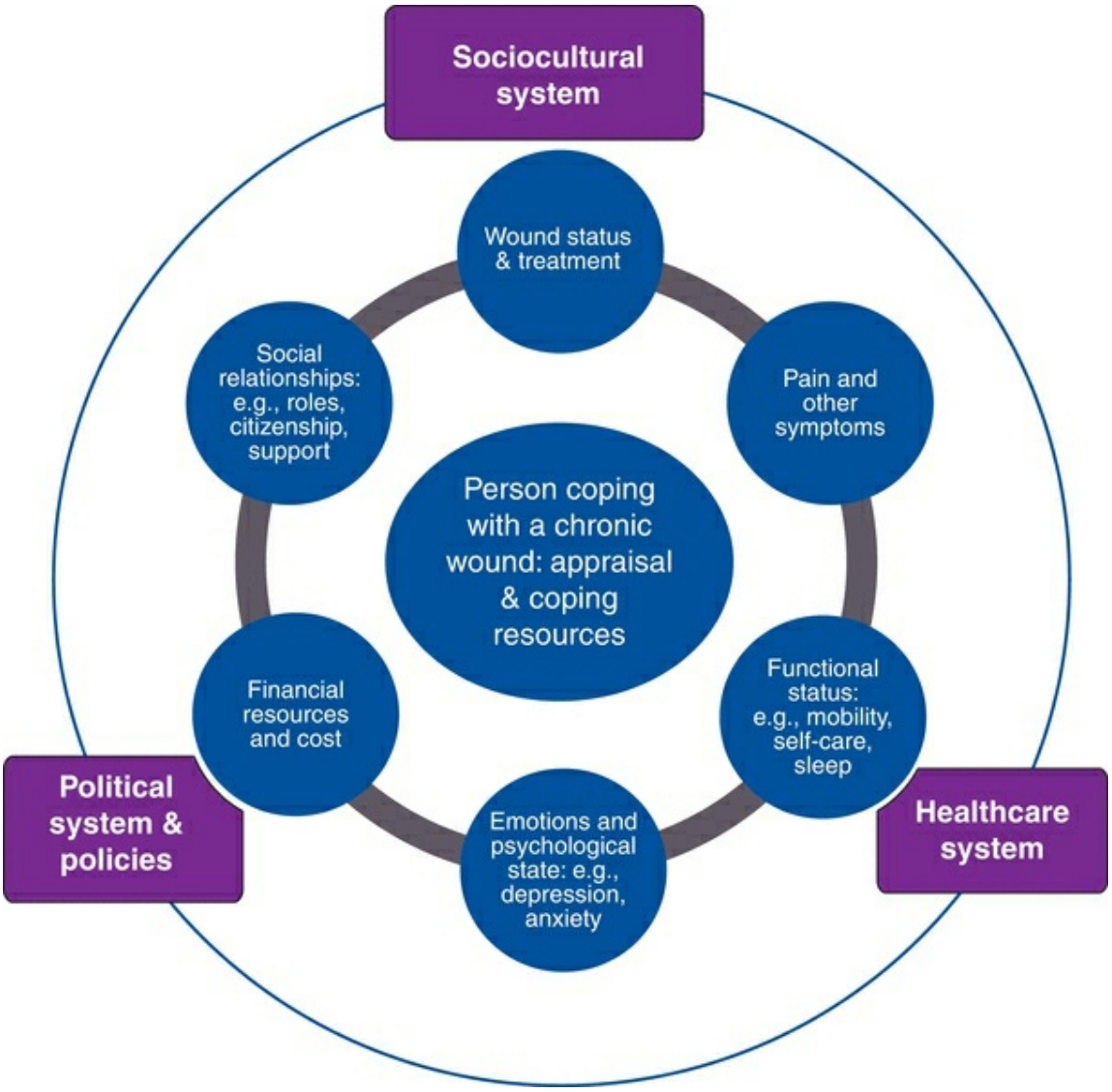

Figure 1-1. Chronic wound–related quality of life (CW-QoL) framework. (Copyright © 2014 KY Woo.)

1. Wound status and treatment
2. Pain and other wound-related symptoms
3. Function status and mobility
4. Emotions and psychological state
5. Financial resources and cost
6. Social relationships

To improve patients' QoL, this paradigm places greater emphasis on the need to foster a climate that elicits patient engagement accompanied by mindful scanning of the environment and health resource mapping. Individualized wound care plans that address specific patient-centered concerns are most likely to succeed and promote the best outcomes for the

patient with a wound. Standardized wound care plans often fail because they do not promote patient adherence/coherence. Patients may be labeled "noncompliant" when the real problem is that the care plan has not been properly individualized to their specific needs taking into account their perspectives on QoL.

Person Coping with a Chronic Wound

People who are living with chronic wounds describe the experience as isolating, debilitating, depressing, and worrisome, all of which contribute to high levels of stress. Stress has a direct impact on QoL. Lazarus and Folkman[25] postulated that stress is derived from cognitive appraisals of whether a situation is perceived as a threat to one's well-being and whether coping resources that can be marshaled are sufficient to mitigate the stressor. Stress appraisal is constructed when the demands of a situation outstrip perceived coping resources.[25] While no one is immune to stress, the impact of a chronic wound on individual's perception of well-being and QoL depends on personal meanings and values that are assigned to the demands that arise from living with a chronic wound. Coping is less adaptive or effective if people lack self-esteem, motivation, and the conviction that they have the aptitude to solve a problem. Woo[26] evaluated the relationship between self-perception and emotional reaction to stress in a sample of chronic wound patients. Findings suggest that people who are insecure about themselves tend to anticipate more wound-related pain and anxiety.

Chronic Stress Is Not Innocuous

Stress triggers a cascade of physiological responses featured by the activation of the hypothalamic–pituitary–adrenal axis that produces vasopressin and glucocorticoid (cortisol).[27] Vasopressin is known for its property to induce vasoconstriction that could potentially be harmful to normal wound healing by compromising the delivery of oxygen and nutrients. Cortisol attenuates the immunoinflammatory response to stress. Excessive cortisol has been demonstrated to suppress cellular differentiation and proliferation, inhibit the regeneration of endothelial cells, and delay collagen synthesis. The body of scientific evidence that substantiates the deleterious impact of protracted stress on wound healing is convincing.[28] In one study, Ebrecht et al.[29] evaluated healing of acute

wounds created by dermal biopsy among 24 healthy volunteers. Stress levels reported by the participants via the Perceived Stress Scale were negatively correlated to wound healing rates 7 days after the biopsy ($P < 0.05$). Subjects exhibiting slow healing (below median healing rate) rated higher levels of stress during the study ($P < 0.05$) and presented higher cortisol levels 1 day after biopsy than did the fast-healing group ($P < 0.01$). Kiecolt-Glaser and colleagues[30] compared wound healing in 13 older women (mean age = 62.3 years) who were stressed from providing care for their relatives with Alzheimer disease, and 13 controls matched for age (mean age = 60.4 years). Time to achieve complete wound closure was increased by 24% or 9 days longer in the stressed caregiver versus control groups ($P < 0.05$).

Cognitive–behavioral strategies and similar psychosocial interventions are designed to help people reformulate their stress appraisal and regain a sense of control over their life's problem within an empathic and trusting milieu. Ismail et al.[31,32] identified 25 trials that utilized various psychological interventions (e.g., problem solving, contract setting, goal setting, self-monitoring of behaviors) to improve diabetic self-management. Patients allocated to psychological therapies demonstrated improvement in hemoglobin A1c (12 trials, standardized effect size = −0.32; −0.57 to −0.07) and reduction of psychological distress including depression and anxiety (5 trials, standardized effect size = −0.5; −0.95 to −0.20).

Simple problem-solving technique is easy to execute and provides a step-by-step and logical approach to help patients identify their primary problem, generate solutions, and develop feasible solutions. The key sequential steps are[31]:

1. explanation of the treatment and its rationale
2. clarification and definition of the problems
3. choice of achievable goals
4. generation of alternative solutions
5. selection of a preferred solution
6. clarification of the necessary steps to implement the solution
7. evaluation of progress.

Wound Status and Management

The trajectory for wound healing is tortuous and unpredictable punctuated by wound deterioration, recurrence, and other complications. Despite appropriate management and exact adherence to instructions, there is no

guarantee that healing will occur. The following quotes are some of the narratives that patients voiced to convey their worry, frustration, and feeling of powerlessness.

> "The wound doctor asked me to use this dressing, but the wound is not getting better. I don't know what else to do?"[33]
> "The wound is getting bigger, and now I am getting an infection; I don't know why this is happening to me?"[33]

Even when best practice is implemented, some treatment options are not feasible and they are not conducive to enhance patients' QoL, for example, a patient with foot ulcers who cannot use a total contact cast because he needs to wear protective footwear at work and he cannot maintain his balance walking on a cast; a patient with venous leg ulcer who likes to take a shower every day to maintain personal hygiene but cannot do so because she needs to wear compression bandages; or a patient with a PrU who refused an air mattress because it generates too much noise that interferes with sleep. While turning patients every 4 hours has been recommended, repositioning can be painful, especially among patients who have significant contractures, increased muscle spasticity, and spasms. Among critically ill individuals, repositioning may precipitate vascular collapse or exacerbate shortness of breath (as with, e.g., advanced heart failure).[34] According to the study of hospitalized patients with PrUs,[35] it was surprising for investigators to learn that even assuming a side-lying position could be uncomfortable. Briggs and Closs[36] indicated that only 56% of patients in their study were able to tolerate full compression bandaging, with pain being the most common reason for nonadherence. Patients should be informed of various treatment options and be empowered to be active participants in care. Being an active participant involves taking part in the decision making for the most appropriate treatment, monitoring response to treatment, and communicating concerns to healthcare providers.

Much discussion in the qualitative literature has been centered on patients trying to find explanations on how chronic wounds develop.[22,23] As a reminder, only a small proportion of patients are cognizant of factors contributing to their chronic wounds and treatment strategies to improve their conditions.[37] Patients and family need to understand that chronic wounds are largely preventable but not always avoidable. When circulation is diverted from the skin to maintain hemodynamic stability and normal functioning of vital organs, skin damage is inevitable.

Complications such as wound infection are common but upsetting. According to an analysis of an extensive database comprising approximately 185,000 patients attending family medical practitioners in Wales, 60% of patients with chronic wounds had received at least one antibiotic in a 6-month period for the treatment of wound infection.[38] Bacteria compete for nutrients and oxygen that are essential for wound healing activities, and they stimulate the overproduction of proteases leading to degradation of extracellular matrix and growth factors.[39] Among patients with diabetic foot ulcers, wound infection is one of the major risk factors that precede amputations. Surgical site infection has been linked to prolonged hospitalization and high mortality.[40] In fact, the mortality rate has been reported to be over 50% in patients with bacteremia secondary to uncontrolled infection in PrUs.[38] Receiving a diagnosis of an infection is anxiety provoking; patients often fear that infection is the beginning of a downward vicious cycle leading to hospitalization, limb amputation, and death. The need to align expectations and dispel misconceptions cannot be underestimated.

Pain and Other Symptoms

Wound-associated pain continues to be a common yet devastating symptom, often described as one of the worst aspects of living with chronic wounds.[41] Sleep disturbance, immobility, poor appetite, and depression are some of the consequences of unremitting pain. In an international survey of 2018 people with chronic wounds, over 60% of the respondents reported the experience of pain "quite often" and "all the time."[42] Szor and Bourguignon[43] reported that as many as 88% of people with PrUs in their study expressed PrU pain at dressing change and 84% experienced pain even when unprovoked. Of people with venous leg ulcers, the majority experienced moderate to severe levels of pain described as aching, stabbing, sharp, tender, and tiring.[44] Pain has been documented to persist up to at least 3 months after wound closure. Contrary to the commonly held belief that most patients with diabetic foot ulcers do not experience pain due to neuropathy, up to 50% of patients experience painful symptoms at rest and approximately 40% experience moderate to extreme pain climbing stairs or walking on uneven surfaces.[45] Patients with diabetes who report pain most or all of the time had statistically and clinically significantly poorer health-related QoL than those who did not report pain.[46] However,

pain in diabetes is often underestimated and undertreated. The need to improve pain assessment and management is incontestable. Pharmacotherapy continues to be the mainstay for pain management. Appropriate agents are selected based on severity and specific types of pain (see Chapter 12, Pain Management and Wounds).

Pruritus

Pruritus is another frequent complaint among people with chronic wounds. Of 199 people with chronic wounds who were surveyed, Paul, Pieper, and Templin[47] documented that 28.1% complained of itch. Peripheral pruritus is often triggered by pruritogens (e.g., histamine, serotonin, cytokines, and opioids), giving rise to signals that are transmitted via pain-related neuronal pathways and terminated in somatosensory cortex where the sensation of itch is perceived.[48] In contrast, central pruritus is associated with psychiatric disorders or damages to the nervous system mediated through opioid and serotonin receptors. For patients with wounds, itch is commonly caused by peripheral stimulation of itch receptors due to irritation of the skin and related dermatitis.[49] People with chronic wounds are exposed to a plethora of potential contact irritants accounting for approximately 80% of all cases of contact dermatitis. Excessive washing and bathing strips away surface lipid and induces dryness that can exacerbate pruritus. To replenish skin moisture, humectants or lubricants should be used on a regular basis. Drug treatment with paroxetine, a selective serotonin reuptake inhibitor, and gabapentin has been shown to be beneficial in palliative care patients.

Odor

Probst interviewed people with fungating breast wounds, and odor was highlighted as one of the most distressing symptoms that compromised their QoL.[50] Unpleasant odor and putrid discharge is associated with increased bacterial burden, particularly involving anaerobic and certain gram-negative (e.g., pseudomonas) organisms. Metabolic by-products that produce this odor include volatile fatty acids (propionic, butyric, valeric, isobutyric, and isovaleric acids), volatile sulfur compounds, putrescine, and cadaverine.[48] To eradicate wound odor, topical antimicrobial and antiseptic agents are recommended.

Exudate

Wound exudate contains endogenous protein-degrading enzymes, known as proteases or proteinases that are extremely corrosive and damaging to the intact skin.[51] When drainage volume exceeds the fluid handling capacity of a dressing, enzyme-rich and caustic exudate may spill over the wound margins, causing maceration or tissue erosion (loss of part of the epidermis but maintaining an epidermal base) and pain. Leakage of highly exudative wounds on to clothing, furniture, and bed linens can lead to feelings of embarrassment and inhibited sexuality and intimacy.[52–54] Careful selection of discreet absorbent dressings (avoid bulky materials) will improve patients' QoL.

Functional Status

According to the International Classification of Functioning, Disability and Health of the World Health Organization, disability refers to impairment, activity limitations, and participation restrictions because of the interaction between a health condition and other physical, social, mental, or emotional factors.[55] The majority of patients with chronic wounds suffer mobility problems, and their ability to perform activities of daily living is limited. Activities often taken for granted by the general population, such as taking a shower, getting dressed, and even walking up the stairs, could become an enormous challenge for people with chronic wounds. In a study consisting of 88 patients with chronic leg ulcers, 75% reported difficulty performing basic housework.[56] Yet another study by Hyland et al.[57] revealed that of 50 patients with leg ulcers, 50% had problems getting on and off a bus and 30% had trouble climbing steps. Due to increased disability, patients are becoming ever more dependent on others for help. Requesting and receiving assistance could be a hassle and embarrassment, especially if the patient lives alone and needs regular help. Easy access to transportation and changes to living arrangements (such as widening doors for a wheelchair) will enhance individual's ability to functional independently, but the effort to organize and execute the plan could be daunting.[34]

Emotional and Psychological State

People with chronic wounds tend to experience more emotional problems than people without wounds in the community and are less capable to cope

with stressful events.[58] Healthcare practitioners (n = 908), in responding to a Web-based survey, acknowledged that mental health issues are common in people with chronic wounds. Over 60% of the survey respondents indicated that between 25% and 50% of people with chronic wounds suffer from mental disorders.[59] Among all the symptoms, anxiety was rated the most common (81.5%). These results are consistent with findings from a pilot study in which over 60% of people living with chronic wounds expressed higher-than-average anxiety.[60]

Financial and Cost

Patients with chronic wounds are often unemployed, marginalized, and isolated. In a study of 21 patients with diabetic foot ulcers by Ashford, McGee, and Kinmond,[61] 79% of patients reported an inability to maintain employment secondary to decreased mobility and fear of someone inadvertently treading on their affected foot. In another study, all patients interviewed felt that the leg ulcer limited their work capacity, with 50% adding that their jobs required standing most of their shift.[62] In that same study, 42% of patients identified the leg ulcer as a key factor in their decision to stop working. Even for younger patients, leg ulceration was correlated with time lost from work and job loss, ultimately affecting finances.[62] Beyond occupational stressors and dilemmas, patients may incur additional out-of-pocket expenses for transportation, parking, telephone bills for medical follow-up, home health aide services, dressing supplies not covered by insurance, and drug costs if they have no prescription plan. Those who have no insurance but don't qualify for public assistance may be forced to tap into their savings or refinance their homes. Healthcare professionals, rather than simply dismissing patients as nonadherent, should show empathy, acknowledging access and financial hardships faced by patients, and partner with their patients in addressing these issues.[63,64]

Social Relationships and Role Function

Feeling embarrassed about the repugnant smell and fluid leakage from wounds and their bodies, people with chronic wounds may intentionally avoid social contacts and activities. Patients often feel detached and emotionally distant from their friends and families, rendering it difficult to maintain meaningful friendship and romantic relationships. Patients with

chronic wounds are frequently isolated and lack social support. The concept of social support refers to an interactive process that entails perceived availability of help or support actually received. In a study of 67 patients with venous leg ulcers, Edwards and coinvestigators[65] evaluated the impact of a community model of care on QoL. Subjects were randomized to receive individual home visits from community nurses (the control group) or to pay a weekly visit to a nurse-managed leg club (the intervention group). Leg clubs offer a setting where the subjects could obtain advice/information to manage their ulcers through social interaction with expert nurses as well as with their peers. Subjects who attended the leg club expressed significant improvement in QoL ($P < 0.014$), morale ($P < 0.001$), self-esteem ($P = 0.006$), pain ($P = 0.003$), and functional ability ($P = 0.004$).

The notion that social media could be leveraged to provide virtual social support is gaining popularity. Social media encompasses a variety of platforms that provide opportunities for multiple users to exchange experiences and information and to provide support through multisensory communication. According to a 2012 survey, 61% of adult Internet users searched online and 39% used social media to obtain health information.[66] Of all the posted messages and dialogues that were abstracted from 15 Facebook groups focused on diabetes management, almost 30% of the content was related to the exchange of emotional support among members of a virtual community.[67] In one study that evaluated an online diabetes self-management program,[68] individuals randomized to the program exhibited a significant improvement in blood sugar control (A1c level), self-reported knowledge/skill, and self-efficacy compared with those who received usual care. However, the actual participation in household activities, recreation, and exercise was not different between the two study groups. Nicholas et al.[69] designed an online module to educate and provide support to adolescents with diabetes. Participants who were randomized to the treatment group received eight online information modules and participated in peer-to-peer online dialogue that was moderated by a social worker specialized in diabetes care. Perceived social support was rated higher in the treatment group compared to the control group, but the result was not significant given the small sample size ($n = 31$).

Healthcare System

Navigating through the healthcare system could be extremely confusing. A trusting and therapeutic relationship between patients and their healthcare

providers may serve to buffer the effects of adversity and stress. However, patients sometimes criticize about feeling rushed and spending limited time during routine visits at wound care clinic. Patients discussed the importance of having healthcare providers who care and display a genuine interest in their well-being. In their description of the key attributes of someone who cares, patients use terms like “caring,” “holistic,” “friendly,” being “vigilant,” “cheerful,” “gentle,” and “knowledgeable.” Healthcare providers should provide clear and consistent communication, to avoid confusion.

Sociocultural System

A recurring theme emerged from the literature that articulated the bleak feeling of isolation due to wound-related stigma. Given the negative image by which wounds are viewed (Table 1-1), it isn’t surprising that patients with wounds are sometimes considered unattractive, imperfect, vulnerable, a nuisance to others, and, in some cases, even repulsive.[70–72] This can dramatically affect a patient’s emotional response to their wound and their self-esteem. For patients who must endure the displeasing stares of others to their bandaged wounds, a wound clinic may be the only place where they can receive positive energy and reinforcement.[37]

Table 1-1 Emotional Impact of Wounds

In addition to the morbidity associated with wounds and the physical discomfort, wounds have an inherent emotional effect on the patient, caregivers, family, friends, and strangers the patient may encounter. Even healthcare professionals aren't immune to an emotional response to a patient's wound.

Wounds are typically perceived as:

- a betrayal of one's own body
- appalling, disgusting, repulsive
- haunting, scary, associated with horror movies
- nuisance, time-consuming, costly
- smelly, dirty, disgusting
- unpleasant, uncomfortable.
- The patient's own perception of his/her wound may include such feelings as:
- embarrassment, humiliation
- guilt, shame
- needing bandages to "hide the evidence" (i.e., of imperfection).

Political System and Policies

Health policy refers to plans, processes/structures, and actions that are established to achieve specific healthcare goals within a society. Priority setting to optimize health services delivery should include easy access to resources, appropriate funding/reimbursement mechanisms, communication strategies, and sustainable training for staff. Pressure redistribution and downloading is critical for the management of foot ulcers by removing pressure and preventing recurrent injury to the affected areas. However, these devices are expensive and may require ongoing modification by a trained professional such as a podiatrist or chiropodist. Diabetic neurotrophic ulcers may have the potential to heal, but fail because downloading is not optimized. Similarly, compression therapies are used to treat venous leg ulcers. However, in Canada, there is no additional funding or reimbursement program to cover the cost for chiropody services and the purchase of therapeutic footwear/stockings.

Conclusion

Provision of wound care requires a systematized and holistic approach to address comorbid conditions and psychosocial issues, expertise that extends

beyond local wound care and dressing selection. A well-coordinated and interprofessional team approach is integral to the delivery of high-performance and evidence-based wound care services. Management of these ulcers involves a detailed examination and discussion with patients to adequately address their concerns. Although traditional educational interventions to improve knowledge are necessary, they are rarely sufficient to change behaviors. Emerging evidence highlights a need to shift the chronic disease management paradigm to focus on patient engagement and self-management (Fig. 1-2).

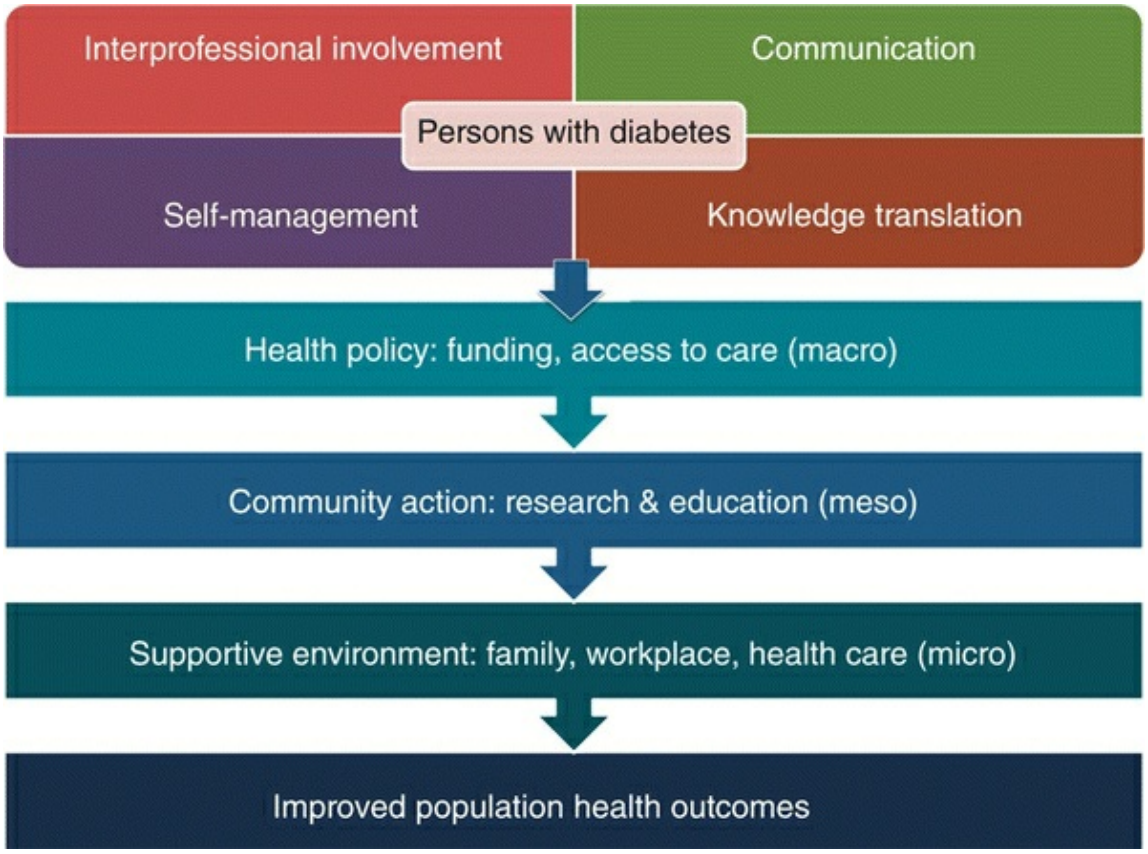

Figure 1-2. Multilevel chronic disease self-management model. (Copyright © 2014 KY Woo.)

Show What You Know

1. **Those afflicted with wounds are often viewed as:**
   A. pleasant and comfortable.
   B. pain-free.
   C. appalling and repulsive.

D. attractive.

2. Wound assessment is commonly lacking in the area of:

A. size.
B. odor.
C. drainage.
D. pain.

3. Quality-of-life treatment decisions should be based on the:

A. patient's perception of well-being.
B. nurses' perceptions of well-being.
C. family's perception of well-being.
D. physicians' perceptions of well-being.

4. Impact of chronic wound healing and the individual's perception of well-being includes:

A. stress appraisal.
B. motivation.
C. coping resources.
D. all of the above.

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Reimbursement Regulations Impacting Wound Care

2

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Objectives

After completing this chapter, you'll be able to:

- discuss the significance of the U.S. Centers for Medicare and Medicaid Services
- discuss reimbursement issues related to hospitals, skilled nursing facilities, and home health agencies
- identify quality improvement efforts
- describe essential wound documentation required for reimbursement.

Chapter Overview

Reimbursement regulations in wound care as in any other sector of health care can be quite complex. This chapter is organized into five major sections, which are as follows: role of regulations in health care, government payers in wound care, principles of wound care reimbursement, reimbursement of clinicians in different practice settings, and finally quality

assessment and improvement issues.

Role of Regulation in Health Care

Regulations are a pervasive feature of the American healthcare system and, not surprisingly, significantly impact the delivery of wound care. Quite often, regulations and reimbursement determine who receives wound care and the level of wound care that's delivered. Thus, if clinicians are to provide optimum care, it is essential for them to have knowledge about the regulations that impact wound care in their specific practice setting.

Although many clinicians may view the current regulatory environment as burdensome and unnecessary, it's essential to recognize the important purpose that regulations fulfill. Quite simply, regulations are the mechanism through which government may promote its interest in the general welfare of society. Experience has demonstrated that government cannot rely solely on conventional market forces, such as the laws of supply and demand, to guide the use of resources to provide optimum care. These market forces, in the absence of the guiding hand of regulations, are often insufficient to ensure that healthcare resources are distributed equitably. In the case of wound care, the goal of current regulations is to ensure access to high-quality wound care, particularly for vulnerable populations such as the elderly and nursing home residents. Wound care regulations must be viewed from the perspective of how well they are achieving this goal.

At least four types of regulatory vehicles are available to the government to help achieve this goal. Government regulations may rely on subsidies or direct payments to providers; they may involve entry restrictions such as licensure and accreditations that seek to limit the ability to offer a particular service; they could use rate-setting or price-setting controls that determine reimbursements for care provided; or they could involve quality controls that seek to improve the care that is provided. Of these different potential mechanisms, the latter two are clearly the major regulatory vehicles used in wound care today and are the focus of this chapter. In this chapter, we describe the government payers in wound care and concentrate on the major regulatory agency involved in wound care in the United States—the Centers for Medicare and Medicaid Services (CMS)—including an overview of how wound care is covered, coded, and reimbursed by CMS and its contractors; the different reimbursement scenarios depending on the clinicians' practice settings; and a description of the agency's efforts in improving the quality of wound care. Through these efforts, CMS aims to improve health and health care while also making care

more affordable.

General Information on Government Payers in Wound Care

Centers for Medicare and Medicaid Services

CMS is a federal agency within the U.S. Department of Health and Human Services. Prior to July 1, 2001, it was called the Health Care Financing Administration (HCFA). CMS administers the Medicare and Medicaid programs—two national healthcare programs that benefit about 75 million Americans. Moreover, because CMS provides the states with at least 50% of their finances for healthcare costs, the states must comply with federal regulations.

The difference is that while both the Medicare and Medicaid programs are administered through federal statutes that determine beneficiary requirements, what is covered, payment fees and schedules, and survey processes of clinical settings (such as skilled nursing facilities [SNFs] or home health agencies) are determined by their respective programs. Both programs have a wide variance on coverage, eligibility, and payment fees and schedules. Therefore, it's important for the clinician to know what's covered and the level of reimbursement prior to developing a treatment plan with the patient. Because CMS remains the largest health insurance agency, Medicaid as well as many private insurance companies will provide coverage at similar levels.

Medicare

The Medicare program was developed in 1965 by the federal government.[1] In order to qualify for Medicare benefits, a person must be age 65 or older, have approved disabilities if under age 65, or have end-stage renal disease.

In 2013, Medicare provided coverage to 54 million people, spending $583 billion[2] on benefits.[3] These benefit payments are funded from two trust funds—the Hospital Insurance (HI) trust fund and the Supplementary Medical Insurance (SMI) trust fund. Most often these are referred to as Medicare Part A and Medicare Part B, respectively.[4]

The HI trust fund pays for a portion of the costs of inpatient hospital services and related care. Those services include critical access hospitals

(small facilities that give limited outpatient and inpatient services to people in rural areas), SNFs, hospice care, and some home healthcare services. The HI trust fund is financed primarily through payroll taxes, plus a relatively small amount of interest, income taxes on Social Security benefits, and other revenues.

The SMI trust fund pays for a portion of the costs of physicians' services, outpatient hospital services, and other related medical and health services. As of 2014, the premium for Medicare Part B is $104.90 per month. This premium will not change in 2015. In some cases, this amount may be higher if the person doesn't choose Medicare Part B when he or she first becomes eligible at age 65 or if the person files taxes greater than $85,000 as an individual or $170,001 as part of a couple. In addition, as of January 2006, the SMI trust fund pays for private prescription drug insurance plans to provide drug coverage under Part D of the program. The separate Part B and Part D accounts in the SMI trust fund are financed through general revenues, beneficiary premiums, interest income, and, in the case of Part D, special payments from the States.

The Medicare+Choice program was authorized by the Balanced Budget Act of 1997.[5] In this program, beneficiaries have the traditional Medicare Part A and Part B benefits, but they may also select Medicare managed care plans (such as health maintenance organizations [HMOs], preferred provider organizations [PPOs], or private fee-for-service plans). Medicare+Choice plans provide care under contract to Medicare. They may provide benefits such as coordination of care or reducing out-of-pocket expenses. Some plans may also offer additional benefits, such as prescription drugs.

Prescription drug benefits are available for all Medicare beneficiaries regardless of income, health status, or prescription drug use[6] through Medicare Part D. A range of plans are available, so beneficiaries have multiple options for coverage. Moreover, persons can add drug coverage to the traditional Medicare plan through a "stand-alone" prescription drug plan or through a Medicare Advantage plan, which includes an HMO or PPO and typically provides more benefits at a significantly lower cost through a network of doctors and hospitals. Presently, no wound care products are covered under this benefit.

Patient Teaching

Explain to the patient and family that wound care products are not covered under Medicare Part D.

Medicaid

The Medicaid program was developed in 1965 as a jointly funded cooperative venture between the federal and state governments to assist states in the provision of adequate medical care to eligible people.[1] Medicaid is the largest program providing medical and health-related services to America's poorest people. Within broad national guidelines provided by the federal government, each of the states:

- administers its own program
- determines the type, amount, duration, and scope of services
- establishes its own eligibility standards
- sets the rate of payment for services
- determines what products are covered in that state.

Thus, the Medicaid program varies considerably from state to state as well as within each state over time. This wide variance also affects what's covered in wound care. For example, the number of times debridement of a wound is reimbursed differs by state, as do product treatment options.

Managed Care Organizations

Manage Care Organizations (MCOs) were developed to provide health services while controlling costs. They combine the responsibility for paying for a defined set of health services with an active program to control the costs associated with providing those services, while at the same time attempting to control the quality of and access to those services. The health benefits, which usually range from acute care services to dental and vision coverage, are usually clearly identified, as are the payment, co-payment, and deductibles that are required for a specific health procedure (e.g., compression therapy for chronic venous insufficiency ulcer). Moreover, the MCO usually receives a fixed sum of money to pay for the benefits in the plans for the defined population of enrollees. Typically, this fixed sum is constructed through premiums paid by the enrollees, capitation payments made on behalf of the enrollees from a third party, or both. There are wide variations in MCOs and the services they provide for patients with wounds.

General Wound Care Reimbursement Principles

Reimbursement directly impacts how clinicians deliver care. Increasingly, third-party payer sources (Medicare, Medicaid, HMOs) are examining where their money is going and whether they're getting the most from providers on behalf of their beneficiaries. Thus, third-party payers are requiring more documentation regarding patient outcomes to justify payment. Clinicians who can document comprehensive and accurate assessments of wounds and the outcomes of their interventions are in a stronger position to obtain and maintain coverage and thus reimbursement.

Evidence-based wound care should always be the goal of clinicians. However, clinicians are increasingly being challenged to provide optimum wound care based on healthcare setting and third-party payers.

Medicare reimbursement is more than just the payment for medical items and services. The key to understanding how Medicare reimburses providers, physicians and suppliers for wound care involves a greater understanding of three main components that comprise the Medicare reimbursement system: Coding, Coverage, and Payment. Each is a separate and distinct process. Just because a product is awarded a code does not mean it will be covered. Just because it is awarded a code and covered does not mean it will be reimbursed. Similarly, all procedures performed by clinicians have codes assigned to them and are reimbursed based on the payment system for the setting in which it was performed.

Since coding and coverage are universal to all settings, they will be discussed first. Then we will discuss reimbursement in a setting-specific fashion.

Coding

In order for medical claims to be processed, billing codes are used by physicians, hospitals and other providers to identify the diagnosis, product, service and procedure that the clinician used in treating the patient in which they are billing a payer. Accurate coding is necessary in order for the claim to be properly and accurately processed.

The types of codes that are used include:

- Healthcare Common Procedure Coding System (HCPCS) Level I and Level II
- Diagnosis-Related Group (DRG)

- International Classification of Diseases (ICD-9 and soon to be released ICD-10)

HCPCS Level I

HCPCS Level I or Common Procedural Terminology (CPT®) codes are numbers assigned to a procedure that a clinician (e.g., physician, nurse practitioner, podiatrist) may perform on a patient, including medical, surgical, and diagnostic services. The codes are then used by insurers (Medicare, Medicaid, and private payers) to determine the amount of reimbursement for the clinician. Every clinician uses the same codes to ensure uniformity, but the amount of reimbursement may differ depending on the type of clinical professional. An example of a CPT code for wound care is CPT 11042—debridement of subcutaneous tissue, first 20 cm^2 or less.

HCPCS Level II

HCPCS Level II code set is made up of five-character alphanumeric codes representing primarily medical supplies, durable medical goods, nonphysician services, and services not represented in the Level I code set (CPT). HCPCS Level II includes services such as ambulance, durable medical equipment, prosthetics, orthotics, and supplies (DMEPOS) when used outside a physician's office. Cellular and/or Tissue Based Products for Wounds (CTPs), an updated and more clinically appropriate term for "skin substitutes"; surgical dressings; support surfaces; and negative pressure wound therapy (NPWT) all have HCPCS Level II codes.

Diagnosis-Related Group

DRG is used for inpatient hospital claims. DRGs are a means of classifying a patient under a particular group where those assigned are likely to need a similar level of hospital resources for their care. This allows hospital administrators to more accurately determine the type of resources needed to treat a particular group and to predict more closely the cost of that treatment.

International Classification of Diseases (ICD-9 and Soon to be Released ICD-10)

ICD-9 International classification of diseases (soon to be ICD-10) is a set of **codes** used by physicians, hospitals, and allied health workers to indicate

diagnosis for all patient encounters. The ICD-9-CM coding system contains about 16,000 diagnosis codes and ICD-10-CM contains over 68,000 codes. Currently the ICD-10 release has been delayed in the United States.

Coverage

Coverage is the existence of a medical benefit category for a service, procedure, device, drug or supply used in healthcare delivery. Coverage varies by the type of health plan (i.e., Medicare, Medicaid, private pay, etc.), the setting of care (i.e., hospital, home health, SNF, physician office, etc.), and the condition of the patient. If coverage is permissible, payers may have a separate coverage policy that will dictate the specific criteria in which they will permit coverage of that product, service, or procedure. The coverage policy will set forth medical conditions, diagnosis, coding, and specific requirements and/or limitations for coverage of that particular service or product.

The different settings by which coverage may be permitted for wound care includes hospitals (inpatient, outpatient, and long-term care hospitals), outpatient clinics—including wound care clinics, SNFs, and physician offices and also home care.

In the Medicare program, there is no national coverage policy for most of the products that would be used to treat a patient with a chronic wound. Rather, coverage for most wound care products are made through local coverage determinations (LCDs) by the Part A and B Medicare Administrative Contractors (AB MACs) for CTPs and the Durable Medical Equipment Medicare Administrative Contractors (DMEMACs) for surgical dressings, NPWT and support surfaces. The only product provided to wound care patients in which there is a national coverage decision issued by CMS is hyperbaric oxygen treatment.

The following is additional information regarding the CMS contractors who create and implement the LCDs—the DMEMACs and the A/B MACs

Durable Medical Equipment Medicare Administrative Contractors

Implementation of the Medicare program (for instance, eligibility requirements and payments) in home care is handled by numerous insurance companies that are subcontracted by CMS. In 1993, CMS contracted four carriers to process claims for DMEPOS under Medicare Part B.[7] CMS divided the country into four regions, with each region having its own DME

regional carrier. The HCPCS, an alphanumeric system used to identify coding categories not included in the American Medical Association's CPT-4 codes, is usually used with DMEPOS.[8]

In January 2006, CMS eliminated fiscal intermediaries who processed Medicare claims (Medicare Part A only) and carriers (Medicare Part B only),[9] eliminated the DME regional carriers, and awarded four specialty contractors through a competitive bidding process. The new DME Medicare administrative contractors (DME MACs) are responsible for handling the administration of Medicare claims from DMEPOS suppliers. The benefit of the new system is a more streamlined process between the beneficiary and the supplier. The DME MACs serve as the point of contact for all Medicare suppliers, whereas beneficiaries can register their claims-related questions to Beneficiary Contact Centers (Table 2-1).

Table 2-1 The Four Durable Medical Equipment Medicare Administrative Contractors

- National Government Services (NGS), serving Illinois, Indiana, Kentucky, Michigan, Minnesota, Ohio, and Wisconsin
- *National Heritage Insurance Company (NHIC),* serving Connecticut, Delaware, District of Columbia, Maine, Maryland, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, and Vermont
- *Noridian Administrative Services,* serving Alaska, American Samoa, Arizona, California, Guam, Hawaii, Idaho, Iowa, Kansas, Missouri, Montana, Nebraska, Nevada, North Dakota, Northern Mariana Islands, Oregon, South Dakota, Utah, Washington, and Wyoming
- Cigna Government Services (CGS), serving Alabama, Arkansas, Colorado, Florida, Georgia, Louisiana, Mississippi, New Mexico, North Carolina, Oklahoma, Puerto Rico, South Carolina, Tennessee, Texas, U.S. Virgin Islands, Virginia, and West Virginia

DME MACs clearly define local medical coverage policies. The beneficiary usually pays the first $100.00 for covered medical services annually. Once that has been met, the beneficiary pays 20% of the Medicare-approved amount for services or supplies. If services weren't provided on assignment, then the beneficiary pays for more of the Medicare coinsurance

plus certain charges above the Medicare-approved amount.

Medicare Part B provides coverage for NPWT pumps. In order for an NPWT pump and supplies to be covered, the patient must have a chronic stage III or IV pressure ulcer, neuropathic ulcer, venous or arterial insufficiency ulcer, or a chronic (at least 30 days) ulcer of mixed etiology. Extensive documentation is required prior to a DME MAC approving coverage for NPWT. Thus, it is important for the clinician to review the coverage policy to ensure that the product is covered under the Medicare program.[10]

Support surfaces are also covered under Medicare Part B.[10–12] CMS has divided support surfaces into three categories for reimbursement purposes:

- Group 1 devices are those support surfaces that are static and don't require electricity. Static devices include air, foam (convoluted and solid), gel, and water overlay or mattresses.
- Group 2 devices are powered by electricity or pump and are considered dynamic in nature. These devices include alternating and low-air-loss mattresses.
- Group 3 devices are also considered dynamic in nature. This classification comprises only air-fluidized beds.

Specific criteria must be met before Medicare will reimburse for support surfaces; therefore, it is essential for the clinician to review the policy.

The surgical dressings benefit covers primary and secondary dressings in outpatient acute care clinic settings (e.g., a hospital outpatient wound center) and physician offices (Table 2-2).[13] This coverage policy is determined by the DMEMACs as well.

Table 2-2 Coverage Under the Surgical Dressings Benefit

To have the dressings reimbursed under the Medicare/Medicaid surgical dressings benefit, the following criteria must be met:

- The dressings are medically necessary for the treatment of a wound caused by, or treated by, a surgical procedure.
- The dressings are medically necessary when debridement of a wound is medically necessary.

In certain situations, dressings aren't covered under the surgical dressings benefit, including those for:

- drainage from a cutaneous fistula that has not been caused by or treated by a surgical procedure
- first-degree burn
- stage I pressure ulcer
- wounds caused by trauma that don't require surgical closure or debridement (such as skin tears and abrasions)
- venipuncture or arterial puncture site other than the site of an indwelling catheter or needle.

Examples of dressing classifications that are covered under the surgical dressing benefit include:

- foam dressings
- gauze
- nonimpregnated and impregnated dressings
- hydrocolloids
- alginates
- composites
- hydrogels.

A/B Medicare Administrative Contractors

A/B Medicare Administrative Contractors, or A/B MACs, are private organizations that carry out the administrative responsibilities of traditional Medicare for Part A and B. They are responsible for claims processing, or cutting checks to Medicare providers for their services; ensuring services are correctly coded and billed for, both before and after payment; deciding which healthcare services are medically necessary, and collecting overpayments. MACs follow the national coverage determinations set by the CMS, but in cases where there is no such determination or the rules are too vague regarding a specific procedure, an MAC may develop a LCD. The coverage polices for cellular and/or tissue products for wound (CTPs) are administered through these A/B MAC contractors in their local jurisdictions

within LCDs. In these policies, the coverage parameters can vary from one jurisdiction to another as can the title of the policy itself. The clinician would need to refer to each of the jurisdictions in order to understand whether CTPs are covered in their jurisdiction and what are the parameters for coverage. There are currently 10 jurisdictions (Table 2-3).

Table 2-3 The 10 AB Medicare Administrative Contractors

Jurisdiction E—Noridian Healthcare Solutions, serving American Samoa, California, Guam, Hawaii, Nevada, and Northern Mariana Islands
Jurisdiction F—Noridian Health Care Solutions serving Alaska, Washington, Oregon, Idaho, North and South Dakota, Montana, Wyoming, Utah, and Arizona
Jurisdiction G—National Government Services serving Minnesota, Wisconsin, Illinois, Kansas, Nebraska, Iowa, and Missouri
Jurisdiction H—Novitas Solutions serving Louisiana, Arkansas, Mississippi, Texas, Oklahoma, Colorado, and New Mexico
Jurisdiction I—Wisconsin Physician Services serving Kentucky, Ohio, Michigan, and Indiana
Jurisdiction J—Cahaba Government Benefits Administrators serving Alabama, Georgia, and Tennessee
Jurisdiction K—National Government Services serving New York, Connecticut, Massachusetts, Rhode Island, Vermont, New Hampshire, Maine
Jurisdiction L—Novitas Solutions serving Delaware, District of Columbia, Maryland, New Jersey, and Pennsylvania
Jurisdiction M—Palmetto Government Benefits Administrators serving North Carolina, South Carolina, Virginia, and West Virginia
Jurisdiction N—First Coast Service Options serving Florida, Puerto Rico, U.S. Virgin Islands

Payment

Payment refers to the methodology used to determine reimbursement to a healthcare provider or supplier. Payment may take the form of a global or bundled payment for the combination of services needed to treat a particular condition, as is the case with many hospital inpatient and outpatient discharges, or may be made on an itemized basis, as is the case for many

physician services such as office visits. In many cases, the payment method will be determined by the site of service rather than by the item or service itself.

As mentioned above, since wound care is provided in multiple settings, a later section on "How Reimbursement Works in Clinicians' Practice Settings" is devoted to the various healthcare settings and how wound care products and services are reimbursed by CMS.

Correct Documentation Is Key for Payment

Comprehensive documentation is the critical foundation for successful reimbursement of services and products. Physician documentation of pressure ulcers at the time of hospitalization is particularly important for identifying present on admission (POA) status. Regulatory agencies, independent of healthcare setting, set forth the requisite documentation for reimbursement, and their requirements for documentation should always be carefully reviewed prior to applying for coverage. Thorough documentation justifies the medical necessity of services and products and should reflect the care required in the prevention or treatment of wounds. In order to promote better documentation as well as the availability of data necessary for quality measurement, CMS is also promoting the "meaningful use" of electronic health records.[14] Specific financial incentives are available to providers using electronic health records and reporting data on quality of care (Table 2-4).

Table 2-4 Essential Wound Documentation

For essential wound care documentation, include the following:

- Change in clinical status or wound healing progress
- Characteristics of the wound, including:
  - Location
  - Length, width, and depth
  - Staging/category/classification
  - Exudate amount
  - Tissue type
  - Pain
- Local wound care and dressing selection
- Nutritional status
- Pressure redistribution/support surfaces (both bed and chair)
- Outcome and Assessment Information Set (OASIS-C) per schedule in home health care
- Minimum Data Set (MDS 3.0) per schedule in SNF
- Regular assessment and reassessment of the wound (such as daily or weekly)
- Repositioning schedule
- Routine daily skin assessment and care

How Reimbursement Works in Clinicians' Practice Settings

Hospital Inpatient

Hospital reimbursement is part of the inpatient prospective payment system (IPPS). Payments made under the IPPS totaled $120 billion and accounted for about 25% of Medicare spending in 2012.[15] The inpatient benefit covers beneficiaries for 90 days of care per episode of illness. There is a 60-day lifetime reserve. The episode of care begins when the Medicare beneficiary is admitted to the hospital and ends when he or she has been out of the hospital or an SNF for 60 consecutive days.[15]

Under the IPPS, the ICD-9, or soon to be ICD-10 CM, is used to track to an MS-DRG. Hospitals are reimbursed on a predetermined, lump-sum fixed rate for each MS-DRG. The payment amount for a particular service is derived based on the classification system of that service. For hospitals, this payment would also include all medical care, procedures, and surgeries, wound care products, devices, and support surfaces. Because the IPPS is based on an adjusted average payment rate, some cases will receive

payments in excess of cost (less than the billed charges), whereas others will receive payment that's less than cost.[16] The system is designed to give hospitals the incentive to manage operations more efficiently by evaluating those areas in which increased efficiencies can be instituted without affecting the quality of care and by treating a mix of patients to balance cost and payments.

CMS does review the MS-DRGs annually to ensure that each grouping continues to be grouped correctly with clinically similar conditions requiring similar resources. If their review concludes that clinically similar cases within an MS-DRG use significantly different amounts of resources, CMS will reassign them to different MS-DRGs or create a new MS-DRG. There are currently approximately 500 groups.

Rehabilitation hospitals and units and long-term care facilities (defined as those with an average length of stay of at least 25 days) are excluded from the PPS. Instead, they're paid on a reasonable-cost basis, subject to per-discharge limits.[16] They are also paid depending on hospital-specific contracts and different payer sources. Note that CMS doesn't recognize subacute status; rather, subacute facilities are governed by the SNF regulations.

The Deficit Reduction Act (DRA) of 2005 was passed in February 2006 in an effort to limit payments to hospitals for conditions resulting from potentially poor quality of care.[17] Section 5001(c) of the DRA requires the secretary of the Department of Health and Human Services or a designee to identify conditions that (1) are high cost, high volume, or both; (2) result in the assignment of a case to a DRG that has a higher payment when present as a secondary diagnosis; and (3) could reasonably have been prevented through the application of evidence-based guidelines. Section 5001(c) provides that CMS can revise the list of conditions from time to time, as long as it contains at least two conditions.

Stage 3 and 4 pressure ulcers and surgical site infections were identified as two of the initial hospital-acquired conditions (HACs) that met the DRA (Table 2-5). Thus, if clinicians do not identify and subsequently document the pressure ulcer(s) or specific surgical site infection as POA, then the hospital will not be permitted to claim payment either as a primary or secondary diagnosis. The POA Indicator requirement and HAC payment provision only apply to inpatient PPS hospitals. At this time, a number of hospitals are exempt from the POA Indicator and HAC payment provisions, including critical access hospitals, long-term care hospitals, cancer hospitals, children's inpatient facilities, and rural health clinics.

Table 2-5 The 10 Categories of Hospital-Acquired Conditions

1. Foreign object retained after surgery
2. Air embolism
3. Blood incompatibility
4. Stage III and IV pressure ulcers
5. Falls and trauma
6. Manifestations of poor glycemic control
7. Catheter-associated urinary tract infection
8. Vascular catheter–associated infection
9. Surgical site infection
10. Deep vein thrombosis/pulmonary embolism

The DRA also directed CMS to develop and standardize patient assessment information from acute and post–acute care settings. This resulted in the development of the Continuity Assessment Record and Evaluation (CARE) Tool. The CARE Tool measures the health and functional status of Medicare acute discharges as well as changes in severity and other outcomes for Medicare post–acute care patients while controlling for factors that affect outcomes, such as cognitive impairments and social and environmental factors. Many of the items are already collected in hospitals, SNFs, or home care settings, although the exact item form may be different. The tool is designed to eventually replace similar items on the existing Medicare assessment forms, including the Outcome and Assessment Information Set (OASIS), Minimum Data Set (MDS), Resident Assessment Protocol (RAP), and Inpatient Rehabilitation Facility–Patient Assessment Instrument (IRF-PAI) tools. (See Chapter 6, Wound Assessment, for more information about the CARE Tool.)

It is anticipated that use of the CARE Tool will help improve the quality of care transitions, leading to a reduction in inappropriate hospital readmissions. Four major domains are included in the tool: medical, functional, and cognitive impairments and social/environmental factors. These domains were chosen either to measure case mix severity differences within medical conditions or to predict outcomes such as discharge to home or community, rehospitalization, and changes in functional or medical status. Section G covers skin integrity and is used to assess pressure ulcers, delayed healing of surgical wounds, trauma-related wounds, diabetic foot ulcers, vascular ulcers (arterial and venous), and other wounds (e.g., incontinence-associated dermatitis).[18,19]

Hospital Outpatient Centers

The Balanced Budget Act of 1997 provided authority for CMS to develop a PPS under Medicare for hospital outpatient services. The new outpatient PPS took effect in August 2000.[20] All services paid under this PPS are placed into ambulatory payment classifications (APCs). A payment rate is established for each APC, depending on the services/procedures provided. CPT codes and modifiers identify clinic visits and services/procedures. The CPT codes track to an APC group based on the cost and the level of resources required to perform the service or procedure. Services or procedures in each APC are similar in cost. Since hospital outpatient claims are submitted to the Part B Medicare Administrative Contractor, the MAC pays a predetermined amount for the APC group—which includes all supplies including, but not limited to, wound care dressings. Beginning in 2013, CMS determined that cellular- and tissue-based products for wounds should be bundled. As a result, these products are now packaged into the facility fee for the procedure.

Hospitals may be paid for more than one APC per encounter. Medicare beneficiaries also can pay a coinsurance, which is the amount they will have to pay for services furnished in the hospital outpatient department after they have met the Medicare Part B deductible. A coinsurance amount is initially calculated for each APC based on 20% of the national median charge for services in the APCs. The coinsurance amount for an APC doesn't change until the amount becomes 20% of the total APC payment. It should be noted that the total APC payment and the portion paid as coinsurance amounts are adjusted to reflect geographic wage variations using the hospital wage index and assuming that the portion of the payment/coinsurance that's attributable to labor is 60%.

Skilled Nursing Facilities

A patient who is eligible for Medicare may receive Medicare Part A for up to 100 days per benefit period in an SNF.[21] The patient must satisfy specific rules in order to qualify for this benefit. These rules include the following:

- Beneficiary is admitted to SNF or to the SNF level of care in a swing-bed hospital within 30 days after the date of hospital discharge.
- Beneficiary must have been in a hospital receiving inpatient hospital services for at least 3 consecutive days (counting the day of admission but not the day of discharge).

- Beneficiary requires skilled nursing care by or under the supervision of a registered nurse or requires physical, occupational, or speech therapy that can only be provided in an inpatient setting.
- Services are needed on a daily basis.
- Skilled services are required for the same or related health problem that resulted in the hospitalization.

After the SNF accepts a patient with Medicare Part A, all routine, ancillary, and capital-related costs are covered in the PPS. Thus, wound care supplies, therapies, and support surfaces are included in the PPS per diem rate. The Balanced Budget Act of 1997 modified how payments were made for Medicare SNF services.[21] After July 1, 1998, SNFs were no longer paid on a reasonable cost basis or through low volume prospectively determined rates but rather on the basis of a PPS. The PPS payment rates are adjusted for case mix and geographic variation (urban vs. rural) in wages. The PPS also covers all costs of furnishing covered SNF services. The SNF isn't permitted to bill under Medicare Part B until the 100 days are in effect.[22]

All SNFs participating in Medicare and Medicaid must also comply with federal and state regulations. In November 2004, CMS released its revised interpretative guidance on pressure ulcers (Federal Tag 314).[23] F-314 is a federal regulation that states that a resident entering a long-term care facility will not get a pressure ulcer or if they have a pressure ulcer it will not worsen. This 40-page document is used by both federal and state surveyors to determine the SNF compliance with F-314. It also provides SNFs with evidence-based approaches to prevent and treat pressure ulcers. SNFs that are found to be noncompliant with the pressure ulcer regulation can receive civil money penalties, which currently range from $500 to $10,000 per day, or CMS and the state can withhold payments and close the facility because of system-wide imminent danger to residents. Additional skin or wound regulations include F-309, in which SNFs can be cited for all other ulcers besides pressure ulcers, and F-315, which addresses the need to protect the skin from the effects of urinary incontinence.

Resident Assessment Instrument

In order to meet its regulatory role, CMS requires that a Resident Assessment Instrument (RAI) be completed on all SNF residents. The RAI included the MDS 2.0 RAPs and utilization guidelines that have been in use

since 1995. The MDS is a 400-item assessment form that attempts to identify the functional capacity of residents in SNFs. Based on the MDS section, further assessments are triggered by RAPs, which assess common clinical problems found in SNFs, such as pressure ulcers and urinary incontinence. RAPs also have utilization guidelines that assist the healthcare team in planning the overall care of the resident. The comprehensive RAI is completed annually, with quarterly MDS assessments (less comprehensive) completed between the annual assessments. The SNF is required to do another RAI if the resident's health status changes significantly. Only pressure and stasis ulcers are clearly delineated on the MDS 2.0 version; all other ulcers are grouped in the "other" category. Section M of the MDS assesses ulcers by stage, type of ulcer (pressure or stasis), other skin lesions present, skin treatments, and foot problems.[24]

CMS has been using the new MDS version 3.0 since October 1, 2010.[25] This revised version is intended to improve reliability, accuracy, and usefulness; to include the resident in the assessment process; and to use standard protocols used in other settings. These improvements have profound implications for enhanced accuracy, which supports the primary legislative intent that the MDS be a tool to improve clinical assessment. The CMS has adapted the National Pressure Ulcer Assessment Panel's 2007 definition of a pressure ulcer as well as the staging categories of pressure ulcers. One of the new areas in section M (skin) has eliminated the confusion that requires staging of all chronic ulcers.[26,27] Staging of pressure ulcers will involve simply staging the deepest tissue involved and worsening pressure ulcers. Another major change has been the delineation between unstageable pressure ulcers and suspected deep tissue injury.

The RAI is a very useful instrument in planning the care of SNF residents. The RAI User's Manual Version 3.0 no longer uses RAPs to connect MDS data to care planning. Instead of RAPs, there are care area triggers (CATs) and care area assessments (CAAs). MDS 3.0 is tied to care planning first through the CAT grid, which triggers each CAA. Like the prior version, the MDS is only a preliminary screen that will identify potential issues that the interdisciplinary team will further explore. The interdisciplinary team should identify current clinical protocols and resources to guide the CAA, and these resources should be identifiable on request by surveyors.[26]

The CAA is therefore designed to expand the assessment process that begins with the MDS. One area that is beneficial from this expanded assessment is whether the ulcer was avoidable or unavoidable. MDS 3.0

section M does not address unavoidability, but this is an important issue that most if not all facilities would like to incorporate. The CAA allows the interdisciplinary team to identify specific guidelines that can be incorporated into the assessment and care planning process. Because the issue of unavoidability may depend on the presence of multiple comorbidities and physiological disturbances, collaboration with the physician will be an important component of this extended assessment.[26]

CAAs triggered by CATs in section M include pressure ulcers, nutritional status, and dehydration/fluid maintenance. The CAA for pressure ulcers is automatically triggered by any resident considered to be at risk, any stage of pressure ulcer, or any worsening ulcer. The net result of these changes is closer linkage of the resident assessment to quality of life, incorporation of updated guidelines for ulcer staging, and broadening of the care planning process to include current clinical protocols and evidence-based standards.[26]

Resource Utilization Groups

The RAI is also linked to payment.[28] All Medicare Part A payments are linked to the RAI, and, in some states, Medicaid payments are based solely on completion of the MDS. Based on the MDS, each resident is assigned to one of 66 resource utilization groups (RUGs). RUGs are clusters of nursing home residents based on resident characteristics that explain resource use.[29] The classification system includes 14 rehabilitation groups, 9 groups for days with rehabilitation and extensive services, 3 groups for extensive services, 16 groups for special care, and 10 groups for clinically complex care. Wound care is typically within the special care group.

RUG rates are computed separately for urban and rural areas, and a portion of the total rate is adjusted to reflect labor market conditions in each SNF's location. The daily rate for each RUG is calculated using the sum of three components:

- a fixed amount for routine services (such as room and board, linens, and administrative services)
- a variable amount for the expected intensity of therapy services
- a variable amount reflecting the intensity of nursing care that patients are expected to require.

Because of RUGs, it's essential for the SNF to complete the MDS correctly.

The SNF must pay close attention to all health problems of the resident because the more intensive the care required, the higher the daily rate will be. Moreover, completing the MDS accurately and in a timely manner will help to ensure correct payments. If an SNF doesn't complete the MDS in a timely manner, it receives a default payment, which is usually significantly lower, or it may not receive payment at all. An SNF is required to evaluate each patient on the 5th, 14th, 30th, 60th, and 90th day.

Home Health Agencies

The Balanced Budget Act of 1997 also called for the development and implementation of a PPS for Medicare home health services. On October 1, 2000, home health PPS was implemented[30] (Table 2-6). Beneficiaries receiving home health care are typically restricted to their homes, need skilled care on a part-time or intermittent basis, and are not required to make any co-payments for these services.

Table 2-6 Qualifying for Home Health Benefits

A patient who is Medicare eligible can also receive Medicare home health services. To qualify for this benefit, the patient must satisfy the following criteria:

- The patient's physician must first determine that medical care is needed in the home and thus generate a care plan.
- The patient must need at least one of the following:
  - intermittent physical therapy
  - intermittent skilled nursing care
  - intermittent speech/language therapy.
- The patient must be classified as homebound according to the condition for participation in Medicare.

Medicare purchases home health services in units of 60 days—or episodes of care. To capture the differences in expected resource use, patients receiving 5 or more visits are assigned to 1 of 153 home health resource groups (HHRGs) based on their clinical and functional status and service use as measured by the Outcome and Assessment Information Set (OASIS). Each of the 153 HHRGs is then subdivided into one of five categories. The first four categories are based on whether the episode is early or late as well as the number of therapy visits. The 5th category is for

those episodes that exceed 20 or more therapy visits and not impacted by episode timing. There is a base rate for the HHRGs, which is then adjusted based on geographic factors. The MACs pay the HHRG rate and will also pay a small additional amount for specific nonroutine supplies—such as surgical dressings. However, the HHRG payment does not include drugs, biologics, or DME—including but not limited to NPWT and pressure-relieving devices.

OASIS-C

The process of quality wound management begins on admission. Suggested components of a quality program are assessment (including risk assessment and intervention), documentation and wound measurement, case manager report and collaboration, protocols and physician orders, ulcer care, management of tissue loads, nutrition, and outcomes tracking.[31]

When it's determined that a Medicare patient can receive home health services, an OASIS form must be completed. OASIS is a group of comprehensive assessments that form the basis for delivering patient care, measuring patient outcomes for purposes of outcome-based quality improvement (OBQI), and, since 2000, assisting in the prospective payment system. Revisions to the OASIS tool introduced in late 2002 resulted in a 25% reduction in dataset questions. OASIS-C represents the most comprehensive revision to OASIS since its original release. This revised instrument, which was rolled out January 1, 2010,[32] better aligns measures in both the MDS 3.0 and the CARE Tool. Major items on the OASIS-C include sociodemographic, environmental, support system, health, and functional status. Based on these assessments, a care plan can be generated. The OASIS-C document specifically classifies stasis ulcers, surgical wounds, and pressure ulcers.[33]

OASIS-C includes data items to measure the use of "best practice" care processes. To that end, data elements were created to measure processes of care in 10 new domains, two of which focus on wound care:

- Pressure ulcer risk assessment, prevention measures, and use of moist healing principles (effective care and prevention)
- Diabetic foot care plan, education, and monitoring (disease specific: high risk, high volume, problem prone)

Payment for home health services is directly linked to the completion of OASIS-C. A case mix is also applied to calculate reimbursement. The case

mix involves 20 data points to assess three factors: clinical severity, functional status, and service utilization. The system has created 80 HHRGs.[34] Patients are grouped into the HHRGs based on the OASIS-C results.

Medicare pays home health agencies for each covered 60-day episode of care, and a patient can receive an unlimited number of medically necessary episodes of care. Payments cover skilled nursing and home health aide visits, covered therapy, medical social services, and routine and nonroutine supplies. For each 60-day episode, the payment system can vary, depending on the HHRG, with adjustments to reflect area wage differences.[34]

Home health agencies are required to transmit OASIS-C data electronically to their state system. Improper completion of OASIS-C can lead to significantly lower payments or no payments at all. Thus, accurate assessments and charting are essential for recouping payments. Some authors[31,35–37] have described innovative ways of teaching staff and ensuring their competency in completing OASIS-C.

Practice Point

Accurate completion of OASIS by clinicians is essential. If you don't answer the questions appropriately, accurately, and completely, your facility won't receive the money and will lose reimbursement.

Physician Offices/Qualified Health Practitioners

The Medicare program pays for physician and other qualified healthcare practitioner services based on the Medicare Part B Physician Fee Schedule. Under this schedule, physicians and other qualified healthcare practitioners are paid for each medically necessary (and documented) service and procedure they perform. This includes office visits, surgical procedures, and a broad range of other diagnostic and therapeutic services. The Physician Fee Schedule is a list of 7,400 unique covered services and their payment rates. All services—surgical and nonsurgical—are classified and reported to CMS on claims according to the HCPCS code. When

determining rates for each service, CMS considers such factors as the amount of work required to provide the service, practice expenses (the expenses related to maintaining a practice), and liability insurance. Through a calculation taking into account geographic variations, etc., Medicare pays the provider 80% of the fee schedule amount and the Medicare beneficiary is liable for the remaining 20%.

Providing Wound Care in a Complex Reimbursement Environment

The challenge of providing quality wound care can be magnified when the patient moves from one healthcare sector to another. That's why it's imperative for wound care professionals to understand some of the nuances of the reimbursement agency. A good illustration would be a Medicare beneficiary who was discharged home with a pressure ulcer that had 100% eschar covering the surface. In this scenario, the home care agency would receive no reimbursement for providing wound care until the eschar was removed. However, if the same Medicare beneficiary was discharged to an SNF, the nursing home could receive full CMS payment for the pressure ulcer with 100% eschar. This reimbursement schism can make providing quality wound care extremely challenging.

Quality Improvement Efforts

Regulations related to reimbursements are tightly integrated with efforts in quality assessment and improvement. Indeed, care that's found not to meet quality standards may not be reimbursed. Even appropriate care may not be reimbursed if the condition being treated is the result of a medical error. Moreover, claims for reimbursements for substandard care could be viewed as fraudulent and result in criminal penalties. CMS doesn't rely solely on such punitive methods and various other initiatives exist. Most of these efforts center on pressure ulcers, which may serve as a future model for other wounds.

The CMS strategy for pressure ulcers entails measurement, management, and eventually reduction. The specific approach employed, though, will vary depending on the healthcare setting.

Role of Quality Measurement

Measuring quality is central to ensuring quality care. If you don't measure

quality, you can't improve it. Facilitating such quality measurement is the wealth of data available in existing CMS databases, such as MDS 3.0 and OASIS-C, which provide patient-specific information on processes and outcomes of care. ICD-9-CM codes from hospital stays are also now much more informative. Since 2008, they describe pressure ulcer location, stage, and whether the wound was POA. These changes may address some of the problems that have been identified when using ICD-9-CM codes to measure rates of pressure ulcers in hospitals.[38]

Using these data sources, CMS is disseminating quality measures specific to different healthcare settings. In nursing homes, CMS reports two measures on its Nursing Home Compare Web site: the percent of short stay residents with pressure ulcers that are new or worsened and the percent of long stay high-risk residents with pressure ulcers. In home care, specific outcome measures such as a worsening number of pressure ulcers were initially developed. However, CMS currently reports on the Home Health Compare website only on several process measures including how often the home health team took doctor-ordered action to prevent pressure ulcers and how often the home health team included treatments to prevent pressure ulcers in the plan of care. The Hospital Compare website includes pressure ulcer rates among its complications of care. Using ICD-9-CM codes, rates are defined as number of patients with a stage 3, 4, or unstageable pressure ulcer as a secondary diagnosis, which was not POA, per 1,000 discharges.

While these measures of quality of care are widely available on CMS websites, their use has not been free of criticism. One issue has been the quality of the data, particularly ICD-9-CM codes. Pressure ulcer rates calculated from clinical data are 10-fold higher than rates based on ICD-9-CM codes.[39] Not surprisingly, the positive predictive value of the hospital pressure ulcer rate based on these ICD-9-CM code is limited, although this may be less of an issue since the implementation of the POA code.[40] A second issue is whether these rates should be adjusted for residents' risk of developing a new ulcer. Studies have shown that differences in facility performance mostly represent differences in resident/patient mix rather than in facility performance.[41] Finally, these rates may not capture true differences in performance. Nursing homes that performed well and poorly on the pressure ulcer measure were found to have few differences in how care was actually delivered.[42] Another study in 30 nursing homes found that a successful quality improvement effort was not associated with an improvement in the CMS quality measure, but there was a decline in the incidence of stage 3 or 4 ulcer.[41] This again suggests that the CMS quality

measures may not always capture differences in care performance.

Many other measures of wound care quality exist. Organizations such as the National Quality Forum will review these measures and determine whether they meet specific standards for reliability and validity. Those measures meeting these standards can be found on their website. One example is the Assessing Care of Vulnerable Elders (ACOVE) indicators for pressure ulcers, which consist of a set of 11 indicators that capture different aspects of pressure ulcer care.[43] Each indicator is structured as an *if … then* statement, where the *if* component specifies a specific situation and the *then* component indicates what should be done in that situation.

Managing and Improving Pressure Ulcer Care

There are at least four ways in which quality measurement can be used to improve care, and different healthcare settings employ different approaches.

First, quality measurement is being used to empower consumers of health care. The assumption is that patients and their families, if given information about quality of care, will select those providers offering the best care. Such information then needs to be made available to patients in a timely fashion. Further, providers need to proactively improve their care in order to attract patients. This approach is exemplified by the Home Health Compare and Nursing Home Compare websites maintained by CMS.[44] These sites contain not only facility rates of performance but also national and statewide rates to permit easy comparisons. To further facilitate use of this information by consumers, Nursing Home Compare employs a five-star rating system that combines information on these quality measures with results from state surveys and staffing levels. Whether this approach will indeed be successful in improving care, however, remains uncertain.[45]

Second, quality measures are being used in quality improvement activities. The systematic use of such data can aid in the identification of quality-of-care problems and help determine the nature of these problems.[46] Nearly all healthcare provider organizations are involved in continuous quality improvement activities, with varying levels of implementation into clinical practice. A central component of such activities is feedback on performance. Indeed, demonstration projects have suggested that providing home care agencies with performance feedback, a process known as OBQI, does result in reduced rates of hospitalization.

Third, quality measures may help to focus more detailed analyses of the care provided to individual patients. Patients flagged by the Hospital

Compare indicator may undergo a more detailed review of the care processes associated with the development and treatment of a pressure ulcer. In nursing homes, state survey agencies are required to conduct annual unannounced surveys at SNFs to determine compliance with federal regulations regarding quality of care. A major focus of these surveys is an evaluation of pressure ulcer prevention and treatment practices and whether the SNF is compliant with care as specified in F-314.[47] Cases reviewed are often identified based on the MDS quality indicators.

Finally, CMS is increasingly relying on pay for performance as an important way of using reimbursements to improve care. Providers delivering the best care will be reimbursed more than providers delivering poor-quality care. While in theory this should be a highly effective mechanism for quality improvement, the data to date, which do not involve wound care, have not been convincing.[48] Basic issues such as the appropriate dollar amount to incentivize care, whether pay for performance represents a reward or an agent of change, and how best to measure care have not been completely resolved. While a number of projects have evaluated pay for performance in hospital and ambulatory care settings, demonstration projects involving nursing homes are in early stages. The extent to which pay for performance will focus on wound care is uncertain.

Beyond these nonspecific approaches, CMS also actively promotes quality improvement activities directed toward Medicare beneficiaries. The primary mechanism for this is through quality improvement organizations (QIOs), formerly known as Peer Review Organizations (PROs). PROs initially relied on an "inspect and detect" approach to quality assessment in which medical record reviews would identify problems and be linked to interventions to correct substandard care. The approach was adversarial, penalties for substandard care could be harsh, and few improvements in quality of care could be documented.

In 1992, the Health Care Quality Improvement Initiative significantly changed the role of PROs. Rather than individual case reviews, PROs were to focus now on patterns of care. National guidelines, rather than local criteria, were to be used in evaluating quality of care. Most importantly, PROs were to work collaboratively with providers to improve healthcare delivery. Recognizing this new emphasis on quality improvement, PROs were renamed QIOs in 2001.

QIOs have since developed initiatives in diverse clinical areas and settings. In wound care, most of these efforts have again centered on pressure ulcers. In New York, toolkits have been developed with which hospitals can assess and improve their pressure ulcer prevention and

treatment practices. In nursing homes, QIOs from three states developed a strategy to train nursing home teams in quality improvement methods and proper pressure ulcer care. This training was reinforced through the use of outside mentors who regularly met with the teams. As a result of these initiatives and interventions, key processes of care improved dramatically.[49] A particularly impressive quality improvement collaboration within the New Jersey Hospital Association that involved over 150 hospitals and nursing homes resulted in reduction of more than 70% in pressure ulcer rates statewide.[50]

Government agencies other than CMS are also promoting initiatives to improve skin care. The Agency for Healthcare Research and Quality (AHRQ) has recently developed a toolkit for preventing pressure ulcers among hospital patients.[51] This toolkit is unique in the strong emphasis it places on evaluating organizational readiness for implementing changes in practice. For nursing homes, AHRQ has recently promoted a clinical decision support instrument that has been shown to lead to significant reductions in pressure ulcer incidence.[52]

Results of Improvement Efforts

Evidence is demonstrating that these efforts spearheaded by CMS and other healthcare organizations are having an impact on improving pressure ulcer preventive care. Many hospitals have significantly reduced their rates of pressure ulcer development. As one example, the Collaborative Alliance for Nursing Outcomes (CALNOC), a registry of 78 hospitals mostly in California, demonstrated a decline in hospital-acquired pressure ulcers from 10.4% to 1.8% between 2003 and 2010.[53]

Summary

Regulatory agencies play a major role in wound care. In March 2010, the Affordable Care Act became law. This law guarantees health insurance for a minimum of 35 million people and will have profound implications for wound care professionals. As the new law is implemented, new regulations will undoubtedly be developed and executed. With the increasing need to evaluate the cost-effectiveness of wound care, regulatory agencies will likely impose further regulations, which will lead to greater complexity in obtaining and maintaining reimbursements. Thus, the key to providing optimum wound care will depend on good documentation that clearly

articulates the need for services and products and clearly identifies assessment of the patient, interventions instituted, and outcomes achieved. When this is accomplished, the patient, the provider, and the regulatory agency all benefit.

PATIENT SCENARIO

Clinical Data

Mr. Y, a 72-year-old resident from a long-term care facility, is admitted to the hospital for treatment of pneumonia. He was receiving treatment for a stage III pressure ulcer on his sacrum at the long-term care facility. There is no documentation about the ulcer by the physician in the hospital admission medical record. The nursing admission record documents the presence of a stage III pressure ulcer on the sacrum. Mr. Y is treated successfully for his pneumonia and is returned to the long-term care facility.

Case Discussion

The financial implications regarding use of POA coding have been in effect since October 1, 2008. Under CMS ruling, the practitioner responsible for establishing the medical diagnosis needs to document the diagnosis on admission. In this case, the POA pressure ulcer was not documented by the physician; therefore, the hospital was poised to lose a higher amount of reimbursement for the DRG of a stage III pressure ulcer as a secondary diagnosis. The hospital coder noticed the difference between the physician and nursing documentation and queried the physician. Once it was established that the pressure ulcer was indeed POA, the physician completed his progress note and documented the location and stage of the ulcer. The coder could then submit this secondary diagnosis for billing.

Show What You Know

1. **Medicare Part B is a federal program that:**
   A. supports state programs to provide services and products to the

poor.

B. reimburses hospitals for wound care services.

C. reimburses for selected wound services and products in SNFs and home health agencies.

D. doesn't require co-payment from the beneficiary.

2. For which one of the following healthcare settings is completion of OASIS-C required?

A. Hospitals

B. Home health agencies

C. Hospital outpatient centers

D. SNFs

3. Which one of the following criteria must a patient with a wound meet in order to qualify for SNF care?

A. Skilled services must be required for the same or related health problem that resulted in the hospitalization.

B. The beneficiary must be in the hospital for 2 consecutive days.

C. Services are needed once per week.

D. The beneficiary must be admitted to the SNF within 90 days of admission to the hospital.

4. Which of the following approaches <u>is not</u> being used by CMS to improve the quality of care?

A. Empower consumers to select high-quality providers through the provision of information on performance.

B. Increase payments to providers of better care.

C. Develop computer reminders on when to turn patients.

D. Work with providers through regional QIOs.

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Legal Aspects of Wound Care 3

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Objectives

After completing this chapter, you'll be able to:

- explain the major litigation players and their roles in a lawsuit
- define the four elements of a malpractice claim
- describe the general rules for proper wound care charting
- indicate ways the medical record, standards, and guidelines can be used in a malpractice case
- describe documentation practices that predispose the medical record to legal risks
- describe strategies to improve consistency and accuracy of medical record documentation that minimize potential litigation risk.

The Current Climate

In recent years, the concept of patients as "consumers of health care" has risen to the forefront. Rather than blindly trusting clinicians, the consumer–patients of today are better educated, more aware of healthcare issues, and more willing to make use of legal resources when treatment goes awry. Although wound care generates no more litigation than many areas of healthcare practice, and arguably less than some others, the threat of litigation may affect the way clinicians approach the delivery of care.

Clinicians need to protect themselves while ensuring evidence-based, high-quality care to their consumer–patients. This chapter sets forth basic legal principles and suggests practice strategies that advance patient care *and* protect clinicians.

Litigation

During the course of human history, it became apparent that nonviolent means of settling disputes must be developed. The law and the legal process, including litigation, continue to be one of civilized society's experiments at achieving nonviolent resolutions to disputes. The success of this experiment is itself the source of much dispute, to which no resolution (nonviolent or otherwise) is currently in sight.

Contrary to television and film portrayals, the real-life litigation process is arduous and time-consuming. While fictitious television and film lawsuits resolve in a matter of weeks or months, usually ending with a dramatic trial resulting in a stunning jury verdict, most real-life cases take years to get through the legal system. In some jurisdictions with crowded dockets, they can take as long as 5 years to resolve. Those that require appeals can take considerably more time before all issues are finally put to rest. Trials (dramatic or not) are few and far between, as nearly all lawsuits are settled before trial. When trials do happen, they're usually slow-moving, uninteresting events that tax the patience and attention of jurors. Litigants expecting "Perry Mason" moments from their attorneys are sure to be disappointed, and, as anyone who has ever served on a jury knows, closing arguments by attorneys are never, ever over in the 5 minutes before the final commercial.

Despite the difficulties and drawbacks, the litigation process does afford citizens an impartial forum for dispute resolution grounded in the law. And the law, as Plato stated, is "a pledge that citizens of a state will do justice to one another."

The discussion in this chapter is limited to *civil litigation,* that is, litigation in which citizens have a dispute with each other—rather than *criminal litigation,* in which the state or a government seeks to prosecute a party for the violation of law. There are significant differences between the two forms of litigation (e.g., standards of proof). The remedy sought in civil litigation is monetary damages. In contrast, only the prosecuting state or government may seek to deprive the alleged lawbreaker of his or her liberty by incarceration.

How Is a Medical Malpractice Lawsuit Born?

Litigation begins the moment a person believes he or she has been wronged

by another and seeks the advice and counsel of an attorney in an effort to "right the wrong" or "get justice." During the initial interview between the prospective client and the attorney, the attorney makes a number of preliminary judgments usually based solely on the client's presentation:

- Is this the type of case the attorney is capable of handling? Does it fall within his or her expertise and practice experience? Does the attorney have the time to handle the matter?
- Is the client's story credible?
- Will the client make a good witness?
- Are the damages, if proven, sufficient to warrant entering into the litigation process?
- Is there a party responsible (liable) for the client's injuries?
- How likely is it that both liability and damages can be proven?
- Are there any glaring problems or difficulties with the case?

If the answers to these questions are satisfactory and the client wishes to retain the attorney, a lawsuit has then been conceived.

Before filing the legal documents that start the litigation process in a medical malpractice case, most attorneys perform an intensive investigation in order to definitively answer questions concerning liability and damages. Medical records and other information must be obtained and examined by an expert to determine whether a malpractice claim can be made. Information related to the identities of potential defendants must be analyzed, and strategic legal issues related to jurisdiction (which court can the case be brought in) must be thought through. If after this investigation the attorney still believes the case has merit, legal papers starting the actual lawsuit will be filed, and a lawsuit will be born (Box 3-1).

Box 3-1 Players in the Litigation Process

The litigation process is initiated and enacted by people with a dispute to resolve and those whose task it is to aid in resolving that dispute.

The Parties

The principal parties involved in litigation are the *litigants*—the individuals on either side of the dispute. The *plaintiff* is the person who initiates the lawsuit and who claims he or she has

suffered injury due to the actions of another. A lawsuit may be filed by multiple plaintiffs.

The plaintiff sues the *defendant*—the person or organization alleged to have injured the plaintiff by his/her or its actions. In most cases, the parties are individuals, but parties can be corporations, companies, partnerships, government agencies, or, in some cases, governments themselves.

The Judge

The *judge* is an individual, usually an attorney, who has been appointed or elected to oversee lawsuits on behalf of the state or government under whose jurisdiction the lawsuit is brought. The judge acts as referee during the pretrial phase of the case and decides legal issues that arise as the lawsuit progresses toward trial. In a trial, the judge's responsibility is *to interpret the law.*

The Jury

The *jury* is a panel of citizens chosen by the attorneys for the litigants to hear evidence in the case and render a decision or verdict. The jury's responsibility is *to determine the facts* in a trial. It's up to the jury to decide whether the plaintiff and his or her attorney proved their case, thereby rendering a decision about the defendant's liability and the amount of damages the defendant should pay to the plaintiff.

The Pretrial Litigation Process

The pretrial litigation process consists of several steps: complaint and answer, discovery, and motion practice.

Complaint and Answer

The initial legal paper that gives rise to a lawsuit is called the *complaint.* While procedural requirements vary between jurisdictions, generally the complaint is a document that sets out the claims made by the plaintiff against the defendant, the basis of the jurisdiction of the court, the legal theories under which the plaintiff is making the claims, and, in some jurisdictions, the amount of damages claimed.

The defendant must then file an *answer* within the permitted time that

responds on a count-by-count basis to the plaintiff's complaint and that, depending again on jurisdictional rules, may also include claims against the plaintiff. These two basic *pleadings* initiate the formal lawsuit.

Discovery

Discovery is the process by which the parties find out the facts about each other, about the incidents that have given rise to the claims of malpractice alleged by the plaintiff, and the defenses to those claims asserted by the defendant. The law has provided discovery devices—procedural mechanisms by which the parties ask for and receive information. Demands are routinely made for documents and other tangible items related to the lawsuit's claims, for statements made by the parties to others, and for the identification of witnesses to the incidents. Then, pretrial testimony (*deposition*) is taken of the parties to the lawsuit. This testimony, while out of court, is sworn testimony transcribed by a certified court reporter and can be used for any purpose in the lawsuit, including for purposes of *impeachment*—the demonstration of prior untruthful or inaccurate testimony, or a challenge to the credibility of a witness—at trial.

Finally, *expert discovery*—information about the opinions of experts retained by the parties—is usually permitted. Experts are individuals accepted by the court to assist the finder of fact—the jury—in understanding issues that commonly fall outside of the experience of the typical juror. In medical malpractice cases, the plaintiff must prove that there was a deviation from the standard of care that resulted in an injury. Expert testimony related to the field of medicine, treatments, and standards of care at issue in the case is essential to successfully meet proof requirements for each element of a malpractice claim brought by a plaintiff. Likewise, the defense of such claims requires opposing expert testimony—in essence, an explanation by a credentialed individual supporting the actions taken by the defendant from which the claim of malpractice stems.

Motion Practice

Disputes over discovery often arise in the context of a lawsuit, and those disputes that can't be resolved by the parties require court intervention. Formal resolution of these disputes usually requires an application to the court—a *motion*—setting forth the dispute and the position of the party making the application (the moving party, or *movant*) and requesting certain *relief* or results to be *ordered* by the court. Naturally, this requires a

response from the other party—the *opposition*—that sets out the reasons why the court shouldn't grant the relief requested.

Some motions can be decided by the court *on the papers*, that is, without a formal oral presentation (*oral argument*) by the parties before the judge is assigned. More complicated motions, especially those seeking to eliminate or modify legal claims, almost always require argument before the presiding judge or court.

The Trial

While the vast majority of lawsuits settle before trial ("out-of-court settlements"), some cases do proceed to trial. Medical malpractice trials are almost without exception jury trials. Once it's determined that settlement isn't an option, a trial date is set and the attorneys begin to prepare. In federal jurisdictions and many state courts, litigants are required to prepare pretrial statements and submissions. They also disclose exhibit lists (materials and documents the attorneys anticipate they will use at trial). They may also designate deposition testimony to be read or, if the testimony was videotaped, to be shown at trial. The pretrial submission and disclosure process helps to ensure that the trial is as fair as possible and eliminates the possibility of "trial by ambush." Thus, the "Perry Mason" moments of television and film renown are relatively few and far between.

On the day of the trial, the attorneys for the parties proceed with jury selection. Each attorney tries to select jurors that he or she believes will decide in favor of (*find for*) his or her client. Procedurally, the jury selection process varies widely by jurisdiction. In some courts, the trial judge will take an active role by questioning the jurors. The fight over selection is then left to the attorneys. Other jurisdictions permit the attorneys to question jurors directly without court supervision and the trial judge becomes involved only when a dispute arises. As you can imagine, jury selection in a jurisdiction with strong judicial control is a much briefer process than in those jurisdictions where the attorneys are left to their own devices. No matter what the individual procedure, once the jury is chosen (*empanelled*), the trial begins.

At trial, the parties each give an opening statement, one of the two times in the entire trial that the attorneys are permitted to speak directly to the jurors. After opening statements, the plaintiff's attorney presents the plaintiff's case. As the burden of proof is on the plaintiff, the plaintiff's attorney goes first. After the plaintiff's direct case is finished, the plaintiff "rests," and the defendant's attorney presents the defendant's case. The

direct case consists of evidence (testimony, documents, etc.) presented by the attorney for a party. The party has the right to cross-examine each witness after the direct examination, and then additional examination may follow ("redirect" and "recross") as necessary. After all the evidence has been presented by both sides, the parties make closing statements (*summations*), which is the last time the attorneys are permitted to speak directly to the jurors.

Once summations are completed, the judge then instructs the jurors on the appropriate law that they're to apply to the facts of the case. Remember that the jury is the *finder of fact*—it determines what happened, when it happened, who did it, where it happened, and how it happened—and the judge is the *interpreter of the law.* After the jurors receive the judge's instructions, they leave the courtroom and begin deliberations.

Every trial attorney hopes to be lucky enough to serve on a jury that goes to deliberations. For trial lawyers, understanding what happens inside the jury room during deliberations is the Holy Grail of trial practice. In jurisdictions that permit attorneys to interview jurors after verdict, attorneys often spend many hours with the jurors who are willing to discuss the case in order to determine what did—and what didn't—work during the trial. It's often surprising to find that what the lawyer thought was of prime importance wasn't so important to the jury. The jury room in our legal system is sacrosanct, and, no matter how it happens, the jury will arrive at a verdict that will be delivered to the parties in open court. Once the verdict is read and the jury excused, the trial is over.

Appeals

Each jurisdiction has an appellate process, of which the litigants may take advantage. Depending on the jurisdiction, appeals may add years (and many dollars) to the resolution of claims and lawsuits.

Legal Elements of a Malpractice Claim

A medical malpractice claim is made up of four distinct elements, each of which must be proven to the applicable standard of proof in the jurisdiction of the case. The usual standard of proof for civil cases is a *preponderance of the evidence.* The preponderance standard can be best described as a set of scales that represent the plaintiff on one side and the defendant on the other, which are evenly balanced at the start. The party that wins is the one on the side of the scale that dips lower at the end of the trial. In other words,

in order to prevail, plaintiffs need to show by only 50.0000001%—just a bit more than one-half—that they've proven each of the elements that make up a malpractice claim.

The four general elements that make up a malpractice claim are:

- existence of a duty owed to the plaintiff by the defendant
- breach of that duty
- an injury that is causally related to that breach of duty
- damages flowing from that injury that are recognized by law.

Duty

In general, there is no duty to protect a person endangered by the actions or omissions of another if there is no special relationship between the two persons. The patient–physician relationship is the basis for the claim of duty between the plaintiff–patient and the defendant–healthcare professional in medical malpractice cases because that relationship permits the patient to rely on the physician's knowledge, expertise, and skill in treatment. Thus, the allegations of medical negligence arise within the course of that professional relationship. Translating that definition into healthcare terms, some examples of a duty may be the obligation of a healthcare practitioner to give patients care that is:

- consistent with the level of his or her experience, education, and training
- permitted under the applicable state practice act
- authorized or permitted under the policies and procedures of the institution that are applicable to the position.

Practice Point

Duty: In negligence cases, *duty* may be defined as obligation, to which the law will give recognition and effect, to conform to a particular standard of conduct toward another. The word *duty* is used in the law to denote the fact that the actor is required to conduct himself in a particular manner at the risk that if he doesn't do so, he becomes liable to another to whom the duty is owed for any injury sustained by such person, of which that actor's conduct

is a legal cause. (Restatement, Second, Torts, Section 4)[1]

Breach of Duty

In addition to proving the existence of a duty, the plaintiff must also prove the defendant breached that duty. Breach of duty can result from commission, omission, or both. Most often, to establish this element of the claim, the plaintiff in a medical malpractice case must also show that the defendant healthcare practitioner deviated from an accepted standard of care or treatment. The practitioner isn't required to provide the highest degree of care, but only the level and type of care rendered by the *average practitioner*. What the standard of care is, and whether and how it was deviated from, must be established for the jury, and this is most often the province of expert testimony.

Breach of duty in the healthcare setting may be illustrated in the following ways:

- failure to give care within the applicable practice act
- failure to perform professional duties with the degree of skill mandated by the applicable practice act
- failure to provide care for which the circumstance of the patient's condition warrants.

Practice Point

Breach: The failure to meet an obligation to another person that's owed to that person; the breaking or violating of a law, right, obligation, engagement, or duty by commission, omission, or both.[1]

Injury Causally Related to a Breach of Duty

In a medical malpractice case, proof of an injury isn't enough unless that injury can be causally linked to a breach of duty by a healthcare practitioner. That breach of duty is then considered the proximate cause. Without the breach of duty, the injury wouldn't have occurred (Box 3-2).

Box 3-2 Proving Proximate Cause

While standards of proof related to proximate cause may vary among jurisdictions, one of two questions is almost always used to determine this issue:

- Was the healthcare practitioner's negligent conduct a "substantial factor" in causing the injury?
- Would the injury not have happened if the healthcare practitioner hadn't been negligent?

Proximate cause in the healthcare setting can be illustrated by the following examples:

- fractured hip due to a fall because of failure to raise the side rails of the bed
- decreased total protein due to failure to provide nutrition (either failure to provide actual nourishment or failure to order/call a consult)
- osteomyelitis resulting in limb amputation following failure to attain/call an infectious disease consult and provide antibiotic therapy.

Practice Point

Proximate cause: That which, in a natural and continuous sequence, unbroken by any efficient intervening cause, produces injury, and without which the result wouldn't have occurred and without which the accident couldn't have happened, if the injury be one that might be reasonably anticipated or foreseen as a natural consequence of the wrongful act.[1]

Damages

Finally, the fourth element that makes up a malpractice claim is damages. A healthcare practitioner may be held liable for damages when the jury finds that the practitioner deviated from the applicable standard of care in treating the plaintiff–patient and, as a result, caused injury resulting in legally

recognized damages. In most jurisdictions, a plaintiff may recover for proven monetary losses (lost wages and unreimbursed medical expenses) and for pain and suffering that result from the proven injury. As noted previously, it's the jury—the finder of fact—that sets the monetary award to the plaintiff.

Practice Point

Damage: Loss, injury, or deterioration caused by the negligence, design, or accident of one person or another, with respect to the latter's person or property.

Damages: A pecuniary compensation or indemnity that may be recovered in the courts by any person who has suffered loss, detriment, or injury, whether to his or her person, property, or rights, through the unlawful act or omission or negligence of another.[1]

As we have shown, in order for a plaintiff to prevail in a medical malpractice claim, all *four of the elements* discussed above must be satisfied. Three of four won't do. They must score perfectly on all four to prevail before a jury.

The Medical Record in Litigation

The medical record is arguably the single most important piece of evidence in a medical malpractice case. It serves as a crucial tool for the delivery of science-based care. It is also:

- a legal document
- a communication tool
- the supporting basis for treatment decisions and modifications
- one of the primary tools for the evaluation of treatment modalities.

At one time or another in the education of a healthcare practitioner, whatever the specialty or discipline, this directive is taught: "If it wasn't written down, it didn't happen." Nowhere does this statement ring more true than in a medical malpractice case (Box 3-3). Before we consider the role documentation plays in the medicolegal world, let's first consider for a

moment how important the medical record is in the care and treatment of patients.

Box 3-3 Effects of Incomplete Charting

What happens when charting is incomplete? In addition to providing a poor medical record of a patient's care to help jog the practitioner's memory if a lawsuit occurs, it can create other problems. Competent attorneys can create havoc when gaps exist in the record. Nothing makes proving the plaintiff's case easier than such gaps, especially near or around the time of the alleged malpractice if the claim revolves around a single incident. If the claim concerns a continuous or extended course of treatment, the absence of documentation related to treatment outcomes, observations, and the basis for the treatment is strong evidence of negligence. Where the record contains gaps, you can be certain that the plaintiff's attorney will be happy to suggest to a jury what happened during those undocumented times, and those suggestions won't be of benefit to the healthcare facility or the individual practitioner.

The medical record serves several purposes. First and foremost, it is a communication tool that allows real-time coordination of care by multiple disciplines. It also acts as a historical record to determine the efficacy of past interventions and guide future care. The medical record is also a factual record utilized in lawsuits to determine the quality of care rendered, the occurrence of physical harm, and other legal issues. Most state boards (nursing, medical, and other licensed healthcare professionals) and federal regulatory agencies require "timely and accurate" documentation of findings in the medical record. Due to the wide range of specialty care areas and ever-changing rules, regulations, and laws, state and federal boards offer little guidance on *how* to meet this documentation standard. It's left to the individual facility or provider to determine the appropriate standards. The absence of standards has resulted in a wide range of documentation practices.

Communication Tool

The medical record is the primary method of communication between

members of the healthcare team. Oral report and rounding are essential communication devices, but it's impractical and unrealistic to expect that every healthcare team member be present during report or rounds. Such disciplines as physical therapy, occupational therapy, and respiratory therapy may not be present for report or rounds. The myriad medical specialists available to the primary physician (e.g., infectious disease consultants) are also rarely present during rounds, yet it's imperative for the delivery of good science-based care that every healthcare team member have the most current and up-to-date patient information. The medical record is the only way to accomplish this. It's available 24 hours per day to any practitioner who can utilize it to stay informed about the patient's progress.

Treatment Evaluation and Support

Documenting patient treatment outcomes and responses in the medical record is a key method for evaluating treatment modalities and therapies. The typical patient with pressure ulcers will undergo an extended course of treatment that will change over the course of time. In order to establish a basis for treatment and modification, there must be well-documented observations and evaluations of the patient. Upon initiation of treatment, careful observation and documentation of the patient's condition is critical in order to establish a baseline for initial treatment and care. Without a carefully documented record, treatment, evaluation, patient outcomes, and treatment modifications are impossible to justify in court (Box 3-4).

Box 3-4 General Documentation Guidelines

Listed here are some general rules for documentation that serve your patient's needs and can help in the defense of a lawsuit.

- Be thorough—record the date and time for each entry.
- Be accurate—use units of measure instead of estimates (e.g., "patient had a 6-oz cup of ice chips" instead of "patient had some ice chips").
- Be factual—think of yourself as a newspaper reporter and answer the following questions: who, what, when, where, why, and how.

- Be objective—record only the facts. Remember that you're communicating information that others will rely on. If your patient is to benefit from your professional training, judgment, and observational skills, your colleagues must have objective, factual information to rely upon.
- Select appropriate descriptors and drop down menus when using computerized documentation systems; follow facility policy regarding system/computer failure for documentation. Write legibly for manual records, and print if necessary.
- Only use approved abbreviations.
- Make contemporaneous entries—finish your documentation before you leave work for the day. Don't add notations days later unless your facility permits such additions—and even then, adhere strictly to your facility's policy governing such additions.
- Be truthful—don't fake, misrepresent, exaggerate, or misstate the facts in the medical record.
- Most importantly, don't assign blame. While it's important to relate the facts completely and accurately, assigning blame in the medical record is fodder for malpractice actions and does nothing to advance the care of your patients.

Practice Point

Accurate and complete patient outcomes and responses to treatment and care must be documented in the record, as they're the basis for care decisions and legal defense.

Legal Aspects of Wound Documentation

Wound assessments (see Chapter 6, Wound Assessment) are some of the most detailed and time-consuming documentation a healthcare provider will perform. Radiologists can view internal organs with a variety of internal imaging techniques and generate detailed, consistent reports. Myriad laboratory values are also available to monitor internal organ system functions. Wound assessment and documentation is still mostly a subjective,

visual, pen-and-paper/computerized exercise that requires a good base of knowledge to perform accurately. Wound assessment and monitoring are typically left to the staff nurse or wound specialist. Wounds require an intricate, multifaceted assessment of their many attributes. Different levels of knowledge among caregivers can result in inaccurate, inconsistent, and erroneous wound documentation. Multiple areas of documentation for wound issues can make quick access to this information difficult. Multiple wounds on a single patient add even more of a documentation burden. Such complexity can lead to inconsistent documentation—and treatment—and may leave a provider or facility open to legal liability.

The American Nurses Association[2] has clarified that nurses are expected to record their assessments and diagnoses of the patient's skin integrity in the medical record. Staging of pressure ulcers and differentiating them from other wounds is within the scope of nursing practice.

We recommend the following processes and procedures to improve the consistency and documentation of care for pressure ulcers, acute and chronic wounds, or any other untoward event that occurs while a patient is under care. These recommendations are not designed to promulgate or establish a particular standard of care for wound documentation but rather to make providers aware of the common difficulties that may occur with wound documentation and to propose solutions.

The Flow of Information

To find the latest laboratory value in a chart, you go to the lab section. To find the latest chest x-ray result, you go to the radiology section. To find the latest wound description, you go to . . .? Wound documentation is often found scattered throughout the chart in activity of daily living (ADL) forms, nursing assessments, narrative notes, wound assessment forms, and many other sections. The organization and composition of the medical record in any given facility often evolves over the years with no real appreciation for how one would look at the chart globally. This is especially true of nursing documentation forms as they are added to and modified over time. In such situations, duplication of information becomes increasingly common. Consistent documentation by nursing staff then becomes much more difficult to provide and to review at a later date.

In order to evaluate the effectiveness of the medical record as a communication tool, one should critically examine from an outsider's perspective just how information flows within the record. A medicolegal reviewer attempting to determine if care was accurately and consistently

provided can sometimes be stymied by a poorly structured chart whose organization makes sense to the facility—yet to no one else. Information that cannot be found readily (drop-down screens) will often be ignored. If the appropriate form or the proper location in the chart cannot be found easily, certain information may not be documented. If wound care documentation can take place in multiple areas of the chart, it may be documented multiple times—or not at all—because of the uncertainty as to where the information should be properly entered. The medical record is a documentation system. If documentation is inconsistent, a *systems approach* may be applied in order to evaluate and improve the structure and flow of documentation, rather than actually getting staff to just "document more."

Admission Assessment

Both the medical and nursing admission assessments provide a "snapshot" of the patient's status at the time of admission. The admission assessment is an area where one cannot overdocument. The more pertinent information documented about the patient at the time of admission, the better informed the healthcare team will be, and thus, decisions can be made based on the best information available. Discovery and description of any lesion during admission assessment are critical in determining the course of care and, in a lawsuit, in determining the ultimate liability for any wounds that develop or deteriorate during the patient's stay. Preexisting lesions should be documented carefully and thoroughly with regard to size, location, and characteristics. A detailed description of the wound is more important than an actual wound diagnosis during admission assessment. The chart should reflect what interventions were taken and who was notified of the existence of the wound and any other findings.

Since pressure ulcers are a frequent reason for litigation in health care, they will be highlighted as a key exemplar in the remainder of this chapter. A general rule often heard in nursing and wound care circles is that any pressure ulcer that develops after 24 hours of admission is considered to be acquired at the facility rather than inherited. However, the definition of suspected deep tissue injury (DTI), a deep tissue discoloration under intact skin or a blood-filled blister that may ultimately evolve to a full-thickness lesion—as further defined in 2009 by the National Pressure Ulcer Advisory Panel (NPUAP)–European Pressure Ulcer Advisory Panel (EPUAP)[3] Pressure Ulcer Prevention and Treatment guidelines—makes this general rule difficult to defend. For example, the patient may be admitted with an inconspicuous-looking skin discoloration in the sacral area or other bony

prominence that ultimately evolves into a full-thickness pressure ulcer. Emerging research supports the process that tissue ischemia and necrosis can take several days to become visible to the naked eye when all that was observed and documented was a skin discoloration.[4,5] Therefore, nursing, medical, and all license healthcare professional staff should be made aware of this NPUAP-EPUAP definition[3] and incorporate it into their wound assessments. (For more information, see Chapter 13, Pressure Ulcers.)

Pressure Ulcer Risk Assessment

All patients should be assessed for pressure ulcer risk. A pressure ulcer risk assessment can include any validated scale, such as the Braden or Norton scale. Risk scales are tools that quantify risk factors associated with pressure ulcer development, such as nutrition, moisture, and mobility, among others. A pressure ulcer risk assessment scale can provide detailed insight into the care needs of the patient far beyond skin protection. The frequency of risk assessment is open to debate and is based primarily on the guidelines or custom of the individual nursing unit.

Ideally, every patient admitted to a healthcare facility should be assessed upon admission to identify individuals at risk for pressure ulcer development. Those at risk should then have routine follow-up assessments during their stay. A risk assessment is also recommended when the patient is transferred to another unit or whenever there is a significant change in the patient's condition. The nursing admission and/or daily assessment is a logical chart area in which to document risk assessment. The most important aspect with any risk assessment is: What is done with the information? Validated risk assessment tools are powerful and accurate predictors of pressure ulcer development but are useless if the information they provide is not acted upon. Each risk factor is ideally suited as an individual plan to prevent, mitigate, or improve a decline in the level of functioning.

Pressure Ulcer Development

Sometimes, the underlying problems that result in the development of pressure ulcers can be managed, healed, or avoided altogether. In other instances, the disease burden can be so great that ulcers will occur or fail to heal despite the best of care. Indeed, the Centers for Medicare and Medicaid Services (CMS) recognizes that pressure ulcers are unavoidable in long-term care if the facility had (1) evaluated the resident's clinical condition and pressure ulcer risk factors; (2) defined and implemented

interventions that are consistent with resident needs, goals, and recognized standards of practice; (3) monitored and evaluated the impact of the interventions; and (4) revised the approaches as appropriate.[6] In 2010, the NPUAP hosted a consensus conference and modified this definition to make it applicable to other care settings. The revised definition of "unavoidable" in reference to pressure ulcer development is "that the individual developed a pressure ulcer even though the provider had evaluated the individual's clinical condition and pressure ulcer risk factors; defined and implemented interventions that are consistent with individual needs, goals and recognized standards of practice; monitored and evaluated the impact of the interventions; and revised the approaches as appropriate."[7] In 2014, the NPUAP had another consensus conference that affirmed this definition and reached consensus on several clinical conditions that support pressure ulcer being unavoidable.[8]

Any documentation system regarding pressure ulcer prevention should be able to clearly and efficiently outline these criteria.

Practice Point

Consider the following NPUAP consensus conference statement that there are patient situations that could lead to unavoidable pressure ulcers:[7,8]

- skin failure
- hemodynamic instability that may preclude turning or repositioning
- patient refusal to reposition.

Nursing units where the disease burden of patients is extremely high include intensive care units (ICUs), long-term care units, and hospice. For ICU patients, frequent and consistent monitoring should be performed on the high-risk areas of the sacrum, heel and trochanter, and the occipital area. We frequently find in chart reviews that dynamic, pressure-distributing mattresses are obtained *after* the development of a pressure ulcer. While this may be a logical and justifiable escalation of care and intervention for a general medical–surgical population, it's important that special emphasis be

placed on these high-risk populations.

The Infamous "Turn Q 2" Check Box

Many nursing patient care flow sheets have "Turn Q 2" (turn every 2 hours) on their checklist of pressure ulcer prevention strategies. The presence or lack of a check in this box on these flow sheets is often used by attorneys to undermine or paint a negative picture of the quality and consistency of care delivered by nursing staff. The origins of the requirement to reposition patients every 2 hours for pressure ulcer prevention are obscure and not well grounded in science. The 1994 Agency for Health Care Policy and Research (AHCPR) guidelines specifically recommended repositioning every 2 hours.[9] Such an absolute time requirement for repositioning or other interventions does not permit individual clinical judgment. Patient care should be based on the dynamic evaluation of a patient's status by qualified personnel and not on a single, fixed point. Some patients may require more frequent repositioning and some less as a result of the use of a pressure-distributing mattress[10,11] or the need for uninterrupted sleep. Some may be unable to turn due to critical illness. Some may have undergone diagnostic procedures, thereby precluding staff from attending to them every 2 hours. There are simply too many variables that determine when and how a patient is positioned to require a rigid timetable that likely bears little or no resemblance to the patient's actual needs. The NPUAP-EPUAP Prevention and Treatment Guidelines state that repositioning frequency should be influenced by the support surface used (Strength of evidence = A).[3]

We recommend removing the "Turn Q 2" check box from nursing forms, admission order templates, pressure ulcer prevention orders, and other areas. The check box could be replaced with a statement such as "reposition according to patient needs as determined by pressure ulcer risk assessment" or some other language determined to better meet patient care needs and risk management requirements for proper documentation. We note, however, that this more flexible standard requires a more rigorous approach to documentation of the actions taken by healthcare personnel. Many staff members are accustomed to the checkoff system and may fail to adequately document interventions without such a system. Management should consider training and monitoring to ensure compliance with documentation standards. This new method connects the risk assessment to the intervention and allows much more flexibility for staff to deliver timely and effective care rather than basing care on a single number.

Discovery of a Pressure Ulcer

The initial discovery of a pressure ulcer is typically documented in the nursing or interdisciplinary progress record section. Any ongoing assessments (and actions, including notification of appropriate medical personnel) should then be documented per facility policy in a wound care form, ADL form, or progress notes, but preferably in just one place in the chart for easy access. The response to the discovery of the lesion is just as critical as documenting the lesion itself. Documentation should include what immediate interventions were taken; who was notified (the charge nurse/nurse manager, the incoming staff, the physician, the wound care specialist, and/or the family); what topical care was provided to the lesion; and any actions that were taken to minimize further pressure/shear (e.g., redistribution of mattress/beds, heel suspension boots, lowering the head of bed, repositioning). Such documentation demonstrates that your facility has a system in place to act quickly and appropriately to changes in the patient's condition.

Correct Identification of the Pressure Ulcer

The etiology of the lesion must be correctly identified in order to provide the most appropriate and effective care. Examine the wound for yourself, review the patient's medical history, make your own judgment, and if it differs with others, discuss your concerns with the team. When in doubt about the etiology or progress of a wound, don't make a speculation in the chart; simply document what you observed. Remember, *objective description beats subjective guessing every time*.

Differentiating between a pressure ulcer and an ischemic ulcer in the lower extremity can be particularly difficult. For example, is the development of a wound on the lateral aspect of the foot in a person with peripheral vascular disease the result of pressure or arterial insufficiency? The argument could be that "but for" the pressure, the lesion would not have occurred. The counterargument could be that "but for" the arterial insufficiency, the tissue could have easily tolerated the pressure exerted on the foot. Objective data are required to solve this dispute. In this case, formal vascular laboratory studies are needed to determine the extent of ischemia and the avoidability of such lesions. Most chronic wounds have distinctive locations, sizes, and presentations and can be easily differentiated by trained personnel. However, some wounds will defy easy categorization or diagnosis. All lesions require as much objective data as

possible to establish the correct diagnosis and appropriate care plan.

Notification and Participation of the Physician

The patient's primary physician must be notified of any untoward event in a timely manner, including development of a pressure ulcer or other wounds. Good practice requires documentation of when the physician was notified, the response to the notification, any orders given, and the plan for examination and follow-up. Physicians must also meet the standard of care (what a reasonable and prudent provider would do in the same or similar situation) when managing a patient's wound. In facilities with an active wound care team/department, the routine management of the lesion is often handed to these specialists by the primary physician. The physician typically signs verbal orders that are written by these specialists. This allows interdisciplinary care and provides maximum potential to heal or mitigate the wound. This does not, however, relieve the physician of the responsibility to monitor the condition of the wound.

Physicians should arrange to examine the wound on a routine basis, have a good understanding of the rationale behind the wound care orders being signed, and be involved in consulting other specialties as needed to maximize healing. Physicians should take the lead in notifying the patient's family about the development of any lesion, just as they would any other negative event that occurs under their care. Pressure ulcers are symptoms of underlying medical, physical, and psychosocial problems. They are therefore a multidisciplinary issue involving nursing, nutrition, social work, physical therapy—and medicine—among many other specialties.

Notifying the Patient and Family

Prompt and thorough notification of the patient and family of any new wound or other adverse event is critical to ensure full understanding and participation in care. In some states, such as New Jersey, this is required by law.[12] Full disclosure of all facts related to the development of the wound should be provided: When was it discovered? How was it discovered? What interventions were being taken to prevent the wound? What interventions are being taken now? Give plenty of time for the information to be absorbed, and allow for questions. Many in the lay community believe that pressure ulcers or "bedsores" are the result of negligence. An initial negative reaction to an adverse event may be expected, but prompt and full disclosure of the situation will go a long way toward minimizing lingering

doubts and suspicions about the adequacy of care in your facility. When discussing the situation with the patient and family, use explanations, not excuses. While the patient's health status may have played a significant role in the development of the adverse event (e.g., a pressure ulcer, dehisced surgical incision, or other chronic wound), it is probably best not to dwell on this topic initially as it may be interpreted by family members as "blaming the patient." Follow-up conversations and briefings with the patient and family may serve as a better time to discuss the realistic goals of healing, once they digest the initial information and the effects of the care plan are better known. More in-depth information about communicating with the family regarding pressure ulcers can be found elsewhere in the literature.[13]

The patient and family should also be educated that new wounds, especially pressure ulcers, are likely to look worse before they look better. A suspected DTI may look rather innocent to the family as a "simple deep bruise" (discoloration) with maybe a little torn skin. The suspected DTI may evolve through a course that can include tissue ischemia, tissue necrosis, necrotic tissue separation, and even ultimate cavitation or ulceration. Lay persons could easily and incorrectly construe such a change in the wound as substandard care. Preparing the family in advance and setting expectations will reduce the shock of seeing a wound go through this natural process. Any conversations with family members and their response to the information should be promptly documented.

Ongoing Wound Documentation

Wound documentation places a significant burden on the healthcare provider due to the intricate nature of wound assessments. One way to ease this burden is to use logical, well-structured wound documentation forms or computer templates. Check boxes or drop-down lists are recommended for efficient documentation and to limit erroneous entries. Wound assessment forms can be structured in many different ways and will almost always improve the accuracy and consistency of documentation. Such forms can be created easily with word processing or spreadsheet software. A glossary of terms should also be developed for the more obscure terminology used on the wound care form. Drop-down menus, forms, and check boxes, however, are no substitute for narrative nursing assessments when required, and space must be provided in the medical record for such notes as needed.

Frequency of assessment will depend on the wound type, its phase of healing, the resources available to the wound care specialist, and other

factors. CMS recommends a weekly thorough assessment with daily monitoring of the dressing and wound to assess for complications in long-term care patients.[6] Weekly assessments by a wound specialist allow subtle changes to be noticed that would ordinarily be missed with more frequent inspection. Daily monitoring can be noted in the narrative notes, on treatment sheets, or on a wound assessment form per facility policy, but preferably in just one location for ease of reference. Wound documentation should be consistent and concise. Frequent brief, but thorough, notes indicate consistent care.

Wound Photography. . . or Wound Imaging?

Wound photography has become more popular with the advent of inexpensive, quality digital cameras. Two national organizations have position statements about the use of wound photography in wound care.[14,15] What is the rationale behind wound photography? Is it for assessment and diagnosis or just an attempt to mitigate legal liability? Consider *wound imaging* as an assessment and diagnostic tool just like an x-ray or magnetic resonance imaging. If thought of in this manner, wound imaging might be obtained routinely and consistently (per your facility policy and procedure) as with any other assessment and diagnostic imaging. A series of wound images will allow for more efficient and informed interventions and may assist in a legal defense should one become necessary. This regular, methodical approach is in contrast to taking one or two photographs during an inpatient stay to "cover ourselves legally"; taking this approach often backfires. What would any individual, and especially a juror, react more positively to: A series of detailed photographs showing progress of the wound or one or two photographs taken at odd intervals throughout the patient's stay? One reveals consistency; the other does not.

Wound imaging supplements—but does not replace—the need for written documentation. Each image should have accompanying text discussing what is observed in the photograph. This is similar to obtaining a radiologist's report after medical imaging. Consent for noninvasive medical imaging is rarely required, and the same should also be true for wound imaging as long as the patient cannot be readily identified. Management should clear the consent issue with legal counsel and risk managers. (See Chapter 6, Wound Assessment.)

Collaboration, Coordination, and

Communication

Collaboration, coordination, and communication of all specialty services are essential in maximizing the potential for wound healing. Documentation of "the three Cs" may also demonstrate to the medicolegal expert—and ultimately to a jury—that coordinated, consistent, interdisciplinary care was provided. Most facilities with a wound care team have policies that specify consultation for certain types of lesions. Many wound, ostomy, and continence (WOC) nurses provide both consult services and hands-on care at their facility.[16] If the WOC nurse acts in a consultant role, he or she should examine how consults by other facility services are structured and document in a similar manner. Consultants not only provide recommendations or establish a care plan but also educate other providers on their specialty and the rationale for their recommendations.

Wound care services are also provided by physical therapists in many facilities. As with WOC nurses, consults ideally should be based on the format at their facility and include a care plan and follow-up. Adequate follow-up by either a WOC or staff nurse should be ensured prior to the patient being discharged from physical therapy services. In addition, consults to plastic surgery, vascular surgery, or other surgical specialties are also part of interdisciplinary care. Because there are wide variations in approaches to chronic wound care by these specialties, disagreements can arise. Therefore, consistent documentation of communication among the specialties will resolve any differences and is an indicator of quality, interdisciplinary care.

Policies and Procedures: Normative or Positive?

Policies and procedures (P&Ps) establish standards of care within the facility. In any legal proceeding, P&Ps will be scrutinized and compared with the care that is documented in the chart. P&Ps are typically divided into two types of philosophies: normative and positive. A normative P&P describes what care *can* realistically and consistently be provided. A positive P&P describes what care *should* ideally be provided. P&Ps with a positive, ideal focus can cause great trouble in legal proceedings because they set unrealistic and unattainable goals that often exceed a reasonable standard of care.[17]

When establishing P&Ps in your facility, avoid using absolute terms like

"will" and "must" and specifying exact time frames for routine nursing interventions unless absolutely necessary. For example, a P&P that states "All patients will have a pressure ulcer risk assessment every Tuesday and Friday" sets an unrealistic expectation. Missing 1 day or doing the assessment on a Saturday instead of Friday is a violation of your own standard of care. Rewording the P&P to read "All bedfast or chairfast patients should have a pressure ulcer risk assessment twice a week" gives nursing staff more leeway in their care and documentation. In a lawsuit, "violations" of P&Ps are not always a liability in the defense of such actions. Departures that are explained by and supported by science-based care—and that are fully and completely documented contemporaneously—can often be used to the advantage of the defense. Sometimes, P&Ps must spell out exactly when and where something will or must be done, but mostly they should focus on guiding and educating staff members rather than enforcing strict rules and timelines for care (Box 3-5).

Box 3-5 Preventative Legal Care: Eight Key Areas of Vulnerability for Institutions

1. **Words have meaning—Assessing the legal implications of healthcare facility "policies and procedures"**
   KEY CONCEPT: Healthcare facility policies and procedures are "guidelines," not rules or regulations—and should be created and treated as such. These guidelines should be carefully crafted and periodically reviewed with regard to their clinical currency as well as their legal and healthcare implications. Words such as "never," "must," "shall," and "immediately" should be rigorously avoided.
2. **Assessing compliance with prescribing rules**
   KEY CONCEPT: Healthcare organizations and clinicians should review standing orders to ensure that they are in compliance with prescribing regulations.
3. **Changing and practicing within scope of practice**
   KEY CONCEPT: Healthcare institutions should ensure that caregivers are practicing within their scope of practice with regard to pressure ulcer assessment and documentation.
4. **Managing expectations and communicating carefully**
   KEY CONCEPT: The people most likely to be asked

difficult questions (regarding why, how, and when pressure ulcers develop) by patients and their families are not always in the best position to provide an accurate big-picture response. Frontline staff should be trained in how to delegate questions professionally and with compassion.

5. **Clinical documentation**

a. Skin assessments

KEY CONCEPT: Skin assessments should be conducted regularly and in accordance with the guidelines of a particular institution. Note that the skin assessment is different from the risk assessment and both must be performed.

b. Risk assessments

KEY CONCEPT: Pressure ulcer risk assessment guidelines for an organization should be worded in ways that are compatible with federal terminology.

c. Pressure ulcer assessment

KEY CONCEPT: The importance of reasonably complete documentation cannot be overemphasized. Medical record documentation from any provider involved in the care and treatment of the patient may be used to support the determination of whether a condition was present on admission. A "provider" means a physician or any qualified healthcare practitioner who is legally accountable for establishing the patient's diagnosis.[18]

d. Charting

KEY CONCEPT: Good pressure ulcer documentation should include a wound description, measurement and wound care treatments, as well as documentation of pressure redistribution devices and techniques, including support surfaces and turning schedules.

e. Electronic health records (EHRs)

KEY CONCEPT: Electronic record systems may not accommodate the documentation needs of pressure ulcer patients.

f. Photography

KEY CONCEPT: Photography has advantages and drawbacks in terms of litigation; know the guidelines set forth by the organization.

g. Staging

KEY CONCEPT: Training in the use of NPUAP pressure ulcer staging is recommended for all healthcare professionals, including physicians. When in doubt about a pressure ulcer's stage, all clinicians are encouraged to "describe what they see." Careful attention should be given to the discharge ulcer assessment.

6. **Preventability—Avoidable, unavoidable, preventable, or never events?**

   KEY CONCEPT: Government regulations and governmental language can be used to help juries decide healthcare malpractice and wrongful death cases. Understand these documents and how reimbursement terminology maps onto clinical practice.

7. **Education—The need for learning never ends**

   KEY CONCEPT: Since clinician knowledge of pressure ulcers has been linked to pressure ulcer incidence, initial and ongoing education about best practices is essential. Patient education should do more than address the basics of skin care; it should help patients formulate realistic expectations about their treatment, risks, and recovery.

8. **Preventive clinical care**

   KEY CONCEPT: "Bundles" work and should be implemented when appropriate. While there may be insufficient data for evidence-based product and device selection in pressure ulcer care, evidence-guided selections can be made.

Pressure ulcer prevention and treatment[9] practices have undergone significant revisions and changes since the first AHCPR guidelines were released 20 years ago. In addition to these recommendations, guidelines from NPUAP-EPUAP (2009) and the Wound, Ostomy and Continence

Nurses Society (2010) incorporate the latest research and recommendations.[3,16] The CMS guidelines[6] in particular are thorough, complete, and easy to read by a wider range of healthcare providers. While developed for long-term care, they are easily adaptable to acute care and other settings and provide comprehensive education and guidance while avoiding "absolute" terminology. Incorporation of these guideline recommendations into your practice indicates that your facility is up-to-date on the latest changes that impact pressure ulcer care.

Discharge: To Home or Another Facility

The patient and caregiver should have adequate resources to manage the wound upon discharge to the home or another facility.[17] Documentation of this coordination should include teaching strategies and the patient/caregiver response to them, consults with social work or home care, and any equipment or supplies sent home with the patient. Ensure that the patient has adequate follow-up for medical and wound issues.

The discharge assessment should be a thorough and complete "snapshot" of the patient before leaving your facility. As with the admission assessment, you cannot overdocument in this area. Thoroughly assess and document any wounds and the condition of high-risk pressure ulcer areas. Document any communication with the receiving facility. Alert the receiving facility to any wounds and describe them in detail. The medical discharge summary typically does not go into significant detail about wound therapy, and thus, the onus for this communication is placed on nursing. List all previous and current wound therapies, which will avoid wasted time in trying therapies that have already yielded little or no results. List all previous and current preventive measures and equipment, such as support surfaces, seat cushions, and heel protectors.

Summary

The patient's chart is an important legal document because it provides a written record of the care provided. It also serves as a means of communication among healthcare professionals about the patient's responses to care. Complete and accurate documentation is not only essential for good patient care but also the basis for mounting a defense in the event of legal action. Make your entries in the patient's record legible, thorough, professional, and factual. Ensure that all information is correct and accurate and timely.

Consistent documentation is a reflection of the quality, interdisciplinary care provided to an individual. Chronic wounds are often symptoms of many underlying medical, physical, and psychosocial problems. Documentation of these multiple issues requires a well-structured documentation system. Ensure your documentation system allows healthcare providers to consistently and concisely communicate and access their findings. Policies and procedures should be updated as new research and practices appear. The documentation system should incorporate and reflect these new practices. Such interventions will maximize communication among the interdisciplinary team and help to improve patient outcomes.[17]

PATIENT SCENARIO

Clinical Data

A 66-year-old woman presented herself and was admitted to the emergency department with complaints of headache, nausea, and right upper extremity weakness for 24 hours. Physical examination showed the patient's blood pressure to be 140/86 mm Hg, heart rate 64 bpm, and temperature 100.2°F. Her right extremity grip strength was diminished. Blood test results revealed a decreased leukocyte count and mild hypokalemia.

The patient's prior medical history included a myocardial infarction, hypertension (treated with medication), type 2 diabetes mellitus, and a questionable history of transient ischemic attack.

Before the patient could be sent to radiology for routine x-rays, she suffered precipitous cardiovascular collapse requiring intubation, pressor support, and admission to the ICU. Borderline perfusion was achieved with large doses of pressors (dopamine, norepinephrine) and intravenous fluids. Urine output was diminished but acceptable.

The provisional diagnosis was idiopathic cardiovascular collapse/compromise; sepsis was ruled out.

Hospital Course

The patient remained in the ICU for 24 days. She suffered a cerebral infarct (requiring no surgical intervention) on day 3. Hemodynamic instability lasting 12 days required pressor support and intubation.

Total parenteral nutrition was begun on day 7.

On day 24, the patient was transferred to a step-down unit with a sacral pressure ulcer that measured 5.6 × 11.2 cm with an average depth of 0.5 cm and significant undermining. Necrotic tissue was evident at the deepest parts of the wound and on some edges.

Surgical, nutritional, and neurologic consults were provided on admission to the step-down unit. A stepwise debridement plan was put into place. The patient subsequently underwent three surgical procedures to close the wound.

The ICU nursing notes 72 hours after admission included the formation of a sacral blister; progress of the wound was documented subsequently. Treatment orders were for saline and other moist dressings. A pressure redistribution bed was not ordered or provided until day 16.

The physician notes clearly indicate that the patient was critically unstable through day 12, maintaining only borderline perfusion. Concern for potential digit loss secondary to tissue hypoxia/coagulopathy/hypotension was noted by the ICU physician and in notes by the vascular consultant until day 12.

Case Discussion

This case was reviewed independently by three law firms, all of whom turned the case down. Expert review uniformly cited the critical, unstable nature of the first 12 days of hospitalization and the critically compromised perfusion as the reason for the ulcer. The experts also opined that while the ulcer was clearly a bad result, it was not an unacceptable result of the risk–benefit analysis performed by the caregivers in light of the patient's critically compromised circulatory status. While some experts privately opined that in a perfect world, all sacral pressure ulcers would be preventable, in the real world, as in this case, they would not opine that the formation of this ulcer was the result of medical or other negligence.

From a practice point of view, if a patient's condition is such that the caregivers conclude that measures ordinarily sufficient to delay or prevent tissue breakdown cannot be utilized, clear documentation of the decision-making process and conclusions should be made. In this case, the documentation was inadequate in this regard.

Show What You Know

1. In a medical malpractice trial, what's the role of the jury and the judge?

A. Interpreter of the law; finder of fact
B. Finder of fact; finder of fact and interpreter of the law
C. Finder of fact; interpreter of the law
D. Both judge and jury find fact and interpret the law

2. At trial, how many of the four elements of a medical malpractice claim must a defendant convince a jury that the plaintiff has failed to prove in order to successfully defend against a claim of medical malpractice?

A. One
B. Two
C. Three
D. Four

3. The medical record is:

A. a communication tool.
B. destroyed after 1 year.
C. a tool to communicate opinions related to a patient's care.
D. an optional part of health care.

4. Which one of the following statements about standards of care, practice guidelines, and policies and procedures is <u>false</u>?

A. They should be reviewed at regular intervals but never amended.
B. They should be reviewed at regular intervals and amended to reflect new information and research.
C. They should be based on research and practice experience.
D. They should be patient outcome oriented and quantifiable.

5. Given the new definition and understanding of suspected deep tissue injury (DTI) from NPUAP, an area of purplish discoloration on an immobile patient's sacrum that was not documented on admission but appears 24 hours later would:

A. be classified as an acute medical wound.
B. be documented as a stage IV pressure ulcer.

C. have occurred prior to admission.
D. be documented as a stage III pressure ulcer.

6. Which of the following exemplifies the best way to document frequency of turning a patient in the medical record?

A. A 2-hour check box
B. Regular turning q 2 hours
C. q 4-hour turning
D. Individualized turning schedule based on patient assessment q 4-hour turning

7. Normative policies and procedures describe care that can be:

A. realistically and consistently provided.
B. used for normal staffing situations only.
C. ideally strived for.
D. exceeded to achieve magnet status.

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Skin: An Essential Organ

4

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Objectives

After completing this chapter, you'll be able to:

- discuss the functions and different layers of the skin
- identify the components of a skin assessment
- list skin changes associated with the aging process
- differentiate between skin assessment and wound assessment
- identify and classify common skin conditions
- explain moisture-associated skin damage (MASD)
- describe ISTAP classification system for skin tears
- indicate prevention and treatment care options for skin tears
- define the concepts of skin failure and skin changes at the end of life.

Skin Anatomy and Physiology

The skin is the largest external organ of the body. Human skin is composed of two distinct layers: the epidermis, the outermost layer; and the dermis, the innermost layer (Fig. 4-1). The dermal–epidermal junction, commonly referred to as the basement membrane zone (BMZ), separates the two layers. Under the dermis lies a layer of loose connective tissue, called subcutaneous tissue, or hypodermis (Table 4-1).

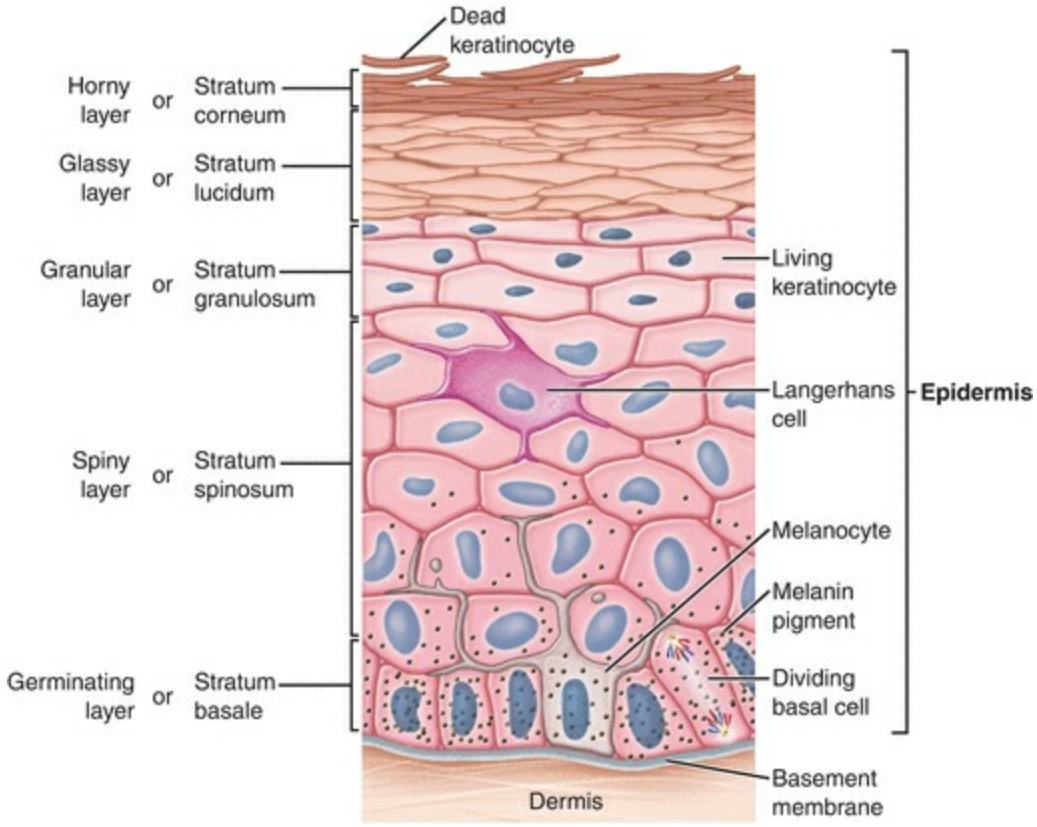


Figure 4-1. Layers of the skin. Two distinct layers of skin, the epidermis and dermis, lie above a layer of subcutaneous fatty tissue (also called the hypodermis). The dermal–epidermal junction (also called the basement membrane zone) lies between the dermis and epidermis.

Table 4-1 Skin Layer Functions

This table shows characteristics and general functions of each layer of the skin.

Skin Layer	Characteristics	General Function
Epidermis	• Outer layer of skin • Consists of five layers (or strata): corneum, lucidum, granulosum, spinosum, and basale (or germinativum) • Repairs and regenerates itself every 28 days	• Protective barrier (sun damage, transepidermal water loss [TEWL]) • Organization of cell content • Synthesis of vitamin D and cytokines • Division and mobilization of cells • Maintaining contact with dermis • Pigmentation (contains melanocytes) • Allergen recognition (contains Langerhans cells) • Differentiates into hair, nails, sweat glands, and sebaceous glands
Dermis	• Consists of two layers—papillary dermis and reticular dermis—composed of collagen, reticulum, and elastin fibers • Contains a network of nerve endings, blood vessels, lymphatics, capillaries, sweat and sebaceous glands, and hair follicles	• Supports structure • Mechanical strength • Supplies nutrition • Resists shearing forces • Inflammatory response
Subcutaneous tissue (hypodermis)	• Composed of adipose and connective tissue • Contains major blood vessels, nerves, and lymphatic vessels	• Attaches to underlying structure • Thermal insulation • Storage of calories (energy) • Controls body shape • Mechanical "shock absorber"

The epidermis is a thin, avascular layer that regenerates itself every 4 to 6 weeks. It's divided into four layers or strata (presented in order from the outermost layer inward).

- *Stratum corneum (horny layer)*—consists of dead keratinocyte cells; flakes and sheds; is easily removed during bathing activities and more efficiently by scrubbing the surface of the skin.
- *Stratum granulosum (granular layer)*—also contains Langerhans cells in addition to keratinocytes.[1]
- *Stratum spinosum (spiny layer)*—contains keratinocytes and Langerhans cells.
- *Stratum basale (germinating layer)*—single layer of epidermal cells (keratinocytes); contains melanocytes; can regenerate.

A fifth layer, the *stratum lucidum (glassy layer)*, lies between the stratum corneum and the stratum granulosum. This packed translucent line of cells is found only on the palms and soles and not seen in thin skin.

The epidermis is composed of keratinocyte cells. Basal keratinocytes (stratum basale) have the capacity to divide, giving rise to suprabasal layers of epidermis. Once basal keratinocytes leave the stratum basale, they start the process of differentiation, during which they die. This process involves making insoluble proteins and their crosslink which, along with lipids and

membrane components, form the insoluble, horny stratum corneum layer.[2–5,5] In order to maintain the barrier, keratinocytes have the capacity to completely regenerate the epidermis. If damaged (such as wounded, burned, exposed to ultraviolet [UV] light or chemicals), keratinocytes, in order to repair the damage, change their biology. Instead of differentiating, they become "activated" and start to divide rapidly and, in the case of a wound, they migrate over the gap to repair the damage.[3] They also signal to other neighboring cell types, such as fibroblasts, Langerhans cells, and melanocytes, that the skin barrier is compromised and that they're needed to help repair the damage. Once the damage is repaired, the keratinocytes cease their activation and resume their normal differentiation process.[6]

The BMZ divides the epidermis from the dermis. It contains fibronectin (an adhesive glycoprotein), type IV collagen (a non–fiber-forming collagen), heparin sulfate proteoglycan, and glycosaminoglycan.[7] The BMZ has an irregular surface—called rete ridges or pegs—projecting downward from the epidermis that interlocks with the upward projections of the dermis. The two interlocking sides resemble the two sides of a waffle iron coming together. This structure anchors the epidermis to the dermis, preventing it from sliding back and forth. As skin ages, the basement membrane flattens, and the area of contact between the epidermis and dermis is decreased by 50%, thus increasing the risk of skin injury by traumatic, accidental separation of the epidermis from the dermis (Fig. 4-2).

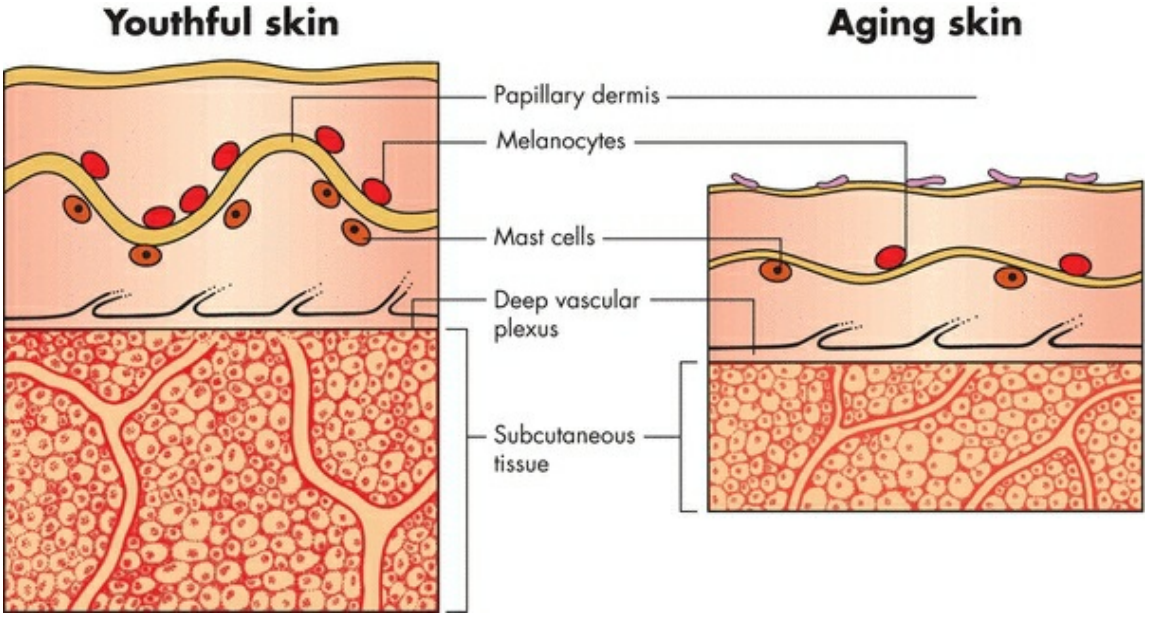


Figure 4-2. Effects of aging on the basement membrane zone (BMZ). The illustrations below show the effects of aging on the BMZ. Specifically, the

basement membrane flattens, reducing the area of contact between the epidermis and the dermis by 50%.

The dermis is an essential part of the skin and is commonly referred to as the "true skin."[8] As the second layer, it's the thickest layer and is composed of many cells. The major proteins found in this layer are collagen and elastin, which are synthesized and secreted by fibroblasts; collagen forms up to 30% of the volume or 70% of the dry weight of the dermis.[7–9] The dermis is a matrix that serves to support the epidermis. It's divided into two areas, the papillary dermis and the reticular dermis.[7]

- The *papillary dermis* is composed of collagen and reticular fibers. Its distinct, unique pattern allows fingerprint identification for each individual. It contains capillaries for skin nourishment and pain touch receptors (pacinian corpuscles and Meissner's corpuscles).
- The *reticular dermis* is composed of collagen bundles that anchor the skin to the subcutaneous tissue. Sweat glands, hair follicles, nerves, and blood vessels can be found in this layer.

The main function of the dermis is to provide tensile strength, support, moisture retention, and blood and oxygen to the skin.[8] It protects the underlying muscles, bones, and organs. The dermis also contains the sebaceous glands that secrete sebum, a substance rich in oil that lubricates the skin. Furthermore, it also contains hair follicles that are the source of multipotent stem cells, which have the capacity to restore the epidermis.[6]

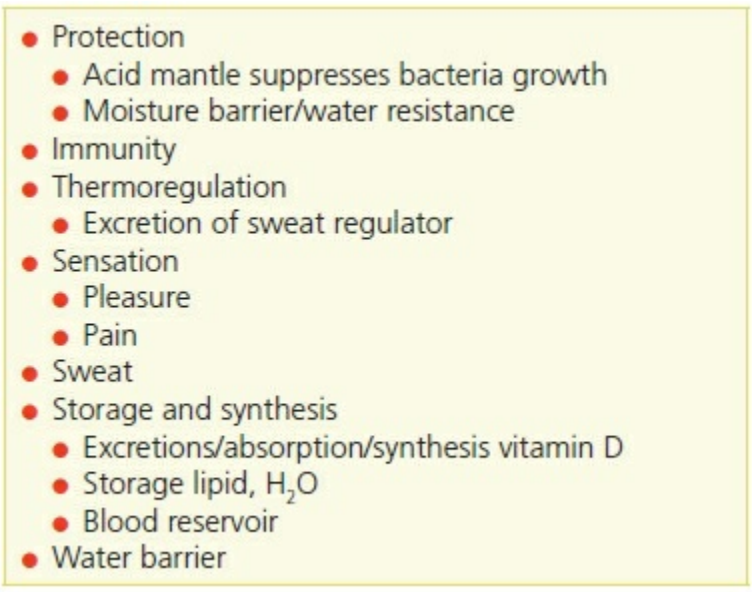
The subcutaneous tissue, or hypodermis, attaches the dermis to underlying structures. Its function is to promote an ongoing blood supply (blood reservoir) to the dermis for regeneration. It's primarily composed of adipose tissue, which provides a cushion between skin layers, muscles, and bones. It promotes skin mobility, molds body contours, and insulates the body.

Skin Roles and Functions

Skin is an important organ whose diverse functions are not always appreciated (Table 4-2). In adults, the skin weighs between 6 and 8 pounds (2.7 to 3.6 kg) and covers over 20 ft^2 (1.9 m^2). Skin thickness varies from 0.5 to 6 mm according to its location on the body; for example, skin can be as thin as $\frac{1}{50}$" on the eyelids and as thick as $\frac{1}{50}$" on the palms and soles, where

greater protection is needed. The skin receives one-third of the body's circulating blood volume—an oversupply of blood compared to its metabolic needs.

Table 4-2 Skin Roles and Functions



Ayello, E.A., Sibbald, R.G., Quiambao, P.C.H.., et al. "Introducing a Moisture-Associated Skin Assessment Photo Guide for Brown Pigmented Skin," *World Council of Enterostomal Therapists Journal* 34(2):18-25, 2014.

The normal range of skin pH is 4 to 6.5 in healthy people.[10,11] This "acid mantle" helps to maintain a normal skin flora by serving as a protective barrier against bacterial and fungal infections. It also supports the formation and maturation of epidermal lipids and assists in maintaining their protective barrier function. The acid mantle also provides indirect protection against invasion by microorganisms and protection against alkaline substances.[10] If the acid mantle loses its acidity, the skin becomes more prone to damage and infection. Frequent use of soap products and overwashing can alter the stratum corneum and its ability to serve as a protective barrier. Alternatively, several skin conditions can *increase* the skin's surface pH, including eczema, contact dermatitis, atopic dermatitis, and dry skin.[12,13] Systemic diseases may also increase the skin's surface pH, such as in diabetes, chronic renal failure, and cerebrovascular disease.[14]

The skin's major functions are protection, immunity thermoregulation, sensation, sweat, storage and synthesis, and water barrier.[15,16] Skin

protects the body by serving as a barrier from invasion by organisms such as bacteria. Epidermis synthesizes natural antimicrobials called defensins.[17] Because staphylococcal species (such as *Staphylococcus aureus* or *Staphylococcus epidermis*) tolerate salt, they are present in large numbers as resident bacteria on the skin.[15,17] Another organism found on the skin is yeast, which is commonly found on the trunk and ears and as fungus between the toes.[15,18] In addition, skin makes its own antimicrobial peptides called defensins.[19,20]

Sensation is a key function of the skin. Areas that are most sensitive to touch have a greater number of nerve endings.[18] These include the lips, nipples, and fingertips. In humans, the fingertips are the most sensitive touch organ and enable us to correctly identify objects by touch (stereognosis) rather than by sight. Many tactile corpuscles lie at the base of hair follicles, and shaving reduces the tactile sensibility of that skin area. In hairless body regions, the tactile corpuscles are called Meissner's corpuscles.[18] Pleasurable, firm touching sensations, such as from a massage or hugs of affection, are transmitted via the skin as they generate nerve transmissions through these tactile corpuscles.

Itch is one of the alarm sensations of the skin and serves as a defense mechanism. Chemicals that are released after skin injury may promote the inflammatory process and can also induce itch or pain. Itch and pain are in close regulatory relationship—for example, central pain inhibition may enhance the act of itching, while also inhibiting the act of itching.[21]

Somatic pain (from the outer body surfaces and framework) is also communicated through the skin. Superficial (acute) pain to a local area is usually transmitted by very rapid nerve impulses by A-delta fibers[18] and tends to be sharp but ceases when the pain stimulus stops. Deep (chronic) pain impulses are transmitted slowly over the smaller, thinly myelinated C fibers. In contrast, this type of pain tends to spread over a more diffuse area, lasts for longer periods of time, and remains even after the pain stimulus is gone.[18] As a sign of possible skin injury, pressure also serves as a protective warning sensation.

Temperature regulation and fluid and electrolyte balance are achieved in part by the skin. Thermoregulation is controlled by the hypothalamus in response to internal core body temperature. Peripheral temperature receptors in the skin assist in this process called *temperature homeostasis*.[22] By losing a copious amount of water—for example, sweating—through the skin, lungs, and buccal mucosa, homeostasis of body

temperature is maintained. Skin temperature is controlled by the dilatation or constriction of skin blood vessels. When body core temperature rises, the body will attempt to reduce its temperature by releasing heat from the skin. This is accomplished by sending a chemical signal to increase blood flow in the skin from vasodilation, thus increasing skin temperature.

Practice Point

Increased temperature → Skin blood vessel vasodilation → Heat loss from epidermis → Body maintains temperature homeostasis.

In contrast, the opposite occurs when the body's core temperature is reduced; the chemical signal causes decreased blood flow from vasoconstriction, thus lowering the skin's temperature.[22,23]

Practice Point

Decreased temperature → Skin blood vessel vasoconstriction → Heat conservation → Body maintains temperature homeostasis.

The skin also aids in the excretion of end products of cell metabolism and prevents excessive loss of fluid. Other important functions of the skin include its manufacturing ability and immune functions.[24] For example, when exposed to UV light, the skin can synthesize vitamin D.[25] Although the skin's hypersensitivity responses in allergic reactions are commonly seen, the skin's role in immune function isn't always fully appreciated. Indeed, Langerhans cells and tissue macrophages, which play an important role in digesting bacteria, as well as mast cells, which are needed to provide proper immune system functioning, are all present in the skin.[1,7,15] In addition, keratinocytes are very powerful in generating rapid inflammatory response because they contain prestored proinflammatory signals, such as interleukin-1, which gets released the moment the barrier is broken and is considered the first signal of wounding.[26]

Aging and the Skin

Intrinsic and Extrinsic Causes of Aging Skin

Changes that occur to human skin as it ages are classified as intrinsic and extrinsic. Intrinsic refers to physiologic changes taking place in the aging process and are most evident in sun-protected areas, while extrinsic refers to environmental influences. It is sometimes difficult to separate intrinsic from extrinsic factors due to the pervasive impact of diet and lifestyle factors, but there are profound genetic and ethnic differences in the body's response to both.

One major factor impacting both intrinsic and extrinsic aging is oxidative stress leading to macromolecular damage and cell senescence. Changes of aging depend largely upon homeostasis between free radical production and the proper working of repair systems. The term reactive oxygen species (ROS) refers to free radical and nonradicals that contain an oxygen atom. ROS are by-products of cell respiration that takes place in mitochondria during oxidative phosphorylation but are generated by other cellular structures such as peroxisomes and endoplasmic reticulum.[27] Not only does ROS increase with age but aging is accompanied by reduced antioxidant activity and decreased DNA repair capability.

Extrinsic causes of aging skin are also mediated by oxidative stress and ROS. The most important cause of aging skin is UV light from the sun, which is divided into UVA and UVB. UVA is considered more damaging because of its deeper penetration into the dermis. Other environmental factors recognized as extrinsic causes of aging include cigarette smoke, ozone, and airborne particulate matter with adsorbed polycyclic aromatic hydrocarbons.[28] These extrinsic factors generate free radicals and ROS that overwhelm the body's natural antioxidant defenses and stimulate lipid peroxidation reaction cascade which in turn releases proinflammatory mediators that include matrix metalloproteases (MMPs). Mitochondrial DNA mutations resulting from ROS leads to defective electron transfer activity and oxidative phosphorylation.

Telomere shortening is a known result of oxidative insults resulting from environmental factors but is also associated with psychological stress.[29] Telomeres are repetitive DNA sequences at the end of linear DNA that shorten each time a cell divides and ultimately leads to cessation of cell division and apoptosis, or programmed cell death.

Polycyclic aromatic hydrocarbons adsorbed to airborne particulate

matter induce xenobiotic metabolism that releases ROS and MMPs that accelerate aging. Xenobiotic metabolism refers to the metabolic pathways the body uses to eliminate environmental toxins such as polycyclic aromatic hydrocarbons that are adsorbed to airborne particulate matter.

Changes in Aging Skin

Age-related changes in the dermis are numerous, and their cumulative effect increases susceptibility to environment and internal stresses that lead to increased fragility and impaired wound healing. The most striking is the approximately 20% loss in dermal thickness that accounts for the paper-thin appearance of aging skin.[30,31] This decrease in dermal cells and proportional reduction in collagen fibers, blood vessels, nerve endings, and collagen lead to altered or reduced sensation, thermoregulation, rigidity, moisture retention, and sagging skin.[30,32]

A decrease in differentiation and formation of the stratum corneum is also detected in aging skin. In the epidermis, there is reduced keratinocyte proliferation and turnover time, and surface pH is less acidic. Desquamation is less effective, and lipid biosynthesis in the stratum corneum is impaired. There are decreased melanocytes that protect from UV radiation and decreased Langerhans cells that process microbial antigens and present them to other immune system cells. This is accompanied by altered T- and B-cell function, and a general proinflammatory environment that is now an accepted component of the aging process.

The dermal–epidermal junction is flattened with smoothing of the rete ridges and decreased adhesion of this critical mechanical defense barrier. The dermis becomes atrophic with reduced numbers of fibroblasts and mast cells, and collagen becomes disorganized with change in synthesis from type I to type III. There is decreased synthesis of elastin, and elastic tissue is degraded with overall loss of elasticity.

Reduced collagen deposition in elderly skin could explain the development of dermal atrophy and might relate to poor wound healing.[33] The subcutaneous fat below the dermis consists primarily of adipose tissue and provides mechanical protection and insulation. Its loss during aging results in parallel reductions in these protective functions. Subcutaneous tissue undergoes site-specific atrophy in such areas as the face, dorsal aspect of the hands, shins, and plantar aspects of the foot, increasing the energy absorbed by the skin when trauma occurs to these areas.[33]

Many of the changes in aged skin are linked to the hormones estrogen

and androgen. Decreased estrogen in menopausal women, similar to ovariectomized mice, leads to a decrease in collagen deposition, slower epithelialization, and delayed wound healing. These effects may be reversible by hormone replacement therapy (HRT).[34] In addition, a genetic polymorphism in estrogen receptor-beta has been linked to a predisposition to venous ulcerations in both male and female patients.[34] In contrast to estrogen, which is beneficial for wound healing, androgens are implicated in the etiology of venous ulcerations.[35] In addition, use of inhibitor (antagonist of androgen) or castration in mice leads to increased collagen deposition and acceleration of wound healing.[36]

Decreased mechanoreceptors including Meissner's and Pacini's corpuscles result in diminished sensation to light touch, pressure, and vibration. A decrease in pain perception may make elderly people more vulnerable to traumatic environmental insults such as wearing tight shoes, stepping on an object, or hitting legs on the side of a chair. Aging skin is also less able to manufacture vitamin D when exposed to sunlight.[30,31] The number of Langerhans cells and mast cells diminishes in aging skin, translating into decreased immune function.[30–37] Medications also have adverse effects on the skin's immune function. For example, steroids cause thinning of the epidermis.[31,38]

There are decreased pilosebaceous units that are composed of hairs, sebaceous glands, and arrector pili muscles, which contribute to decreased sebum production. Impaired thermoregulation results from loss of subcutaneous fat results in decreased energy stores and along with decreased autonomic nerves from the sympathetic nervous system and decreased dermal vascularity. Loss of sweat glands also contributes to impaired thermoregulation as well as decreased ability to manage water balance in response to antidiuretic hormone.

Skin changes seen with aging are accelerated by sun exposure, specifically due to UV radiation.[30,39] UV irradiation causes local inflammation and local immunosuppression with DNA damage. Changes induced by photoaging are superficially similar but differ slightly under the microscope. Photo-damaged skin looks coarse, rough, and wrinkled and is prone to developing malignancy.

Aging skin is less able to retain moisture due to a decrease in dermal proteins, which leads to oncotic pressure shifts and diminished fluid homeostasis, thereby putting elderly people at risk for dehydration. Normal water content of the skin is 10% to 15%. Below 10%, the skin becomes dry

and is more vulnerable to damage.[31] Because soap increases the skin's pH to an alkaline level, using emollient soap and bathing every other day, instead of every day, can decrease the incidence of skin injury, such as skin tears, in elderly patients.[10]

An elderly person's skin is less stretchable due to a decrease in elastin fibers.[30,33,37] Because of the thinning of the epidermal layer, the skin becomes a less-effective barrier against water loss, bruising, and infection, accompanied by impaired thermal regulation, decreased tactile sensitivity, and decreased pain perception.[30,31,40,41] Due to a decreased amount of dermal proteins, the blood vessels become thinner and more fragile, thereby leading to a type of hemorrhaging known as senile purpura. Hematomas can dissect into surrounding skin, usually over the extensor surfaces of the hands and forearms, resolving to leave brownish discoloration caused by hemosiderin deposits. The appearance of dissecting hematomas can mimic dermal changes associated with bleeding diathesis or impaired clotting system.

Skin Assessment

A skin assessment begins with a head to toe inspection and palpation of the skin.[42,43] A minimal basic skin assessment should include the following five parameters: temperature, color, moisture, turgor, and intact skin or presence of open areas. Looking and touching the skin is part of a patient assessment. Lacking consensus in the literature as to what constitutes a minimal skin assessment, the U.S. Centers for Medicare and Medicaid Services (CMS) recommends to long-term care (LTC) facilities these five parameters for skin assessment (Table 4-3).

Table 4-3 Elements of a Basic Skin Assessment

At a minimum a basic skin assessment should include the following:

Temperature

- Normally warm to the touch
- Warmer than normal could signal inflammation
- Cooler than normal could signal poor vascularization
  (Compare bilateral extremities for temperature changes)

Color

- Culturally component skin assessment: awareness of differences across skin pigmentation
- Normal color tones: light ivory to medium, to deep brown, black, yellow to olive, or light pink to dark, ruddy pink
- Intensity: paleness may be an indicator of poor circulation or anemia
- Erythema, varies with natural skin color (Sommers)
- Hyperpigmentation or hypopigmentation reflect variations in melanin deposits or blood flow

Moisture

- Dry (xerosis)
- Moisture-associated skin damage (incontinence, perspiration periwound maceration, peristomal)
- Edema, mild, moderate, severe

Turgor

- Tented skin normally returns to its original state quickly
- Slow return to its original shape (dehydration or effect of aging)

Integrity

- No open areas
- Type of skin injury (use the appropriate classification system to identify and record injury type)

A review of the patients' history should capture the usual skin care practices including bathing routine (shower, or bath) and type of skin/soap products used or preferred. A family history of any skin conditions should be elicited, as well as any known skin allergies.

Skin Temperature

Skin temperature should be assessed by comparing bilateral extremities for coolness or warmth. Warmth can be an indication of inflammation, or

chronic venous insufficiency while coolness could signal diminished vascular supply.

Skin Color

Skin color can reflect various skin conditions. Assessing for changes in a person's normal skin tones is extremely challenging. In order to evaluate skin color, one needs adequate light (natural light or halogen is preferred) to see if there are any changes in the skin tone. Healthcare professionals are caring for many diverse populations with varying skin colors/tones.[16] Clinicians need to keep this in mind, when assessing the nuances of changes in patients' skin.[16,43,44] Differences in skin color pigmentation can be described using Fitzpatrick's skin categories.[45] The four categories were expanded to include East Asian and Black skin types. Each of the six categories include the skin's response to UV radiation[16] (Table 4-4).

Table 4-4 Variation of Skin Colors

Skin Color	Description	Burns	Tans
1. Pale white	Red/blond hair Blue eyes, freckles	Always	Never
2. Fair white	Red/blond hair Blue, green, hazel eyes	Usually	Minimally
3. Cream white	Any hair color Any eye color	Sometimes (mild)	Uniform
4. Moderate brown	Mediterranean skin	Rarely	Always
5. Dark brown/yellow	Mid-East Asian	Very rarely	Easily
6. Deeply pigmented	Dark brown/black skin	Almost never	Very easily

"The first three skin types are most likely to have chronic sun damage. Lighter skin with the excessive sun-related changes increases the likelihood of an individual developing skin cancer.

Any trauma or injury to the skin can produce postinflammatory hypo- or hyperpigmentation when injury disrupts the dermal–epidermal junction where most of the melanocytes reside. This can also occur with specific skin disease including the end stage of eczema or dermatitis, lichen planus, or collagen vascular diseases such as lupus erythematosus." From Ayello, E.A., Sibbald, R.G., Quiambao, P.C.H., Razor, B. "Introducing a Moisture-Associated Skin Assessment Photo Guide for Brown Pigmented Skin," *World Council of Enterostomal Therapists Journal* 34(2):18-25, 2014.

(Modified from Skin Inc. The Fitzpatrick Skin Type Classification Scale. November 2007, Available at http://www.skininc.com/skinscience/physiology/10764816.html.)

Moisture Balance

Assess skin for the level of moisture seen externally, or felt on the surface. Too little moisture results in dry, scaly, flaky skin commonly referred to as xerosis (Fig. 4-3). Too much moisture on the skin surface can result in moisture-associated skin damage (MASD). Moisture within the skin tissue (beneath the surface) can result in edema. In the following sections, we will go into more detail on these topics.

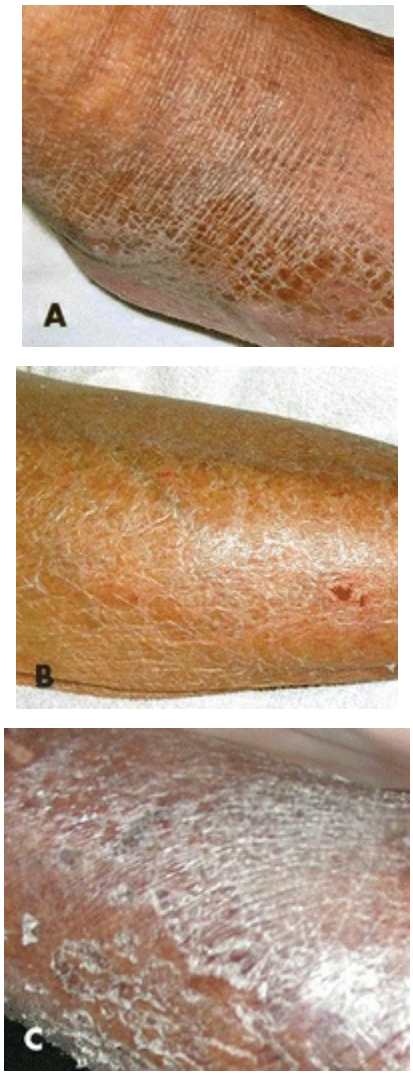


Figure 4-3. Xerosis terminology. **(A) Mild.** Dry skin with minimal flaking. Treatment: Hydrate the skin using a moisturizing agent frequently. **(B) Moderate**. Dry skin with a scaly, fish-like appearance that's easily rubbed off the skin surface. Treatment: Use an exfoliating emollient moisturizing agent. **(C) Severe**. Cracking, parched appearance of skin that resembles dry earth. Treatment: Use moisturizer with urea, AHA, or lactic acid to exfoliate calloused dry skin.

Xerosis

Xerosis is the medical term for dry skin.[46] In xerosis, the skin appears dry, scaly, and flaky. Although there is a xerosis scale, it's not widely used in clinical practice. Clinicians generally classify xerosis as mild, moderate, and severe.

The term xerosis has no particular diagnostic implication. Xerosis can be caused by environmental factors or a symptom of an underlying disease. For this reason, a patient's complaint of "dry" skin needs to be explored further. Skin exposure to a dry environment, such as central heating, wind, temperature extremes, or air conditioning, can all lead to xerosis.

Management

The goal in treating xerosis is to protect the skin from excessive transepidermal water loss (TEWL) and return the natural moisturizing factors to the stratum corneum. This is best accomplished by using moisturizing agents that contain lipids—an essential component in forming an impervious barrier, or seal, on the stratum corneum, thus preventing further water loss (Table 4-5). As water is retained, the skin surface is flattened, and scaling is reduced.[46] Patient, family, caregiver instruction on cleansing, environmental factors, and hydration is also important (see Patient Teaching tips)

Table 4-5 Moisturizer Functions and Ingredients

Moisturizer Functions

- *Humectants* promote water retention within the stratum corneum.
- *Occlusives* minimize water loss to the external environment.
- *Emollients* contribute to stratum corneum hydration.

Moisturizer Ingredients (Main Types)

- Humectants
  - Glycerin
  - Urea
  - Hydroxy acids (lactic acid)
  - Propylene glycol
  - Emollients
  - Protein rejuvenators
- Occlusives and emollients
  - Petrolatum
  - Mineral oil
  - Lanolin

Practice Point

Skin can become dry when TEWL drops below 10%

Patient Teaching

To avoid xerosis, patients should be given clear instructions regarding cleansing, their environment, and remaining hydrated.

Cleansing

Instruct the patient to:

- avoid long baths (<15 minutes) or consider showering instead
- bathe every other day rather than daily
- use tepid water rather than hot water
- use pH-balanced soaps (4.0 to 6.5), avoid excessive use of deodorant soaps, and rinse well
- avoid vigorously using a washcloth to clean the skin
- pat or blot the skin, rather than rubbing with a towel, so some

water is left on the skin
- apply moisturizers *immediately* after bathing or showering.

Environmental

Instruct the patient to:

- use a humidifier during the winter months when central heating is being used
- drink plenty of water
- wear a sunscreen with a sun protection factor (SPF) of 15 or higher that contains a moisturizer
- use nonfragrant laundry detergents, fabric softeners, and products.

Hydration

Instruct the patient to:

- use moisturizers, applying frequently, using correct gentle application technique for specific product (check product directions on how to use), and implementing fall safety precautions because bathing surfaces may be slippery if using bath oils.

Practice Point

Stop the Xerosis Cycle!

- Dry skin (xerosis)
- Pruritus
- Scratching

Because many moisturizers dissipate after 3 to 4 hours, these authors recommend using long-lasting moisturizers to cool, soothe, and restore barrier function. The goal is to break the itch–scratch–itch cycle, which happens because dry skin is often itchy, causing patients to scratch their

skin. In turn, excessive scratching can ultimately lead to a break in the skin. Once the skin barrier is broken, it becomes a portal of entry for bacteria, which can lead to infection. This repetitive scratching causes chronic thickening of the dermis known as lichenification. Therefore, prompt identification of the itch–scratch–itch cycle as well as teaching the patient about skin damage (from scratching) is extremely important in helping the skin to heal and reducing the occurrence of lichenification (Fig. 4-4).

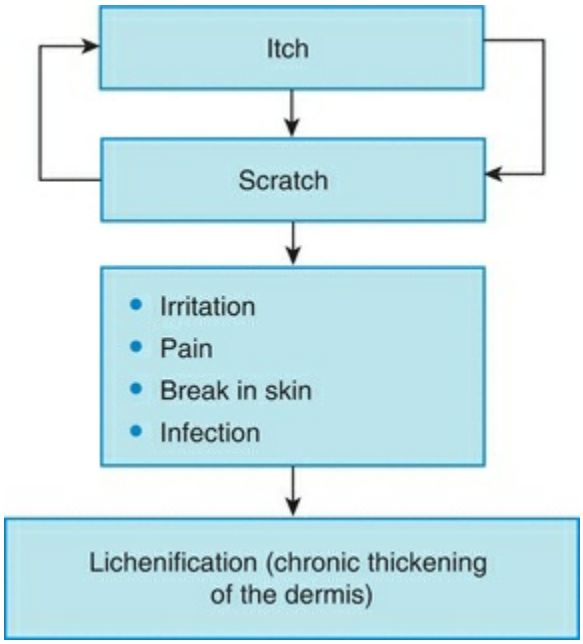


Figure 4-4. Itch–scratch–itch cycle.

Patient Teaching

Teach the patient about the itch–scratch–itch cycle.

Pruritus

Pruritus is the medical term for itchy skin and is a common symptom for several diseases.[5] Therefore, taking a detailed history aids in determining if the cause is from such an underlying disease or if it's just untreated xerosis. For example, pruritus may be a symptom of renal or liver disease, scabies, or dry skin from aging (Table 4-6). Helping patients understand the itch–scratch–itch cycle and their own behavioral pattern is important to successful management. Treating the person with pruritus incorporates many

of the elements in Table 4-7.[31]

Table 4-6 Pruritus

Pruritus is frequently seen in the following conditions and diseases:

- Brain tumor
- Biliary cirrhosis
- Diabetes mellitus
- Drugs
- Idiopathic (has no diagnostic cause)
- Liver disease
- Malignancies (especially in prevalent Hodgkin's where one-third of patients may have itching lasting up to 1 y)
- Multiple sclerosis
- Polycythemia (itch occurs after a hot bath)
- Psychological (anxiety disorders)
- Renal failure
- Senile pruritus (idiopathic pruritus in the elderly)
- Thyroid disorders (improves with treatment)
- Topical infections

Adapted with permission from Tomic-Canic, M. "Keratinocyte Cross-Talks in Wounds," *Wounds* 17:S3-6, 2005.

Table 4-7 Treatment Plan for Pruritus

- Manage the underlying disease that causes the pruritus.
- Use topical emollients and bathing strategies as outlined in the xerosis care plan.
- Implement behavior modification (stopping scratching and break the itch–scratch–itch cycle).
- Keep nails short.
- Wear gloves at night to decrease skin damage.
- Use cotton sheets, which may be more soothing to itchy skin.
- Help the patient avoid wearing clothing that can irritate the skin, such as wool or other "scratchy" fabrics.
- Limit the indiscriminate use of topical steroids and antihistamines as their effectiveness needs further investigation.

Venna, S.S., Gilchrest, B.A. "Skin Aging and Photoaging," *Skin and Aging* 12:56-69, 2004.

Gilhar, A., et al. "Ageing of Human Epidermis: The Role of Apoptosis, Fas and

Telomerase," *The British Journal of Dermatology* 15(1), 2004.

Moisture-Associated Skin Damage

Just as dry skin can be a problem, exposure to excessive moisture can also cause skin damage. Four types of MASD have been defined in the literature: incontinence-associated dermatitis (IAD), intertriginous dermatitis (ITD), periwound moisture–associated dermatitis, and peristomal moisture–associated dermatitis.[47–55] Common causes of MASD include incontinence of stool and/or urine (IAD), wound exudates (see Chapter 6), fistula or stoma effluent (see Chapter 19), and perspiration, mucus, or saliva.[47–50] Skin damage from moisture is distinct from pressure and differentiating the correct etiology between these two types of skin injuries is important for appropriate treatment and prevention[50–53] (Table 4-8). It is not clear whether moisture alone or a combination of wetness coupled with irritants within the moisture source causes damage to the skin.[50] Classification of gluteal and buttock wounds remains a challenge.[54]

Table 4-8 Differential Diagnosis of MASD and Pressure Ulcers in the Perineal and Genital Area

Characteristic	Incontinence-Associated Dermatitis (IAD)	Pressure Ulcer (PrU)
Location	Often in skin folds Diffuse	Usually over bony prominence Well circumscribed
Color	Red or bright red	Red to bluish/purple
Depth	Intact skin to partial-thickness wound	Intact skin to partial- or full-thickness wound
Necrosis	None	May be present
Pain and itching	May be present	May be present

Adapted from Gray, M., Bohacek, L., Weir, D., et al. "Moisture vs Pressure. Making Sense out of Perineal Wounds," *Journal of Wound, Ostomy, and Continence Nursing* 34(2):134-42, 2007.

Incontinence-Associated Dermatitis

The remainder of this section will focus on MASD caused from incontinence of urine, stool, or both. IAD is sometimes referred to as

perineal dermatitis or even in infants as "diaper dermatitis" (Fig. 4-5). Literature[50–52] has advocated for the use of the term MASD to identify these skin lesions. IAD is believed to be reversible, begins as persistent redness, and progresses to partial-thickness skin injury but not full-thickness wounds.[50] Although there are three IAD instruments discussed in the literature, they are not yet widely used in practice.[51]

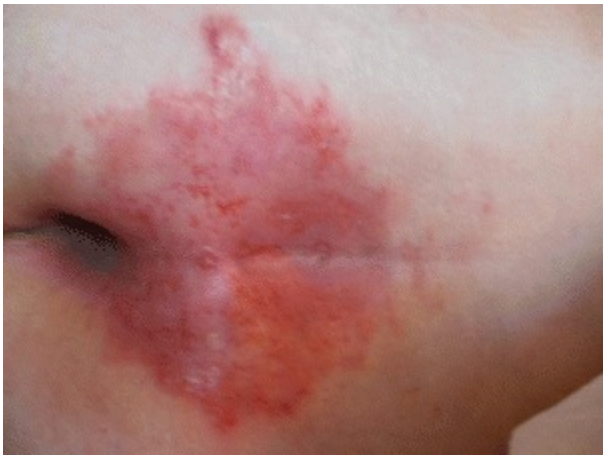


Figure 4-5. Incontinence-associated dermatitis (IAD). (Photo courtesy of KDS Consulting.)

Gray et al.[51] summarized several research studies about prevention of IAD and concluded that a routine perineal skin protocols that avoided soap and used cleansing products with a pH range of normal skin were effective along with reducing skin scrubbing and friction. Use of products that moisturize and protect the skin were recommended.[51] Holistic care also requires interventions to address and minimize the episodes of incontinence such as a scheduled toileting program.[51] Other strategies to prevent skin exposure to urine or stool include use of containment devices such as condom catheter, anal pouch, or bowel management systems. If absorptive products are used, make sure that they wick urine or stool away from the skin. Recommendations for a structured skin care regimen include cleaning the skin daily and after each incontinence episode using a no-rinse cleanser; not scrubbing the skin; applying humectants or emollient moisturizer; barrier creams; ointments with petroleum, zinc oxide, or dimethicone; or skin sealant products.[51–53] If a fungal infection is also present, this will require additional antifungal products.[51,53]

Intertriginous Dermatitis

Another type of MASD is ITD. It is "the term for skin damage caused by trapped perspiration and frictional forces between opposing skin surface."[54] Clinicians should be looking in skin folds including the inframammary, axillary, and inguinal skin folds as well as the abdominal or pubic panniculi[49] (Fig. 4-6). Since skin-to-skin moisture and friction have been hypothesized as the cause of ITD, a high-risk population for ITD is the bariatric and obese patient population. Others at risk include women with large pendulous breasts, persons with stocky necks, and children with skin folds.

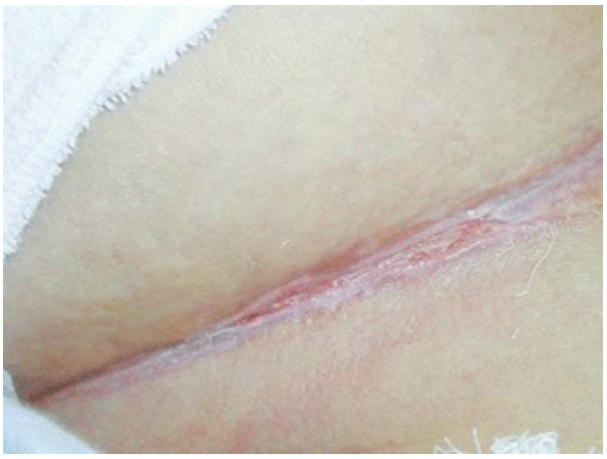


Figure 4-6. Intertriginous dermatitis (ITD). Intertrigo between the skin folds of the breast of a female patient with a body mass index of 60. Note the fissure at the base of the skin fold.

As moisture accumulates in a skin fold, the potential for growth of microorganisms exists.[56] As skin pH becomes more alkaline, Black et al.[49] report that ITD cultures have shown growth of the following organisms: *S. aureus*, group Aβ-hemolytic streptococcus, *Pseudomonas*, *Proteus mirabilis*, *Proteus vulgaris*, enterococci, vancomycin-resistant enterococci, and fungus such as *Candida*.

Careful assessment of skin folds for ITD is important. Typically, ITD begins as mild, mirror-image erythema of the skin.[49] With continued exposure to the moisture, the skin becomes more inflamed and macerated. Skin erosions, oozing, crusting, itching, pain, and odor may be present.[49] Efforts to prevent ITD focus on appropriate skin hygiene including keeping the skin clean and dry, using skin pH neutral products, avoiding abrasive and friction using skin care, and "pat" drying the skin. Treatment includes

continuing the skin care regimens for cleansing as well as, soaks with various astringent solutions, specialized dressings designed to be placed in skin folds to absorb moisture, and if candidiasis is present to use antifungal powders.[49]

Edema

Edema is abnormal swelling caused by accumulation of fluid in the tissues.[57] Swelling caused by edema commonly occurs in the hands, arms, ankles, legs, and feet (Fig. 4-7). It is usually linked to the venous or lymphatic systems.

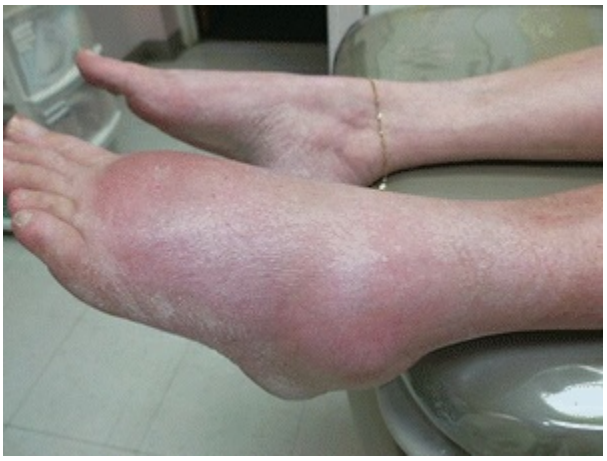


Figure 4-7. Assessment of foot edema. (©Ayello and Sibbald, used with permission.)

Edema may be generalized or local. It can appear suddenly but usually develops subtly—the patient may first gain weight, or wake up with puffy eyes. Many patients wait until symptoms are well advanced before seeking medical help.

Assess area for edema by comparing one extremity to another. Press firmly with finger tip, usually index finger, on edematous site for 5 seconds and release (Fig. 4-8). Edema is "pitting" when it does not return to its normal contour rapidly.[57] Document the amount of pitting edema that you observe. There is no consensus in how edema is documented. Some use descriptive words such as mild, moderate, or severe, while others use numerical and plus signs (+1, +2, +3, +4), follow your facility policy. Elevate if condition warrants, use care in moving and reposition edematous extremities and use appropriate skin care products. Chapter 14 also includes more advanced information about edematous conditions.

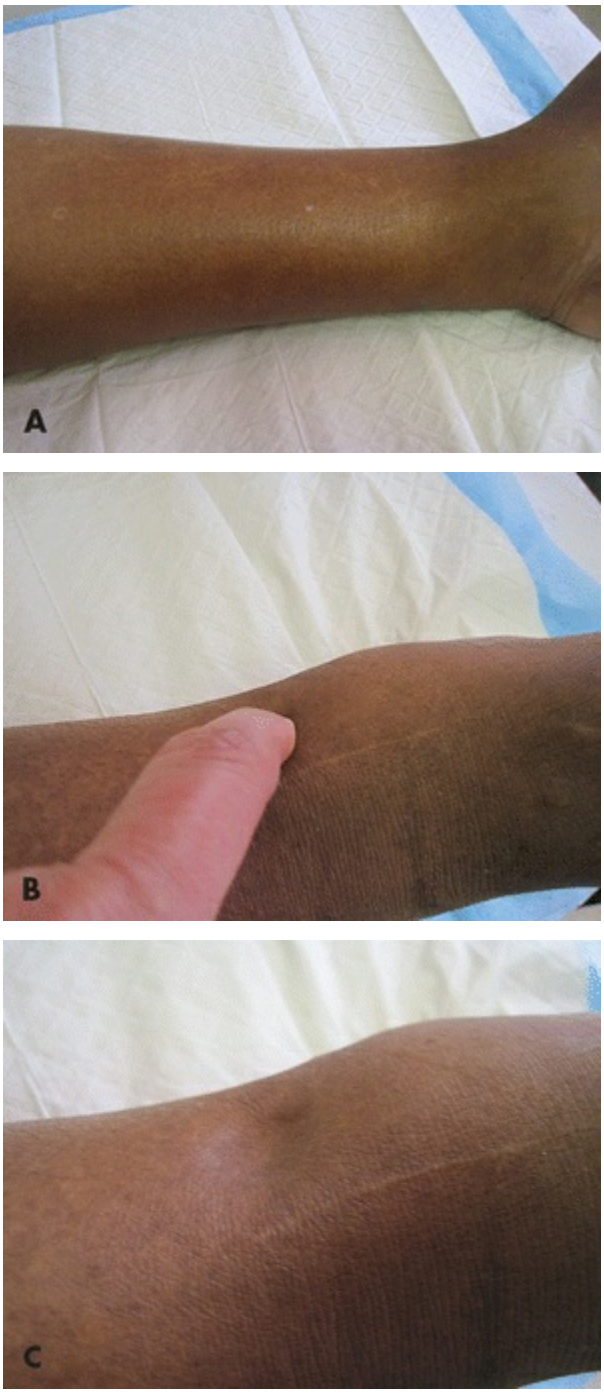


Figure 4-8. Assessing lower leg for edema. (**A)** Examine lower leg. (**B**) Using your finger, depress the skin. (**C**) Remove your finger, note dent in the skin to access edema. (Ayello, E.A., Sibbald, R.G., Quiambao, P.C.H., et al. "Introducing a Moisture-Associated Skin Assessment Photo Guide for Brown Pigmented Skin," *World Council of Enterostomal Therapists Journal* 34(2):18-25, 2014. ©Ayello and Sibbald, used with permission.)

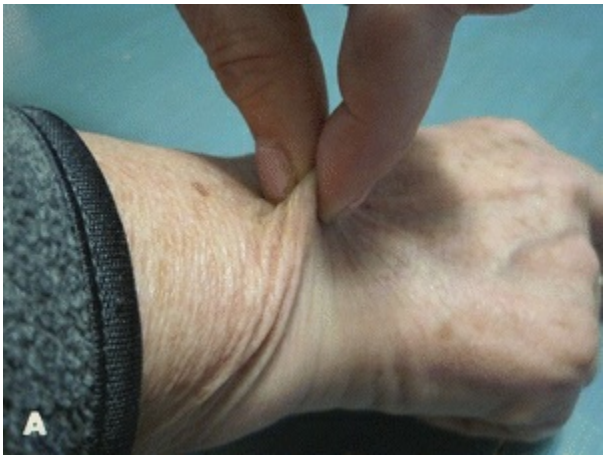
Practice Point

Assessing for pitting edema rating scale

1+ equals 2 mm depression
2+ equals 4 mm depression
3+ equals 6 mm depression
4+ equals 8 mm depression

Turgor

Turgor reflects the skin's resiliency and elasticity as well as hydration status. The assessment of skin turgor is used clinically to determine the extent of hydration, or fluid loss, in the body's tissue. The measurement is done by gently pinching up a skinfold (often on the back of the hand or the sternum region) between thumb and index finger so that it is raised for 3 seconds (Fig. 4-9). The skin is then released to observe how fast it returns to its normal position. If the skin remains elevated (tented) longer than 3 seconds, turgor is documented as decreased. With moderate-to-severe dehydration, decreased skin turgor causes the skinfold to remain elevated and only slowly return to its normal position. If no dehydration is present, the skin should return quickly to its normal position. Skin Turgor also can be decreased in the elderly population as the skin losses its elasticity with aging. It is better to use the sternum region to assess skin turgor in the elderly



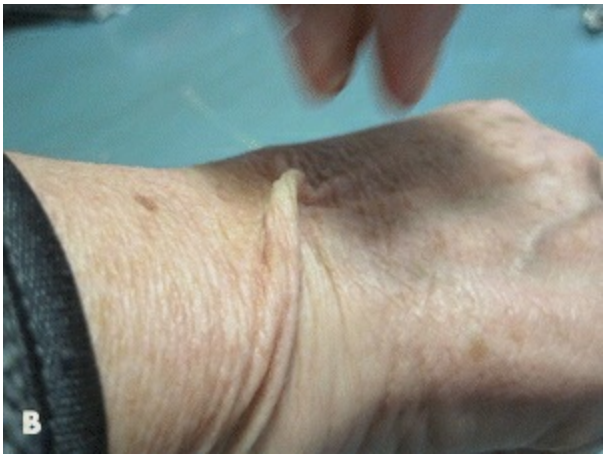


Figure 4-9. Assessing skin turgor. **(A)** "Tent" the skin. **(B)** Normally, skin returns to its original state quickly, but (as shown) a slow return to its original shape may indicate dehydration or effect of aging. (Ayello, E.A., Sibbald, R.G., Quiambao, P.C.H., et al. "Introducing a Moisture-Associated Skin Assessment Photo Guide For Brown Pigmented Skin," *World Council of Enterostomal Therapists Journal* 34(2):18-25, 2014. ©Ayello and Sibbald, used with permission.)

We have just reviewed what constitutes a basic wound assessment. Patient condition, clinical resources, and facility practices may support a more comprehensive and involved skin assessment. Usually comprehensive assessments are completed by those clinicians specializing in skin and dermatology. Comprehensive assessments would include skin inspection, palpation and olfaction, and observation of hair and nails and finally any skin alterations seen. Clinicians should be looking for and documenting any lesions, scars, bruising, areas of senile purpura (Fig. 4-10), or hemosiderin deposits (Fig. 4-11). An example of items to review is included in elements of a comprehensive skin assessment (Table 4-9).

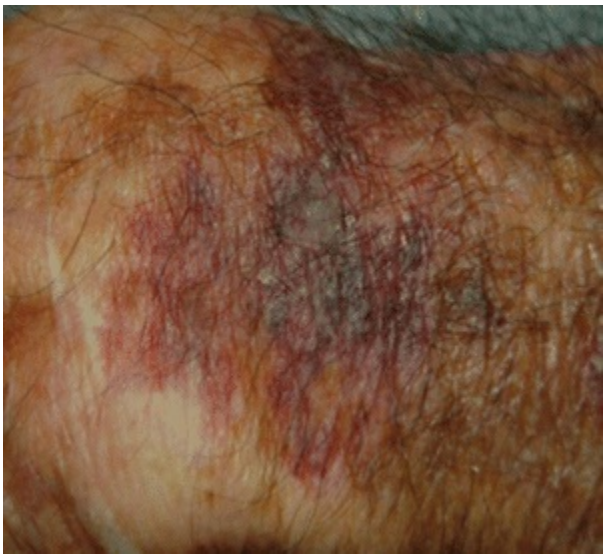


Figure 4-10. Senile purpura with xerosis.

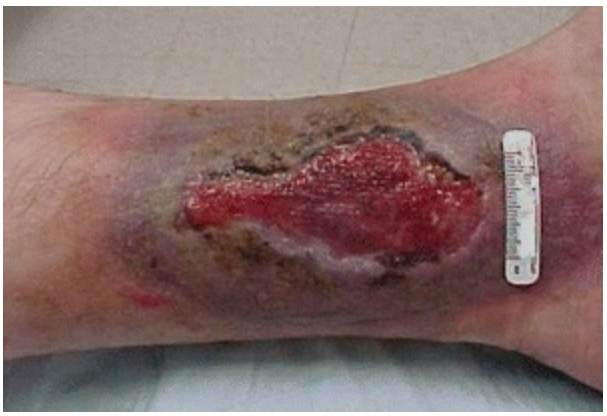


Figure 4-11. Hemosiderin deposit.

Table 4-9 Elements of a Comprehensive Skin Assessment

Consider the following criteria when performing a comprehensive skin assessment

Inspection

- Normally smooth, slightly moist, and same general tone throughout
- Tone depends on the patient's melanocytes—skin pigmentation continuum can vary from light ivory, deep brown, black, yellow to olive, light pink to dark ruddy pink, or red
- Pigmentation can exhibit:
  - Pallor: mucosa, conjunctivae
  - Cyanosis: nail beds, conjunctivae, oral mucosa
  - Jaundice: sclerae, palate, palms
  - Hyperpigmentation: increased (results from variation in melanin deposits or blood flow; palpate for skin temperature and for edema over these areas to assess circulation)
  - Hypopigmentation: decreased vascular/venous patterns, usually symmetric
  - Scars and bruises for location, color, length, and width

Palpation

- Moisture: perspiration
- Edema: extremities, sacrum, eyes
- Tenderness
- Turgor, elasticity
- Texture

Olfaction

- Normal body odor
- Absence of pungent odor
- May indicate presence of bacteria or infection
- Poor hygiene

Observation of Hair and Nails

- Hair
  - Hirsutism: excessive body hair
  - Alopecia: hair loss
- Nails (can reflect the patient's overall health)
  - Color, shape, contour
  - Clubbing, texture, thickness

Skin Alterations

- Previous scars
- Graft sites
- Healed ulcer sites

Skin Assessment Versus Wound

Assessment

Although not always given the priority in clinical practice, the skin or integumentary system should be part of the routine head-to-toe assessment of all patients. A skin assessment should include an actual observation of the entire body. It differs from a wound assessment in that a skin assessment looks at the patient's entire body and not just open wounds.

Practice Point

Factors affecting skin resilience[32]

a. Aging of skin
b. Critical illness
c. Malnutrition and dehydration
d. Excess moisture
e. Skin conditions that cause dryness

Implications for Practice

Alterations in skin integrity, perhaps due to skin tears or trauma or other skin conditions, may cause undue pain and suffering to patients. Healthcare professionals should have up-to-date information about prevention techniques, the appropriate use of dressings and tapes and, most importantly, the prevention of skin integrity injuries.

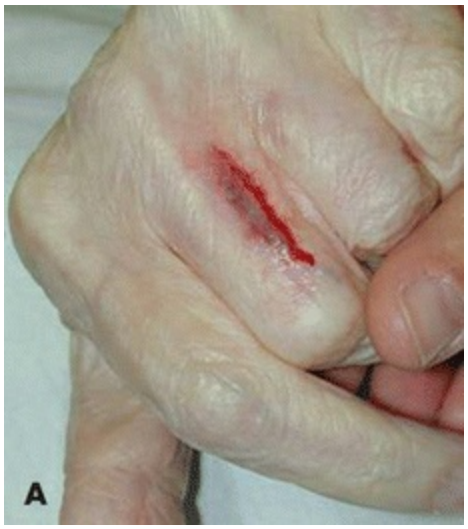
Skin Tears

The International Skin Tear Advisory Panel (ISTAP) defines skin tears as "*a wound caused by shear, friction, and/or blunt force resulting in separation of skin layers. A skin tear can be partial-thickness (separation of the epidermis from the dermis) or full-thickness (separation of both the epidermis and dermis from underlying structures).*"[58] Individuals at the extremes of age and the chronically or acutely ill have an increased skin tear risk.[59] An aging population coupled with an increased prevalence of chronic diseases translate into longer healing times for the majority of wounds, including skin tears.[60] Individuals suffering from skin tears

complain of increased pain, which in addition to other biopsychosocial factors associated with chronic wounds, such as physical disability, social needs, and mental anguish, may negatively impact an individual's quality of life.[61,62]

Healthcare professionals must become cognizant of which individuals are at risk for developing skin tears, how to prevent these wounds, and how to treat them once they occur. In recent literature, there has been increasing attention given to skin tears, but there has been no gold standard developed for their management. While the prevention of skin tears is the primary focus, healthcare professionals must be equipped to manage these wounds when they do occur. By recognizing which patients are at risk for skin tears, preventing skin injuries, and using appropriate nonadherent dressings, we can save patients undue pain and suffering.[58,63,64]

There has historically been limited interest in skin tears, as demonstrated by the paucity of quality publications addressing these wounds. There exists a perception among healthcare professionals that skin tears are minor wounds, not requiring focused attention, resulting in an inadequate scientific study devoted to these wounds.[59] In an effort to redirect awareness toward this largely unheeded healthcare issue, ISTAP, consisting of 12 internationally recognized key opinion leaders, was established. Key consensus statements were produced based on current knowledge and evidence focusing on the epidemiology, etiology, pathophysiology, assessment, prevention, treatment, and documentation of skin tears.[58] Subsequent work by ISTAP also included the development and validation of a skin tear classification system and a comprehensive tool kit to assist clinician with the development of a broad prevention program [63,65] (Fig. 4-12).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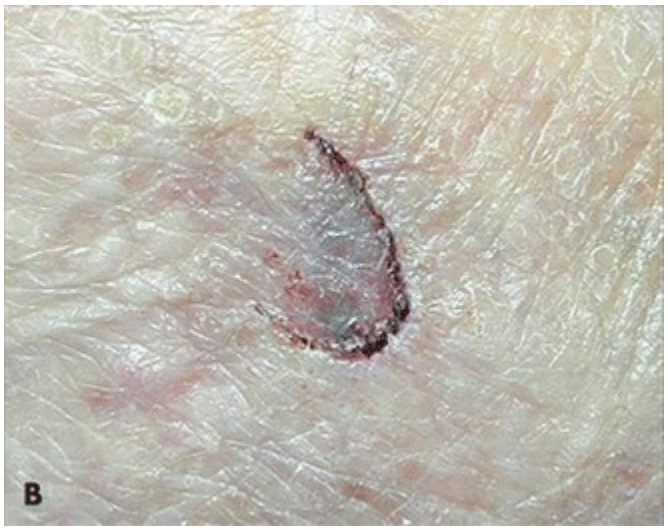
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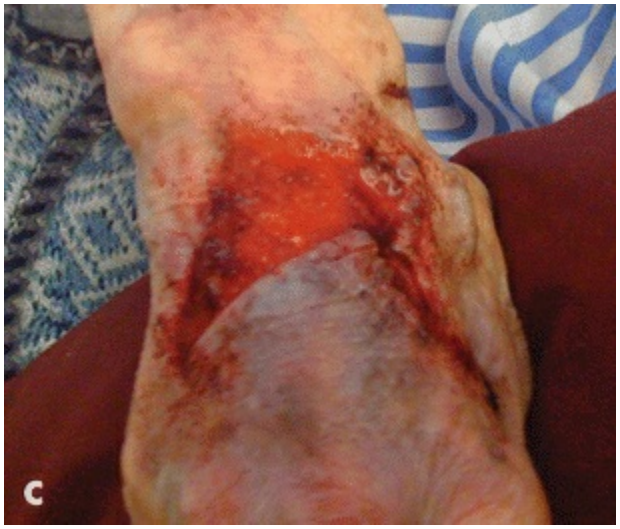
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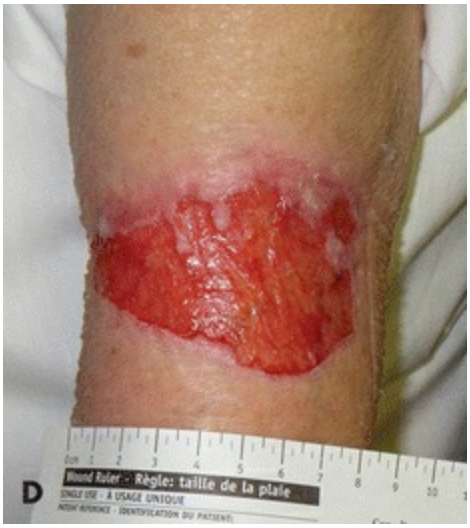

Figure 4-12. ISTAP Skin Tear Classification. **(A, B)** Type 1, no skin loss. Linear or Flap tear that can be repositioned to cover the wound bed. **(C)** Type 2: partial flap loss. Partial flap loss that cannot be repositioned to cover the wound bed. **(D)** Type 3: total flap loss. Exposing entire wound bed. (Copyright ISTAP 2014.)

Prevalence

Skin tears are perceived to be common in the extremes of age and critically ill. Although they are seen in practice, the prevalence of skin tears may be underreported, especially in the community.[66] Some researchers have hypothesized that the prevalence rates of skin tears are equal to or greater than those of pressure ulcers.[67–69] Despite these assumptions, limited prevalence studies have been conducted to support this hypothesis. Several prevalence studies conducted in LTC settings have reported skin tear prevalence rates between 14% and 42%.[70–73] A Canadian study conducted by LeBlanc et al.[71] reported a 22% prevalence rate in one LTC facility. Hanson et al.[74] reported prevalence rates of 6.3% and 6.4% in two rural U.S. nursing homes. These findings support the assumptions that the prevalence rates of skin tears, particularly in the LTC setting, closely resemble those of pressure ulcers.[66,75]

When skin tears are reported, the causative factor is often not known. When the cause is known, skin tears are frequently linked to wheelchair injuries, blunt trauma from accidently bumping into objects, transfers, or falls.[59,74,76] Studies[76,77] has shown that the key times skin tears occur are during the peak activity hours of 6:00 AM to 11:00 AM and 3:00 PM to 9:00 PM. In the elderly population, skin tears are often related to the environment.[78] In 1990, Payne and Martin[79] conducted a 3-month,

descriptive study in 10 long-term care facilities to describe skin tears, identify risk factors, and determine the rate of healing of skin tears. Among the predominant risk factors, impaired activity, mobility, sensation, and cognition all demonstrated an increased risk for skin tear development. McGough-Csarny and Kopac[80] conducted a similar study in a Veterans Affairs nursing home and concluded that dependency in activities of daily living (ADLs), sensory loss, limited mobility, use of assistive devices, and impaired cognition were risk factors for skin tear development. Patients who are dependent on others for total care are at the greatest risk for skin tears.[77] Dependent patients frequently acquire skin tears during routine activities, such as dressing, bathing, repositioning, and transferring. Independent ambulatory patients are at the second highest risk, and the majority of their skin tears occur on their lower extremities.[77,81] In the 2011 survey conducted by LeBlanc and Baranoski et al.,[58,59] the top causes of skin tears included equipment injury, patient transfers, falls, ADL, and treatment and dressing removal.

Although nearly 70% to 80% of skin tears occur on the arms and hands,[74,82] these wounds may occur on other areas of the body as well. Skin tears that occur on the back and buttocks are commonly mistaken for stage II pressure ulcers. Pressure may be a related cause in skin tears, but the etiology of skin tears differs from that of pressure ulcers.[58,63] Skin tears need to be documented as separate occurrences and not grouped into pressure ulcer categories. (See Chapter 13, Pressure Ulcers.)

Risk Factors

ISTAP developed a risk assessment pathway[63] (Fig. 4-13) to aid in the prediction of skin tears. The pathway is complemented by the ISTAP risk reduction program (Table 4-10), which will be discussed later in this chapter.

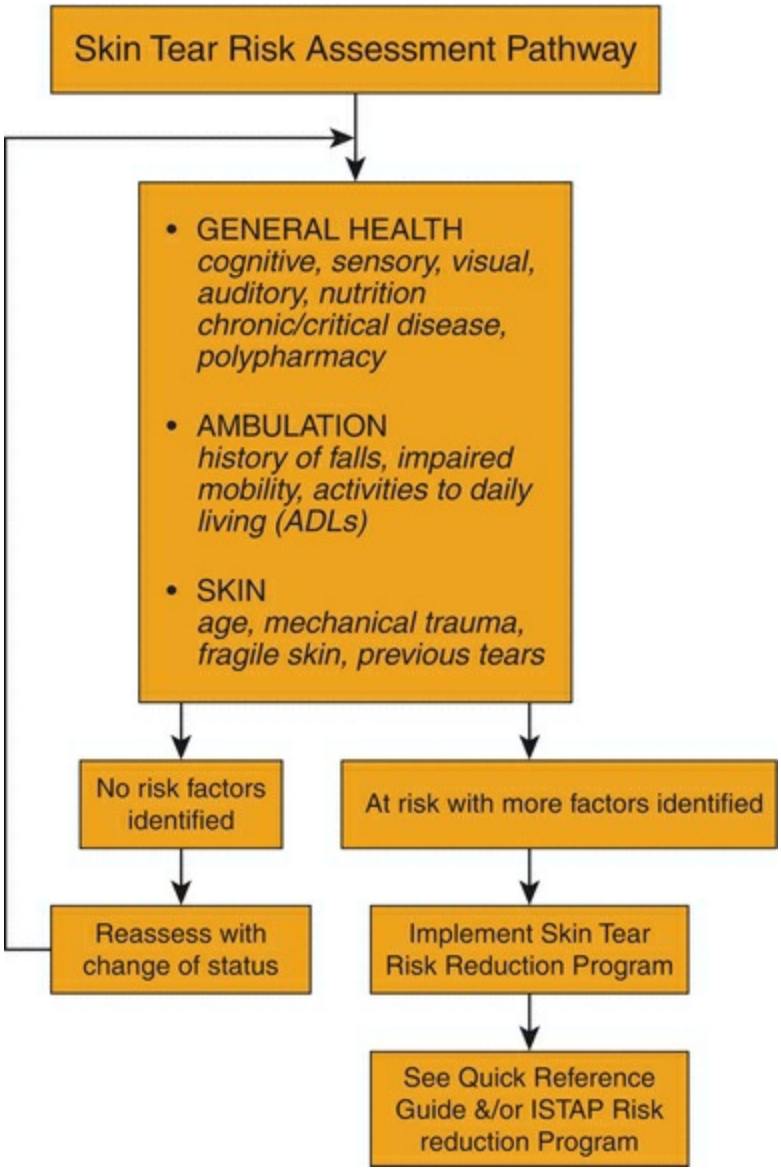

Figure 4-13. Skin tear risk assessment pathway.

Table 4-10 Quick Reference Guide: ISTAP Risk Reduction Program

Risk Factor	Individual	Care Giver/Provider
General health	• Educate the patient on skin tear prevention and promote active involvement in treatment decisions (if cognitive function not impaired) • Optimize nutrition and hydration	• Safe patient environment • Educate the client +/circle of care/ caregivers • Protect from self-harm • Dietary consult if indicated • Extra caution with extremes of BMI (<20 or >30) • Review polypharmacy for medications reduction/optimization
Mobility	• Encourage active involvement if physical function not impaired • Appropriate selection and use of assistive devices	• Daily skin assessment and monitor for skin tears • Ensure safe patient handling techniques/ equipment and environment (trauma, activities of daily living [ADLs], self-injury) • Proper transferring/repositioning • Initiate fall prevention program • Remove clutter • Ensure proper lighting • Pad equipment/furniture (bedrails, wheel chair, etc.) • Avoid sharp finger nails/jewelry when having patient contact
Skin	• Awareness of medication induced skin fragility (e.g., topical and systemic steroids) • Wear protective clothing (shin guards, long sleeves, etc.) • Moisturize skin (lubrication and hydration) • Keep finger nails short	• Individualize skin hygiene (warm, tepid not hot water, soapless or pH neutral cleaners, moisturize skin) • Avoid strong adhesives, dressings, tapes • Avoid sharp finger nails/jewelry with patient contact
Healthcare Setting		
• Implement comprehensive skin tear reduction program • Include skin tears in audit programs • Utilize validated classification system • Develop consultative team (wound care/dietary specialists, rehab/pharmacists)		

General Health

Altered Sensory, Auditory, and Visual Status

Chronic disease, often experienced by the aging population, can lead to altered sensory, visual, auditory, and neuropathic status. In addition, the critically ill and individuals with extremes of age (the very young or the very old) can also be afflicted with altered sensory status.[58] Aging skin puts individuals at risk for skin injury including acute traumatic wounds or skin tears. Care of the geriatric patient often involves managing persons who are among the oldest and fastest-growing segment of the population (85 years and older). As individuals age, the normal wound healing process also slows and the risk of dementia, diabetes, and vascular disease increases.

With the increase in the aging population, more individuals will be at risk for developing skin injuries including chronic wounds, and skin tears.[83,84]

Altered sensory, visual, auditory, and neuropathic status all contribute to increased fall risks.[83,84] History of falls has been strongly linked in the literature to an increased risk of skin tears.[59,71,78,81,83]

The critically ill, pediatric, and premature neonatal population can also be at an increased risk for skin tear development if alterations in sensory, visual, auditory, or neuropathic are experienced. Care should be taken to ensure that the individual and all those involved in their care are aware of the increased risk of skin tear and ensure protection from potential risk factors.[83–85]

Cognitive Impairment

Despite the paucity in the literature surrounding the causative factors of skin tears, there is evidence to suggest that altered levels of cognition in the elderly,[65] pediatric,[83] and critically or chronically ill individual[58] increases the risk of skin tear development.

Various types of dementia and other chronic illnesses lead to altered cognitive status and can be an added challenge to caregivers. Cognitive impairment can lead to decreased adherence to prevention programs due to lack of comprehension. Aggressive behavior and agitation associated with altered cognition and dementia can also increase the risk of blunt trauma and self-injury resulting in skin tears.[86]

The very young or developmentally challenged individual cannot be forgotten when managing skin tears. Lack of cognition or insight can potentially increase the risk of skin tears in this population.[85]

Nutritional Concerns

Adequate nutrition and hydration help maintain tissue viability.[87] Assessment of each individual's nutritional status is vital to insure health and well-being. The elderly can be at great risk for nutritional impairment. Of the hospitalized elderly, 25% to 30% are undernourished, while 46% to 61% are at risk for being undernourished.[87]

Dehydration can be assessed by monitoring intake, urine output, skin turgor, changes in weight, and lab values such as sodium and serum osmolality. With issues such as diarrhea, vomiting, increased body temperature, or heavily draining wounds more fluid intake is needed to

replenish lost fluid and electrolytes. Adequate hydration is essential in maintaining skin integrity. It can make a difference between a bump causing a bruise or a skin tear.[88]

Polypharmacy

Polypharmacy (i.e., the use of multiple medications that might predispose patients to drug interactions/reactions or confusion) is common among the elderly. Numerous medications can be associated with negative health outcomes (i.e., cutaneous reactions, falls), but more research is needed to further delineate the consequences associated with multiple drug use, especially in elderly patients.[89]

A variety of medications can affect the skin, the most common being corticosteroids, which can interfere with epidermal regeneration and collagen synthesis.[86,88] Steroids may be a contributing factor to skin tear development. Polypharmacy has also been indicated to be an independent risk factor for falls.[89,90] Pervin's[89] research concluded that individuals receiving four or more medications are at a greater fall risk. In a recent consensus document pertaining to the prevention, assessment, and treatment of skin tears, LeBlanc et al.[58] identified falls as a major risk factor in the development of skin tears as well.

Mobility

Mobility-Related Issues

When skin tears are reported, the causative factor is often not known.[58,79] Nevertheless, skin tears are frequently linked to wheelchair injuries, falls, transfers, or blunt trauma from bumping into objects.[64]

Assistance with Activities of Daily Living

Individuals frequently acquire skin tears during routine activities of dressing, bathing, positioning, and transferring, and those who are dependent on others for total care are at the greatest risk for skin tears.[81,91] Individuals who are independent in ambulation report high numbers of skin tear occurring primarily on the lower extremities.[92–94] A 2011 survey reported that the perceived top causes of skin tears included equipment

injury, patient transfers, falls, ADL, treatment, and dressing removal.[59]

History or Risk of Falls

Falls pose a serious risk of skin tears for the elderly. An average nursing home with 100 beds reports 100 to 200 falls annually.[95] Predisposing factors for falls include unsteady gait and balance, weak muscles, poor vision, medications, and dementia. In addition, other factors, such as poor lighting, loose rugs, poorly fitting shoes, floor clutter, continence urgency, and beds or toilets without handrails, also may cause falls.[96] Furthermore, medical conditions such as low blood pressure, stroke, Parkinson's disease, arthritis, Ménière's disease (affects the middle ear—causes vertigo), poorly controlled diabetes, poorly controlled epilepsy, brain disorders, and thyroid problem increase the elderly client's risk for falls.[96] Falls have been associated with contributing to the development of skin tears in the elderly and compromised populations.[59]

Mechanical Trauma (Not Related to Mobility Aids)

Intrinsic factors, such as age, pertain to an individual's inherent biologic or genetic makeup. Extremes in age impact not only on how individuals heal but also on their susceptibility to developing a wound.[64] With increasing age, individuals experience dermal and subcutaneous tissue loss, epidermal thinning, and serum composition changes, all of which cause decreased skin surface moisture.[33–35,88] In turn, the skin's elasticity and tensile strength decreases. In the neonatal population, mechanical trauma is the number one cause of skin tears.[85]

Skin Changes Related to Extremes of Age and Critically Ill

Skin tears commonly occur on individuals at the extremes of age (elderly to neonate), the critically ill or medically compromised, and to those requiring assistance with personal care. As the skin ages, there are many changes that occur within the dermis making the skin more susceptible to skin tears as previously addressed.

Prevention

Little has been written about the prevention of skin tears. Following tried and true guideline, recommendation can help prevent skin tears. Best practice guidelines and the ISTAP risk reduction prevention program, gleaned from the literature, may prevent many skin tears.[58,59,63–65]

- Encourage your colleagues and the patient's family members to use proper positioning, turning, lifting, and transferring.
- Promote the use of long sleeves and pants to add a layer of protection.
- Secure padding to bed rails, wheelchair arm and leg supports, and any other equipment that may be used.
- Use nonadherent dressings on frail skin. Always remove these products gently to prevent skin injury.
- Use skin sealants, liquid bandage, or soft silicone or foam dressings to protect vulnerable skin from adhering tapes and dressings.
- Use stockinettes, gauze wrap, or a similar type of wrap to secure dressings and drains rather than tape.
- Use pillows and blankets to support dangling arms and legs.
- Minimize the use of soap and alcohol solvents; consider the use of no-rinse, waterless, or liquid gel cleansers.
- Avoid scrubbing skin when bathing; pat skin dry rather than rubbing it dry.
- Apply a moisturizing or emollient agent to dry skin.
- Provide a well-lit environment to prevent falls.
- Educate staff on the importance of gentle care.

Research[78] has shown that skin tears can be reduced in nursing homes when skin care protocols are used.[64,92] Skin tears in an LTC facility declined from 23.5% to 3.5% with the implementation of a no-rinse, one-step, bed bath protocol rather than soap and water.[97] Skin tears were significantly decreased in two different rural nursing homes when skin care protocols were introduced. Hanson et al. found that skin tear prevalence was reduced from 6.3% to 1.4% in nursing home A and 6.4% to 3.3% in nursing home B.[74] By educating staff and implementing the above-mentioned skin care protocols, skin tears were reduced from a monthly average of 18 to 11.[98] Another study emphasizes the importance of educating nurses and nursing assistance, in a 10-month descriptive study with 30 patients with Alzheimer disease, 26 of the 30 patients remained free of skin tears using a preventive skin program.[99,100] A study of 416 RNs in two affiliated hospitals demonstrated increased ability to identify and assess skin tears,

differentiation of skin tear categories, and increased knowledge of treatment by using a web-based educational program.[101]

Skin Tear Classification System

The initial classification system for skin tears evolved in the late 1980s from the work of Regina Payne and Marie Martin. Their pilot research study led to the development of the Payne-Martin Classification System for skin Tears.[79,82] An international cross-sectional survey conducted by LeBlanc, Baranoski, and Regan found that the Payne-Martin Classification system was not widely adopted into global practice and that clinicians overwhelmingly supported a simplified classification system for skin tears.[59] In response to these findings, the International Skin Tear Advisory Panel (ISTAP) developed and validated the ISTAP Skin Tear Classification System[65] (Fig. 4-12). This system is user friendly and can be used by all healthcare providers to document skin integrity insult.

Patient Teaching

Alert the patient/family/caregiver that skin tears often occur over areas of senile purpura in the elderly. (See *Senile purpura*.)

Skin Tear Management

The management or treatment of skin tears varies according to institution and clinician practices. Skin Tear Management includes assessing the insult, control of bleeding, wound cleaning, remove any necrotic tissue/debris, realign the wound edge/flap or pedicle, classify, measure, and document type of skin tear (type 1, 2, or 3). Basic goal of treatment is to treat the cause, avoid further trauma, avoid infection, control of pain, manage exudates, and use a moist wound therapy, nonadherent wound dressing (Fig. 4-14).

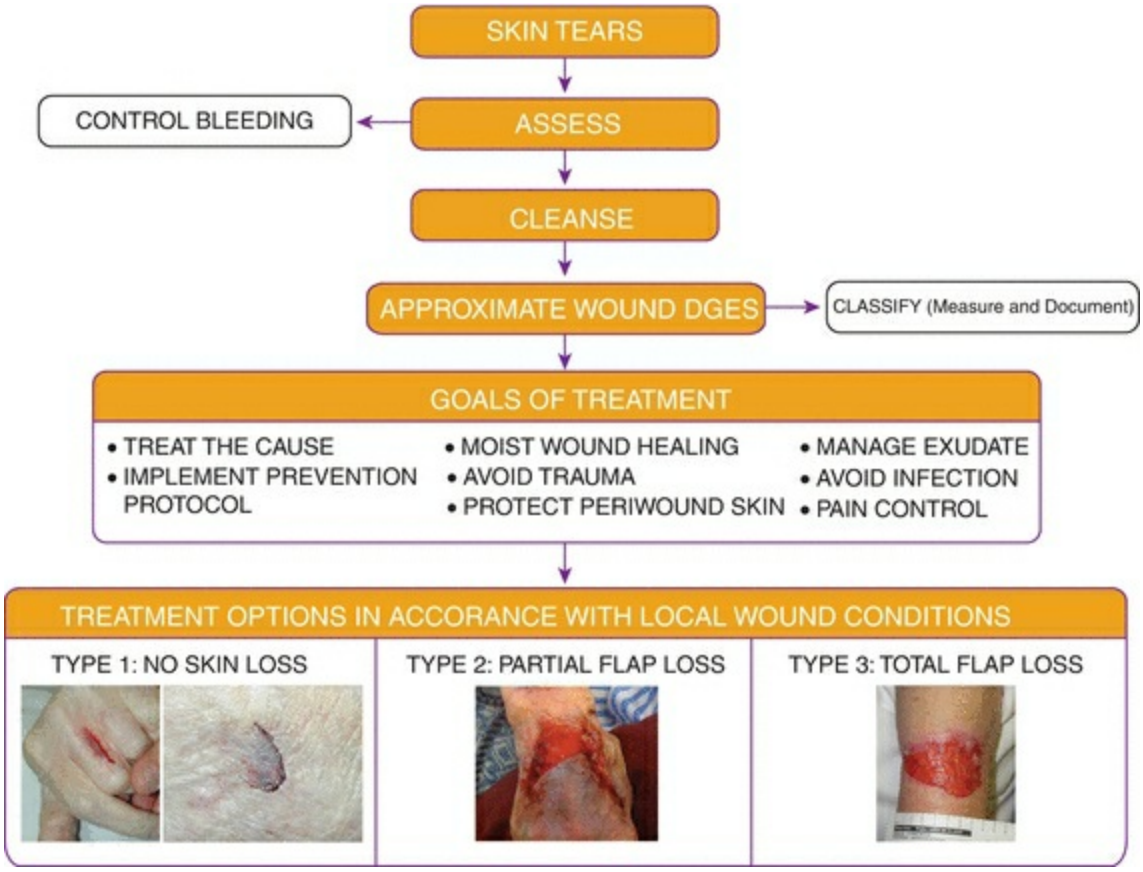

Figure 4-14. Skin tear algorithm.

Patient Teaching

Tetanus is an acute, often fatal disease caused by wound contamination with *Clostridium tetani*. Human tetanus immunoglobulin (TIG) neutralizes circulating tetanospasmin and toxin in the wound but not toxin that is already fixed in the nervous system. It should be given according to individual institutional policy, to individuals with interruption of the skin integrity by a nonsurgical mechanism who have not received a tetanus toxoid (Td) inoculation in the past 10 years. The TIG should be given before wound debridement because exotoxin may be released during wound manipulation.[90]

Skin Failure

We've discussed throughout this chapter that skin is the largest organ of the

body. Can the skin as an organ fail? The literature supporting skin failure is very limited. The concept was first discussed in 1989 by Goode and Allman who noted "multiple/multiorgan failure as a terminal stage of many diseases that occurs as the body wastes away."[102] They concluded that the skin is susceptible to failure and death must be considered in multiorgan death syndrome.[102] The term skin failure was again used in 1991 by LaPuma.[103] Patients who are at end stages of life and in the intensive care setting was the focus of an editorial in 1993 regarding skin and underlying tissue damage.[104] Leijten et al. discussed chronic skin failure in 1996 related to older adults with multiple comorbidities that can lead to pressure ulcers, especially at the end of life.[105] Hobbs et al. discussed the condition of skin failure at a national program as an abstract in 2000.[106]

The definition of skin failure has not been clinically defined or well accepted in the healthcare setting. The first written definition of skin failure was described in 2006 by Langemo and Brown in their article "Skin Fails Too: Acute, Chronic and End Stage Skin Failure."[107]

Skin failure defined by Langemo and Brown[107] is "an event in which the skin and underlying tissue die due to hypoperfusion that occur concurrent with severe dysfunction or failure of other organ systems." They further break this definition down to three types of events that trigger skin failure: acute skin failure, chronic skin failure, and end-stage skin failure.[107] Diagnostic testing is currently not available to detect tissue necrosis in its decisive stages.

- Acute skin failure is an event in which skin and underlying tissue die due to hypoperfusion concurrent with a critical illness.
- Chronic skin failure is when skin and underlying tissue die due to hypoperfusion concurrent with an ongoing chronic disease state.
- End-stage skin failure is when skin and underlying tissue die due to hypoperfusion concurrent with the end of life.[107]

Skin Changes at Life's End (SCALE)

Literature on skin changes at life's end is limited. In 2008, key opinion leaders convened to discuss this needed topic.[108] "General agreement was reached that like any other organ of the body, skin is subject to a loss of integrity due to internal and external insults. Contrary to popular myth, not all pressure ulcers are avoidable. The panel concluded that our current

appraisal of the complex skin changes at life's end and terminal pressure ulcers (including the Kennedy Terminal Ulcer) is limited. Additional scientific research and the consensus of expert knowledge are necessary to assess the important etiological factors of SCALE, to clinically describe and diagnose the conditions, and to recommend appropriate pathways of care. Knowledge transfer into practice techniques must then be implemented for improved patient outcomes. This process must include clinicians, laypeople, and policy makers concerned with the care of people at life's end to adequately address the medical, social, legal, and financial ramifications of SCALE."[108] SCALE will be discussed further in Palliative Care Chapter 23.

Summary

The skin is the largest organ of the body and commonly the most forgotten. Skin is exposed daily to environmental irritants and chemicals as well as physical and mechanical injury, any of which may lead to impaired skin integrity. This chapter provided an overview of skin structure and criteria for a skin assessment versus a wound assessment. Identification and classification of skin tears as an exemplar for acute traumatic skin injury was also presented, which includes skin tear risk factors and prevention opportunities, as well as treatment strategies. The importance of identifying common skin conditions, specifically xerosis, pruritus, including the role of breaking the itch–scratch–itch cycle and MASD are described. The use of moisturizers in their treatment was also highlighted. Skin Failure and SCALE, two multifaceted subjects regarding dysfunctional skin processes were also introduced.

PATIENT SCENARIO

Clinical Data

Mr. JP is a 75-year-old white male with a history of cardiac disease, COPD, and renal disease. He has a pacemaker and a defibrillator for the past 5 years. He has a 20-year history of being on warfarin. He is frequently in and out of the hospital with pulmonary and respiratory distress. Due to his cardiac problems, he has frequent bouts of fluid retention causing him to have edematous legs and feet. He was at home

ambulating to the bathroom when he knocked his walker over onto his edematous foot. Although he did not fall, 24 hours later he complained of pain, discoloration, and more swelling to his left foot (Fig. 4-15). The right foot was normal. His wife brought him to the emergency room where he was subsequently admitted to a medical surgical unit.

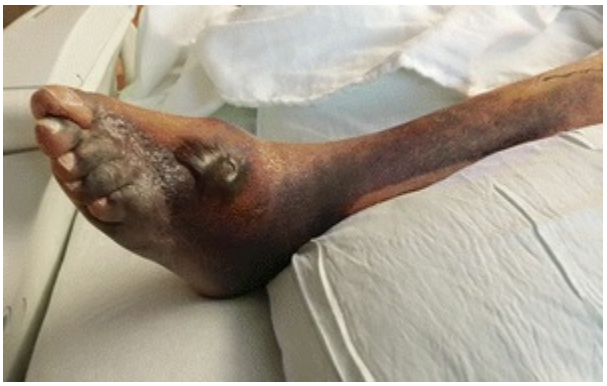

Figure 4-15. Initial presentation of left foot.

There, he was evaluated by a surgeon who said "*this needs to come off*" and recommended that the foot be immediately amputated. Mr. JP and his wife refused. JP's primary physician then took over the case and after discussion with the family, a wound care doctor and podiatrist were called in to handle the case. The joint consultation of these physicians resulted in a recommendation of a different approach. The treatment plan that they presented was as follows: keeping the foot elevated, drying out the blister on the top of the foot by using povidone–iodine, and allowing the blood in the foot to dissipate on its own. Basically, they felt that the best treatment was a slow medical management, with good skin and wound care and getting him up and mobile over time was the best approach. So this conservative management plan was implemented (Fig. 4-16).

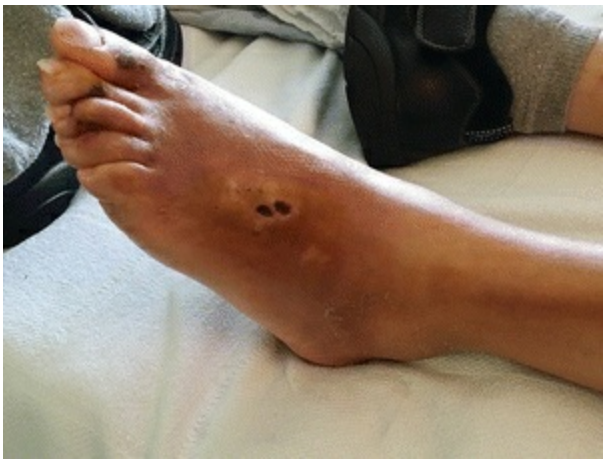

Figure 4-16. Left foot after 2 months of treatment

During his initial skin assessment on the medical surgical unit, the nurse noted numerous areas of skin tears and bruising on his entire back (Fig. 4-17). Mr. JP said that this occurred when he was dragged from the ambulance stretcher to the emergency department gurney. At that time, Mr. JP immediately complained "*my back, my back*." The ED staff did nothing. The medical–surgical unit nurse also noted that on his left arm above the elbow was a large full-thickness skin tear. Mr. JP had no idea how that skin tear occurred (Fig. 4-18). As you recall, the upper extremities are the most common place for skin tears. Because the skin tear is full thickness, it was documented as a type 3 using the ISTAP classification system. The numerous skin tears on his back were classified as types 2 and 3.

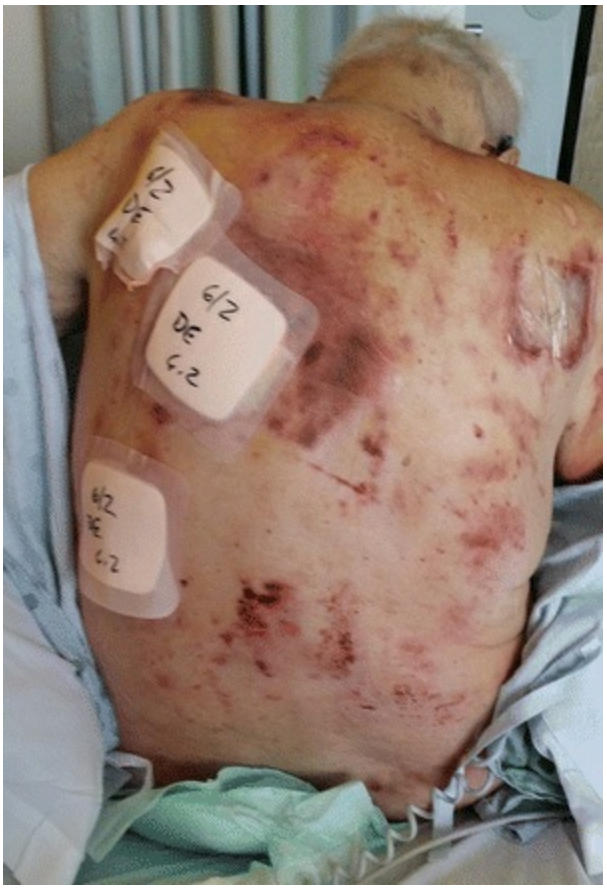

Figure 4-17. Initial presentation of skin tears on his back.

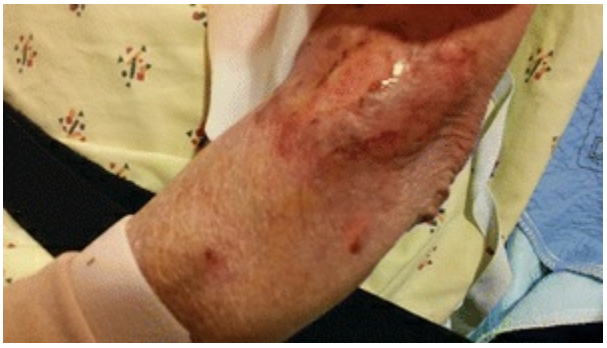

Figure 4-18. A initial presentation of skin tear on left arms.

Case Discussion

Mr. JP had numerous skin care problems that needed to be addressed. Because he already has several skin tears, Mr. JP is assessed as being at high risk for further developing more skin tears. Assessment of his back revealed seven skin tears; four type 3 and three type 2. His left arm also revealed a type 3 skin tear (ISTAP classification system).

Implementing strategies to prevent more skin tears from occurring included protecting other areas of risk on his arms by encouraging him to wear arm protectors to add a layer of padding to protect his skin (Fig. 4-19). This also has the added benefit of keeping him warm as he often complained of feeling chilly. As part of the prevention strategies, staff were reminded of proper transfer techniques such as not grabbing Mr. JP's arms or dragging his back across the mattress when assisting him out of bed.

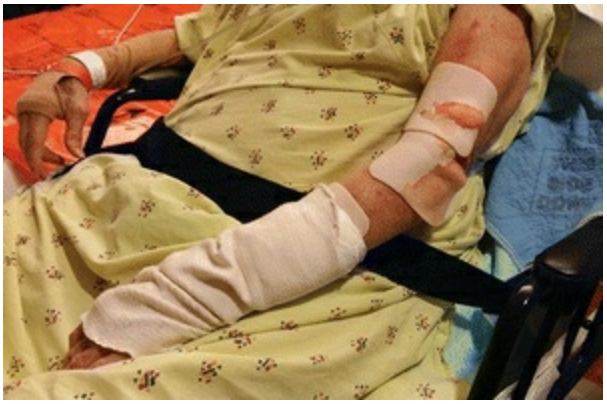

Figure 4-19. Padding the arms for protection and exudate strikethrough on dressings. Dressing needs to be changed.

Local care of Mr. JP's skin tears was required. After cleansing with normal saline, a soft nonadherent silicone dressing was applied to each of the skin tears on his back and arm. Nonadherent silicone dressings were chosen as they would not cause any further skin damage during dressing removal. Moisturizing creams were used over other areas of senile purpura and areas of xerosis (dry skin) that were thought to be at risk. Mr. JP complained about the leaking and wetness from the dressings on his arm. The dressing was overwhelmed with exudate (Fig. 4-19). Staff were reminded to change the dressings every 3 days or more frequently if needed in order to address the issue of dressing strikethrough (Fig. 4-20). After 2 weeks of using the nonadherent dressings, changing them when needed, before fluid was leaking, and using appropriate transfer techniques, many of the skin tears on his back healed (Fig. 4-21).

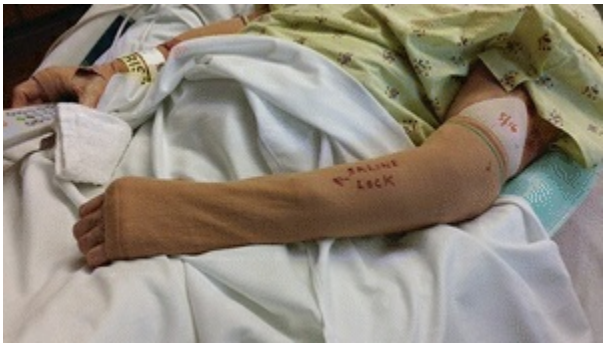

Figure 4-20. Better padding of the arm with no dressing strikethrough on dressing.

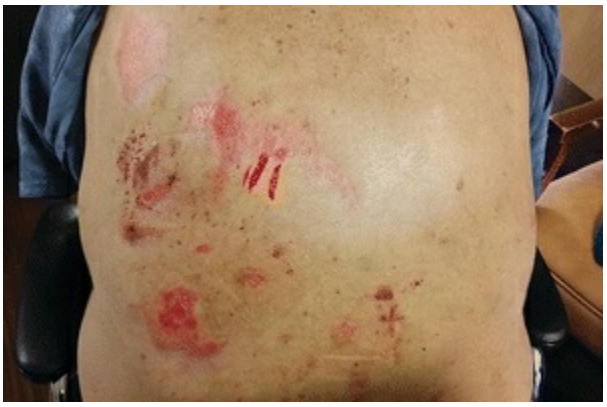

Figure 4-21. Skin tears on his back after 2 weeks of treatment.

Mr. JP remained in the hospital for 2 weeks and was then transferred to a rehabilitation facility. During that time, he developed no new skin tears, and a wound care nurse came to see him frequently and continued to treat the skin tears with use of nonadherent silicone dressings. Figure 4-22 shows the healed skin tears on his left arm after 6 weeks. After 2 months of the conservative treatment for his left foot, most of the discoloration and edema and the blood blister is resolved. Most importantly, Mr. JP has been discharged home and still has his foot and leg.

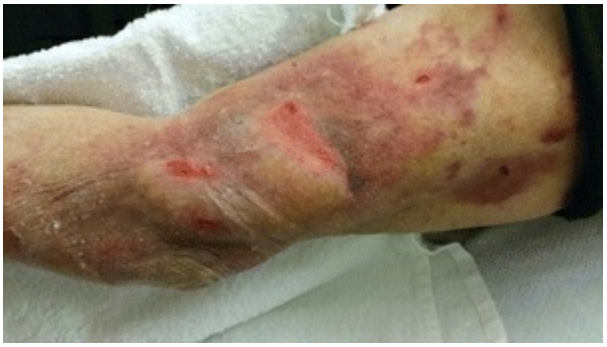

Figure 4-22. Left arm skin tear after 6 weeks of treatment with nonadherent dressing. The original skin tear has healed. Note scarring from previous skin tears.

Show What You Know

1. While bathing a patient, you notice some flakes of skin on the washcloth. Which layer of the skin is this?

A. Stratum granulosum
B. Stratum spinosum
C. Stratum lucidum
D. Stratum corneum

2. Which one of the following is a normal function of the skin?

A. Synthesis of vitamin K
B. Elimination of carbon dioxide
C. Regulation of glucose levels by the Langerhans cells
D. Thermal regulation by skin blood flow dilation or constriction

3. What is the role of keratinocytes in skin?

A. Differentiation
B. Cross-talk to fibroblasts
C. Participating in BMZ
D. Maintenance and repair of the barrier

4. Which one of the following is NOT considered part of a routine skin assessment?

A. Color
B. Turgor
C. Temperature

D. Ankle–brachial index (ABI)

5. Which one of the following patients is most at risk for skin tear injury?

A. A 22-year-old male postoperative for an inguinal hernia repair
B. A 37-year-old male with a fractured humerus
C. A 64-year-old female 3 days post–cataract extraction
D. A 72-year-old female with rheumatoid arthritis on steroid therapy

6. Using the ISTAP system, a partial-thickness flap loss that cannot be repositioned to cover the wound bed is classified as:

A. type 1.
B. type 2.
C. type 3.
D. type 4.

7. Which of the following interventions for a resident in a long-term care facility with a skin tear on the lower right leg should you question?

A. Clean the patient daily using detergent.
B. Pad the wheelchair arm and leg supports.
C. Apply a nonadherent dressing to the skin tear.
D. Encourage the patient to wear soft, fleece-lined pants.

8. Which one of the following should be included in the care plan of a person with xerosis?

A. Have the patient shower daily.
B. Use a deodorant soap.
C. Dry the skin completely with vigorous rubbing.
D. Apply an emollient immediately after bathing.

9. Which of the following best defines pruritus?

A. Multiple blisters on the skin
B. Traumatic open area on the skin
C. Itchy skin
D. Weepy skin

10. _____ is defined as an event in which the skin and underlying tissue die due to hypoperfusion that occur concurrent with severe dysfunction or failure of other organ systems.

A. Xerosis

B. Skin failure
C. Skin tear
D. Pruritus

11. In assessing a patient's skin, you find a raised area larger than 1 cm that is filled with serous fluid. This should be correctly documented as a:

A. papule.
B. vesicle.
C. pustule.
D. bulla.

12. A patient has skin damage from perspiration and friction under his abdominal pannus and the culture comes back as *Candida*. The best way classify this skin damage is as:

A. peristomal skin damage.
B. periwound maceration.
C. incontinence-associated dermatitis.
D. intertriginous dermatitis.

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Acute and Chronic Wound Healing

5

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Objectives

After completing this chapter, you'll be able to:

- describe the physiology of normal and abnormal wound healing
- discuss the cascade of wound healing events
- compare and contrast acute and chronic wound healing
- discuss the effects of biofilms on wound healing
- explain the principles of wound bed preparation and TIME in the management of patients with wounds.

Wound Healing Events

When a patient experiences tissue injury, it's essential that hemostasis is rapidly achieved and tissue is repaired to prevent invasion by pathogens and restore tissue function. The process of wound healing is a complex sequence of events that starts when the injury occurs and ends with complete wound closure and successful, functional scar tissue organization. Although tissue repair is commonly described as a series of stages, in reality it's a continuous process during which cells undergo a number of complicated biological changes to facilitate hemostasis, combat infection, migrate into the wound space, deposit a matrix, form new blood vessels, and contract to close the defect.

However, wound closure isn't a marker of healing completion; the wound continues to change, in a process called remodeling, for up to 18 months postclosure. During this prolonged phase of remodeling and

maturation, the closed wound is still quite vulnerable.

Patient Teaching

Remind your patient that the process of wound healing can take up to 18 months. Although the wound may appear to be closed, changes are occurring in the underlying tissue. This means that the wound is still vulnerable to damage. Tell the patient to seek professional advice if he or she has any concerns about the wound.

Wound Healing Cascade

The process of healing is usually divided into four phases—hemostasis, inflammation, proliferation/ repair, and maturation/remodeling—each of which overlaps the others while remaining distinct in terms of time after injury (Fig. 5-1).

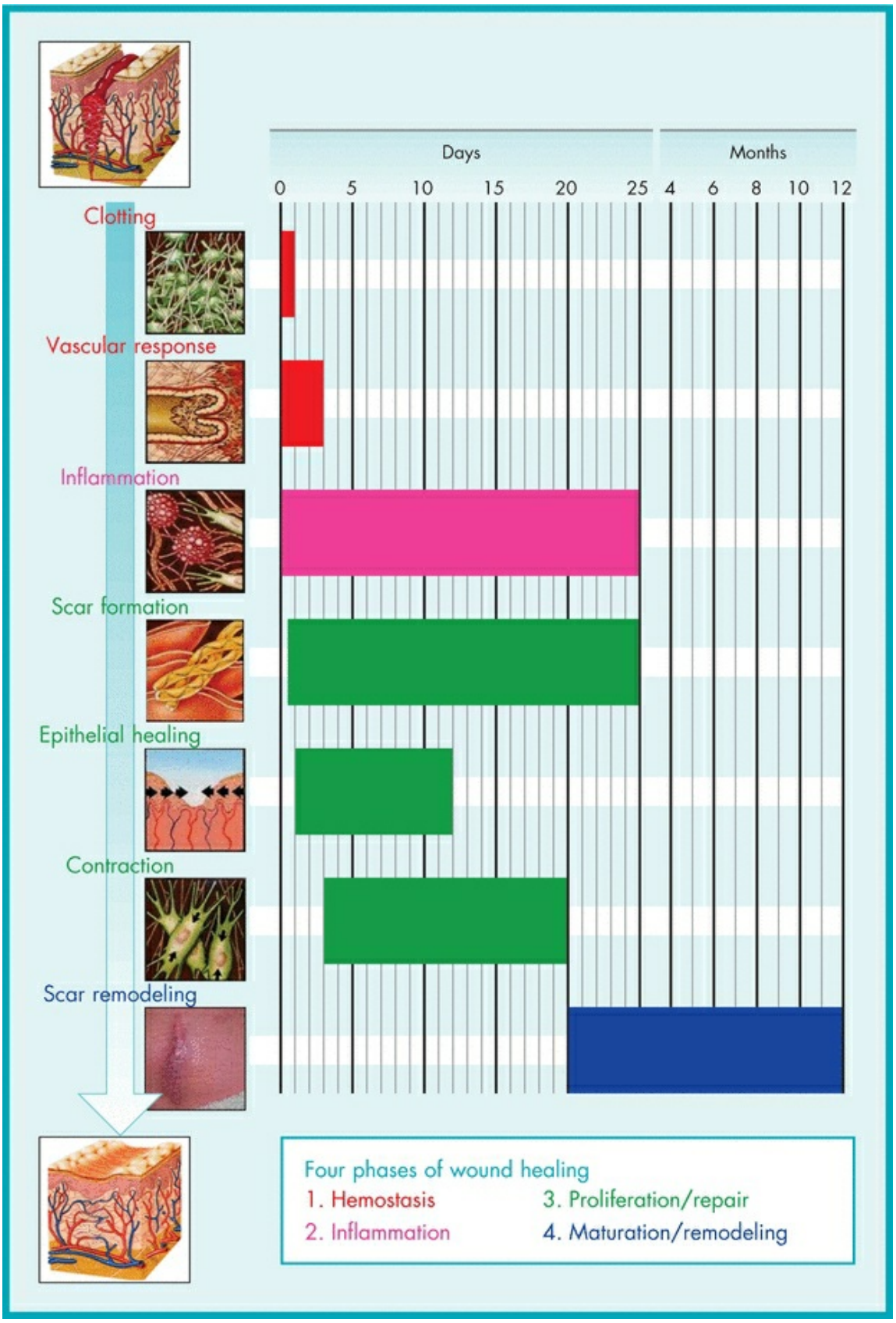


Figure 5-1. Sequence of molecular and cellular events in skin wound healing.

Patient Teaching

Inform your patient that the process of healing is often separated into four different phases. When a wound is being assessed, the clinician is looking for signs of progress as well as deterioration.

Hemostasis

The disruption of tissue following injury causes hemorrhage, which initially fills the wound and exposes the blood to various components of the extracellular matrix (ECM).[1] Platelets aggregate and degranulate, which activates factor XII (Hageman factor), resulting in clot formation and hemostasis. Hemostasis stops hemorrhage at the site of blood vessel damage. This is essential as it preserves the integrity of the closed and high-pressure circulatory system to limit blood loss. A fibrinous clot forms during coagulation, acting as a preliminary matrix within the wound space into which cells can migrate.

After the fibrin clot forms, another mechanism is activated as part of the body's defense system—fibrinolysis—in which the fibrin clot starts to break down. This process prevents clot extension and dissolves the fibrin clot to allow ease of further cell migration into the wound space,[2] allowing the next stage of healing to proceed.

Inflammatory Phase

As the fibrin clot is degraded, the capillaries dilate and become permeable, allowing fluid into the injury site and activating the complement system. The complement system is composed of a series of interacting, soluble proteins found in serum and extracellular fluid that induce lysis and the destruction of target cells. C3b, a complement molecule, helps bind (opsonize) neutrophils to bacteria, facilitating phagocytosis and subsequent bacterial destruction.

Cytokines and some proteolytic fragments that are hemoattractive are also found in the wound space.[2] Their abundance and accumulation at the site of injury initiate a massive influx of other cells. The two main inflammatory cells—neutrophils and macrophages—are attracted to the wound space to mount an acute inflammatory response.[3]

Neutrophils appear in a wound shortly after injury and reach their peak number within 24 to 48 hours; their main function is to destroy bacteria by

the process of phagocytosis. Neutrophils have a very short life span: After 3 days without infection, their numbers reduce rapidly.

Tissue macrophages are derived from blood monocytes and arrive approximately 2 to 3 days after injury, followed by lymphocytes. Like neutrophils, macrophages also destroy bacteria and debris through phagocytosis; however, macrophages are also a rich source of biological regulators, including cytokines and growth factors, bioactive lipid products, and proteolytic enzymes, which are also essential for the normal healing process.[2,4]

Cytokines, Growth Factors, and Chemotaxis

Cytokine is a broad term that includes such molecules as growth factors, interleukins, tumor necrosis factors, and interferons. These molecules act on a variety of cells by exerting a wide range of biological functions by means of their specific receptors on target cells or proteins. Pathogens, endotoxins, tissue degradation products, and hypoxia are all factors that stimulate cells to produce cytokines following injury. The main cellular sources for these cytokines are platelets, fibroblasts, monocytes and macrophages, and endothelial cells. These cells are involved in physiological as well as pathological conditions (e.g., tumors), although in wound healing they play an important role as mediators. Cytokines regulate cell proliferation, migration, matrix synthesis, deposition and degradation, and inflammatory responses in the repair process (Table 5-1).

Table 5-1 Major Cytokines Involved in Wound Healing

Cytokine	Cell Source	Biological Activity
Proinflammatory Cytokines		
Tumor necrosis factor–alpha (TNF-α)	Macrophages	↑ PMN margination and cytotoxicity ↑ MMP synthesis
Interleukin-1 (IL-1)	Macrophages, keratinocytes	↑ Fibroblast and keratinocyte chemotaxis ↑ MMP synthesis
Interleukin-6 (IL-6)	Macrophages, keratinocytes, PMNs	↑ Fibroblast proliferation
Interleukin-8 (IL-8)	Macrophages, fibroblasts	↑ Macrophage and PMN chemotaxis ↑ Collagen synthesis
Interleukin-γ	Macrophages, T lymphocytes	↑ Macrophage and PMN activation ↓ Collagen synthesis ↑ MMP synthesis
Anti-inflammatory Cytokines		
Interleukin-4 (IL-4)	T lymphocytes, basophils, mast cells	↓ TNF-α, IL-1, IL-6 synthesis ↑ Fibroblast proliferation, collagen synthesis
Interleukin-10 (IL-10)	T lymphocytes, macrophages, keratinocytes	↓ TNF-α, IL-1, IL-6 synthesis ↓ Macrophage and PMN activation

Immediately after injury, platelet degranulation releases numerous cytokines, including platelet-derived growth factor (PDGF), transforming growth factor (TGF), and epidermal growth factor (EGF). These cytokines, together with other chemotactic agents, such as tissue debris and pathogenic materials, attract neutrophils and, later, macrophages. In time, these cells contribute to a larger number and variety of cytokines, which participate in the healing process.[3] (See *Patient Teaching: Normal and abnormal signs of the inflammatory process*.)

Cytokines have diverse effects on the healing process, interacting in additive, synergistic, or inhibitory ways (Table 5-2). For example, keratinocyte growth factor enhances the stimulation of collagenase synthesis exerted by insulin-like growth factor. TGF is inhibitory to fibroblast growth in the presence of EGF but stimulates cell division when PDGF is present.

Table 5-2 Major Growth Factor Families

Growth Factor Family	Cell Source	Actions
Transforming growth factor (TGF) β TGF-β1 TGF-β2 TGF-β3	Platelets Fibroblasts Macrophages	Chemotactic for fibroblasts Promotes extracellular matrix formation ↑ Collagen and tissue inhibitors of metalloproteinase (TIMP) synthesis ↓ Matrix metalloproteinase (MMP) synthesis Reduces scarring ↓ Collagen ↓ Fibronectin
Platelet-derived growth factor (PDGF) PDGF-AA; PDGF-BB; VEGF	Platelets Macrophages Keratinocytes Fibroblasts	Activates immune cells and fibroblasts Promotes extracellular matrix (ECM) formation ↑ Angiogenesis ↑ Angiogenesis
Fibroblast growth factor (FGF) Acidic FGF, basic FGF, KGF	Macrophages Endothelial cells Fibroblasts	↑ Angiogenesis ↑ Keratinocyte proliferation and migration ↑ ECM deposition
Insulin-like growth factor (IGF) IGF-I, IGF-II, insulin	Liver Skeletal muscle Fibroblasts Macrophages Neutrophils	↑ Keratinocyte and fibroblast proliferation ↑ Angiogenesis ↑ Collagen synthesis ↑ ECM formation ↑ Cell metabolism
Epidermal growth factor (EFG) EGF, HB (heparin binding), TGF-α, Amphiregulin, Betacellulin	Keratinocytes Macrophages	↑ Keratinocyte proliferation and migration ↑ ECM formation
Connective tissue growth factor (CTGF)	Fibroblasts Endothelial cells Epithelial cells	↑ Collagen synthesis Mediates action of TGF-βs on collagen synthesis

Proliferation Phase

The proliferation phase usually begins 3 days after an injury and lasts for a few weeks. This phase is characterized by the formation of granulation tissue in the wound space. The new tissue consists of a matrix of fibrin, fibronectin, collagens, proteoglycans, glycosaminoglycans (GAGs), and other glycoproteins.[5] Fibroblasts move into the wound space and proliferate. Because the type III collagen in the wound has decreased tensile strength, the patient is at risk for such abnormalities as wound dehiscence or opening of wound edges in a previously closed wound that healed by primary intention. If organs are protruding from the now opened wound, it's called *evisceration,* which is a medical emergency that requires immediate surgery.

Patient Teaching

Normal and abnormal signs of the inflammatory process

Discuss the *normal signs* of the inflammatory process with your patient:

- Redness
- Heat
- Swelling
- Pain

Clarify that in the early stages of the healing process you would expect the wound to exhibit these signs. However, advise the patient to *seek urgent medical attention* if any of the signs listed below are present, as these may be *signs of infection:*

- Wound breakdown
- Bleeding
- Increased pain
- Pus or unusual drainage
- Spreading redness around the wound
- Flu-like symptoms

Practice Point

Keep in mind that the induration, heat, discomfort, redness, and swelling experienced during the inflammatory phase are part of the normal wound healing processes and aren't, at this stage, likely to be due to wound infection. Remember to share this information with your patients.

Practice Point

During the first 3 weeks after surgery, the patient is at high risk for wound dehiscence and evisceration. Advise the patient that he or she should follow any postsurgical advice very carefully as the repaired tissue will not regain its full strength. The wound is at

risk of breakdown if undue pressure is exerted on the area. For instance, patients who have undergone an abdominal procedure should support their abdomen with a soft pillow if they need to cough. They should also avoid any heavy lifting or exertion as designated by their physician.

Role of Fibroblasts

Fibroblasts play a key role during the proliferation phase, appearing in large numbers within 3 days of injury and reaching peak levels on the 7th day. During this period, they undergo intense proliferative and synthetic activity. Fibroblasts synthesize and deposit extracellular proteins during wound healing, producing growth factors and angiogenic factors that regulate cell proliferation and angiogenesis.[6]

Granulation tissue is composed of many mesenchymal and nonmesenchymal cells with distinct phenotypes, inflammatory cells, and new capillaries embedded in a loose ECM composed of collagens, fibronectin, and proteoglycans.

Role of ECM Proteins

ECM consists of proteins and polysaccharides and their complexes produced by cells in the wound space. The two main classes of matrix proteins are fibrous proteins (collagens and elastin) and adhesive proteins (laminin and fibronectin). In addition, the ECM contains polysaccharides called proteoglycans and GAGs.

Collagen is the most abundant protein in animal tissue and accounts for 70% to 80% of the dry weight of the dermis.[7] The collagen molecule consists of three identical polypeptide chains bound together in a triple helix. Made mainly by fibroblasts, at least 19 genetically distinct collagens have been identified. Collagen synthesis and degradation are finely balanced.[2]

Elastin is a protein that provides elasticity and resilience.[8] It is composed of fibrous coils that stretch and return to their former shape, much like metallic coils. Because of these properties, elastin helps maintain tissue shape. Elastin represents only 2% to 4% of the human skin's dry weight; it's also in the lungs and blood vessels. It's secreted into the extracellular space as a soluble precursor, tropoelastin, which binds with a microfibrillar protein to form an elastic fiber network.

Laminin and fibronectin are two fiber-forming molecules. Their function is to provide structural and metabolic support to other cells. Fibronectin is found in plasma and contains specific binding sites on its molecular wall for cells, collagens, fibrinogens, and proteoglycans. It plays a central role in tissue remodeling, acting as a mediator for physical interactions between cells and collagens involved in ECM deposition, thereby providing a preliminary matrix.

Proteoglycans consist of a central core protein combined with a number of GAG chains that may be one or several types. GAGs consist of long, unbranched chains of disaccharide units that can range in number.[2] A highly complex group of molecules, proteoglycans are characterized by their many diverse structural and organizational functions in tissue. Forming a highly hydrated gel-like "ground substance," they can contain up to 95% (w/w) carbohydrates. Originally, however, they were thought to contribute to tissue resilience due to their capacity to fill much of the extracellular space.

Angiogenesis

Angiogenesis is the formation of new vessels in the wound space and is an integral and essential part of wound healing.[9] The vascular endothelial cell (VEC) plays a key role in angiogenesis and arises from the damaged end of vessels and capillaries. New vessels originate as capillaries, which sprout from existing small vessels at the wound edge. The endothelial cells from these vessels detach from the vascular wall, degrade and penetrate (invade) the provisional matrix in the wound, and form a knob-like or cone-shaped vascular bud or sprout. These sprouts extend in length until they encounter another capillary, to which they connect to form vascular loops and networks, allowing blood to circulate. This pattern of vascular growth is similar in skin, muscle, and intestinal wounds.

Epithelialization

Epithelial healing, or epithelialization, which begins a few hours after injury, is another important feature of healing. Marginal basal cells, which are normally firmly attached to the underlying dermis, change their cell adhesion property and start to lose their firm adhesion, migrating in a leapfrog or train fashion across the provisional matrix. Horizontal movement is stopped when cells meet. This is known as contact inhibition.

Wound Contraction

The final feature of the proliferation phase is wound contraction, which normally starts 5 days after injury. Wound contraction appears to be a dynamic process in which cells organize their surrounding connective tissue matrix, acting to reduce the healing time by reducing the amount of ECM that needs to be produced. The contractile activity of fibroblasts and myofibroblasts provides the force for this contraction. These cells may use integrins and other adhesion mechanisms to bind to the collagen network and alter its motility, bringing the fibrils and, subsequently, the wound edges closer.[2] Such contraction may not be important in a sharply incised, small, and noninfected wound; however, it's critical for wounds with large tissue loss.[10]

The myofibroblast theory suggests that the contraction force occurs when the movement of microfilament (actin) bundles (also termed *stress fibers*) contracts the myofibroblast in a muscle-like fashion. Because the myofibroblast displays many cell:cell and cell:matrix (fibronexus) contacts, the cellular contraction pulls collagen fibrils toward the body of the myofibroblast and holds them until they're stabilized into position. This gathering of collagen fibers toward the myofibroblast cell "body" leads to the shrinkage of granulation tissue. The ECM of the wound is continuous with the undamaged wound margin, enabling the granulation tissue shrinkage to pull on the wound margin, leading to wound contraction. The myofibroblast theory further proposes that the coordinated contraction (cellular shortening) of many myofibroblasts, synchronized with the help of gap junctions, generates the force necessary for wound contraction.[6]

The traction theory proposes that fibroblasts bring about a closer approximation of matrix fibrils by exerting "traction forces" (analogous to the traction of wheels on tarmac) on ECM fibers to which they're attached. This theory proposes that fibroblasts neither shorten in length nor act in a coordinated multicellular manner (as proposed by the myofibroblast theory); rather, a composite force, made up of traction forces of many individual fibroblasts, is responsible for matrix contraction. Such traction forces act as shearing forces tangential to the cell surface generated during cell elongation and spreading. According to the traction theory, the composite effect of many fibroblasts gathering collagen fibrils within the wound is thought to bring about wound contraction.[11]

Patient Teaching

Teach your patient who has a wound left open to heal (secondary intention) that clinicians will be looking for indications that wound healing is progressing normally:

- Healthy pink tissue in the wound bed
- Signs of new tissue growth at the wound edges
- Decreasing wound size over time

Maturation Phase

The maturation phase normally starts 7 days after injury and may last for 1 year or more. The initial component in the deposited ECM is fibronectin, which forms a provisional fiber network. Other components include hyaluronic acid and proteoglycans. The network has two main roles: as a substratum for the migration and growth of cells and as a template for subsequent collagen deposition. Collagen deposition becomes the predominant constituent of the matrix and soon forms fibrillar bundles and provides stiffness and tensile strength to the wound.

Collagen deposition and remodeling contribute to the increased tensile strength of skin wounds. Within 3 weeks of injury, the tensile strength is restored to approximately 20% of normal, uninjured skin. As healing continues, the skin gradually reaches a maximum of 70% to 80% tensile strength. Different organs regain tensile strengths to differing degrees. The remodeling process involves the balance between the synthesis and degradation of collagen. A range of collagenases regulates the latter. This process is also characterized by a gradual reduction in cellularity and vascularity.[3] Differentiation of fibroblasts into myofibroblasts with resultant apoptosis (programmed cell death) are also features of tissue remodeling.[12]

Practice Point

A patient history should always include information about prior wounds. Healed wounds never achieve the same tensile strength as uninjured skin, thereby increasing the potential for reinjury.

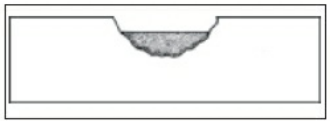
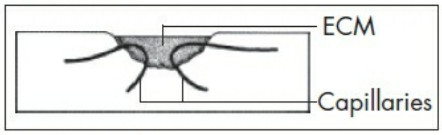
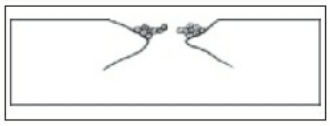
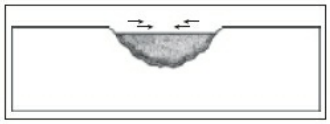
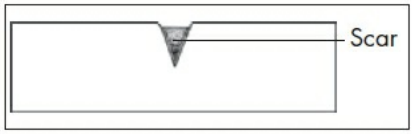
Patient Teaching

Remind the patient that the wounded area is never as strong as uninjured tissue so it is always vulnerable to damage. Simple measures such as keeping the scar tissue moisturized will help to optimize the condition of the tissue. Advise the patient to seek help if the scar begins to break down.

The scar is the final product of wound healing and is a relatively avascular and acellular mass of collagen that serves to restore tissue continuity and some degree of tensile strength and function. However, the strength of the scar remains less than that of normal tissue, even many years following injury, and it's never fully restored (Box 5-1).

Box 5-1 Summary of Wound Healing

The following is a summary of the events that occur during the phases of wound healing.

Hemostasis	
Platelets	Release cytokines (PDGF, TGF-β, EGF)
Inflammatory Phase	
Tissue debris and pathogens	Attract macrophages and neutrophils, which are responsible for: • phagocytosis • producing biological regulators, bioactive lipids, and proteolytic enzymes.
Proliferative Phase	
Fibroblasts	Responsible for: • synthesizing and depositing extracellular proteins • producing growth factors • producing angiogenic factors.
ECM and granulation tissue 	ECM composed of: • collagens and elastin • adhesive proteins • fibronectin and lamina • polysaccharides • proteoglycans • glycosaminoglycans.
Angiogenesis 	Capillary growth into ECM
Reepithelialization 	Migration of marginal basal cells across the provisional matrix
Wound contraction 	Contraction of fibroblasts and myofibroblasts to bring wound edges closer
Maturation 	• Collagen deposition and remodeling • Differentiation of fibroblasts into myofibroblasts with programmed cell death (apoptosis)
Resultant scar	Formation of a scar, an avascular, acellular mass of collagen

Practice Point

The width of the resultant scar of a wound healing by secondary

intention is about 10% of the original defect, primarily due to the process of wound contraction, working in conjunction with proliferation.

Patient Teaching

Inform the patient of the changes that occur to scar tissue over time. Initially, the scar may be red and raised but over time will become paler and flatten out. This process may take up to 2 years. Occasionally, there may be signs of abnormal healing, such as a scar remaining raised or swollen. If this occurs, the patient should seek further advice from a healthcare practitioner as this may indicate hypertrophic or keloid scarring.

Role of Matrix Metalloproteinases in Wound Healing

Proteases, especially the matrix metalloproteinases (MMPs), play essential roles in all phases of normal wound healing (Box 5-2). For example, during the inflammatory phase, damaged ECM proteins (such as collagen) must be removed so that newly synthesized collagen molecules can correctly align with collagen molecules in the wound matrix, permitting migration of epidermal cells and fibroblasts into the wound bed (Box 5-3). To remove damaged collagen molecules, collagenases (Box 5-4), make a single cut in collagen molecules, which permits the gelatinases to further degrade collagen molecules into small fragments that are then removed from the injury area by neutrophils and macrophages. MMPs also play a key role in angiogenesis by first degrading the basement membrane that surrounds VECs. This causes new capillary buds to sprout and “channels” to erode the ECM, through which the VECs migrate, eventually creating new capillary arcs. Furthermore, MMPs are required for myofibroblasts to contract ECM during the maturation or remodeling phase. The actions of MMPs are controlled by their natural inhibitors, the tissue inhibitors of metalloproteinases (TIMPs).

Box 5-2 Role of MMPs in Wound Healing

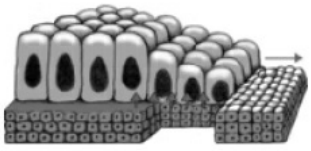
Proteases (especially MMPs) play important, beneficial roles in normal wound healing. They perform the following functions:

- Contract wound matrix through use of myofibroblasts
- Implement angiogenesis (breakdown of capillary basement membrane)
- Migrate cells (epidermal cells, fibroblasts, VECs)
- Remodel scar ECM
- Remove damaged ECM (especially during the inflammatory phase of healing)

Box 5-3 ECM Proteins and MMPs: Critical Factors for Epithelial Migration, Angiogenesis, and Contraction

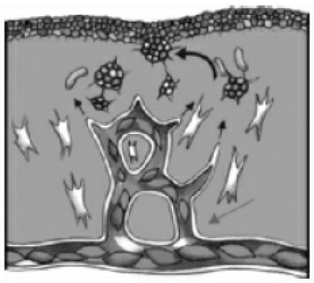
Epithelial migration

Epidermal cells at the leading edge of migrating sheets secrete several types of MMPs; fibroblasts migrating through provisional wound matrix also secrete MMPs.



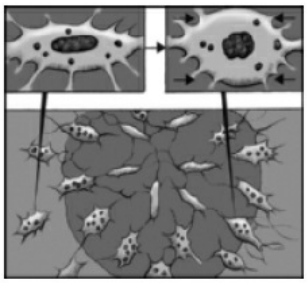
Angiogenesis

Endothelial cells secrete MMPs that degrade the basement membrane surrounding capillaries, allowing endothelial cells to proliferate and migrate toward angiogenic factors produced by cells in ischemic areas.



Contraction

Fibroblasts transform into myofibroblasts, which express contractile fibers and MMPs, and as myofibroblasts contract, force is applied to collagen fibers that reduces the size of the wound.



Box 5-4 Families of MMPs, TIMPs, ADAMs

Collagenases
- MMP-1, MMP-8, MMP-13, MMP-18
- Cut native type I collagen at one site

Gelatinases
- MMP-2, MMP-9
- Cut type collagen after collagenases make initial cut
- Cut native type IV collagen in basement membranes

Stromelysins
- MMP-3, MMP-10, MMP-11, MMP-19
- Cut core protein of proteoglycans

Metalloelastase/matrilysin
- MMP-7, MMP-12
- Cut multiple substrates, including type IV collagen

Membrane-type MMPs (MT-MMPs)
- MT-MMP1 (MMP-14), MT-MMP2 (MMP-15), MT-MMP3 (MMP-16), MT-MMP4 (MMP-17)
- Attached to plasma membrane, active pro-MMPs

Tissue inhibitors of metalloproteinases (TIMPs)
- TIMP-1, TIMP-2, TIMP-3, TIMP-4
- Specific inhibitors for MMPs

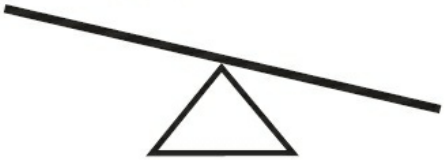
A disintegrin and metalloproteinase (ADAM)
- Aggrecanase-1 (ADAM-1)

Tumor necrosis factor–α (TNF-α) converting enzyme (TACE)

Box 5-5 Imbalanced Molecular Environments of Healing and Chronic Wounds

Healing Wounds
- Functional extracellular matrix (ECM)
- High mitogenic activity
- Low inflammatory cytokines
- Low protease, reactive oxygen species (ROS)
- Mitotically competent cells
- Low bioburden on the left hand side of the figure as another bullet point after "mitotically competent cells"

Chronic Wounds
- Damaged ECM
- High inflammatory cytokines
- High protease, ROS
- Low mitogenic activity
- Senescent cells



Acute Versus Chronic Wound Healing

Molecular and Cellular Abnormalities in Chronic Wounds

There would appear to be little consensus regarding the definition of acute

and chronic wound etiologies. Chronicity implies a prolonged or lengthy healing process, whereas acute implies uncomplicated, orderly or organized, or rapid healing. Bates-Jensen and Woolfolk[13] define an acute wound as "a disruption in the integrity of the skin and underlying tissues that progresses through the healing process in a timely and uncomplicated manner." Typically, surgical and traumatic wounds, which heal by primary intention, are classified as acute.

On the other hand, Sussman[14] defines a chronic wound as "one that deviates from expected sequence of repair in terms of time, appearance, and response to aggressive and appropriate treatment." The Wound Healing Society uses the definition of chronic wound as proposed in 1994 by Lazarus and colleagues: Chronic wounds are wounds that "fail to progress through a normal, orderly, and timely sequence of repair or wounds that pass through the repair process without restoring anatomic and functional results."[15] Such wounds usually heal by secondary intention and are associated with pathology, for example, diabetes, ischemic disease, pressure damage, and inflammatory diseases.

Patient Teaching

- Teaching patients the words used to describe the types of wound healing (acute, chronic, primary, secondary) may help them appreciate the time it may take for their wound to heal.
- Discuss the importance of moist wound healing to your patients, and inform them of what factors have been taken into consideration when deciding the most appropriate treatment for their wound.

The physiological differences between wounds that heal slowly and those that heal rapidly have been studied in a variety of ways (Box 5-5). One experiment explored the effect of chronic wound fluid on cell function.[16] Researchers cultured fibroblasts from human neonatal foreskin to use as a laboratory model of acute wounds. They then exposed the model to either chronic wound fluid or a control and found that chronic wound fluid dramatically inhibited the growth of the fibroblasts. According to

Phillips et al.[16] these results indicate that the microenvironment of chronic wounds impairs wound healing.

Other researchers[17,18] theorize that prolonged inflammation is the most significant factor in delayed healing. Indeed, Hart[17] proposes that the prolonged inflammatory phase is due to the presence of inflammatory leukocytes, typically neutrophils and their production of proinflammatory cytokines that perpetuate inflammation. He also argues that the release of tissue-damaging proteinases, which degrade newly formed tissue, delays or prevents normal wound healing processes. In addition to prolonged inflammation, Hart[17] suggests several other factors that may induce chronicity, including recurrent physical trauma, ischemic reperfusion injury, subclinical bacterial contamination, and foreign bodies.

Because chronic wounds are typically characterized by full-thickness tissue loss, reepithelialization is prolonged due to the loss of appendages.[14] Normally, epithelial cells require the smooth, moist surface of the basement membrane to move across the wound. In chronic wounds, epithelial cells latch onto and pull themselves across the scaffolding of macromolecules of the provisional matrix, such as laminin and fibronectin.

Wound Biofilms

Bacterial biofilms are known to contribute to numerous chronic inflammatory diseases, and recent evidence suggests that biofilms also play an important role in impairing healing in chronic skin wounds.[19–22] Wound bacteria that grow in clumps embedded in a thick, self-made, protective, slimy barrier of sugars and proteins are called a wound biofilm. Biofilms are defined as complex, dynamic microbial communities made up of microorganisms (bacteria and fungi) that synthesize and secrete a protective matrix that attaches the biofilm firmly to the wound surface.[23] They consist of a single bacterial or fungal species or, more commonly, may be polymicrobial, that is, they contain multiple diverse species that are continuously changing.[21]

Biofilms trigger a chronic inflammatory response that results in the accumulation of neutrophils and macrophages surrounding biofilms. The neutrophils and macrophages secrete high levels of ROS that affect the biofilm and the surrounding tissue. Inflammatory cells also secrete high levels of proteases (MMPs and elastase) that can help to break down the attachments between biofilms and the tissue, dislodging the biofilms from

the tissue.[24] However, the ROS and proteases also damage normal surrounding tissue, proteins, immune cells, and tissue cells, impairing healing.

Predisposing Factors for Development of Wound Biofilms

In vulnerable tissue, biofilms arise from planktonic bacteria attaching and forming a protective community before they are killed by the patient's immune system, by antibiotics, or by debridement. Thus, general conditions that impair the immune system or reduce the effectiveness of antibiotic drugs favor the development of biofilms in wounds. These conditions include ischemia or necrosis of tissue; poor patient nutrition; comorbidities that impair immune function, such as HIV, diabetes, major trauma, radiation treatment; or treatment with immune-suppressing drugs.

Assessment of Biofilms

In chronic wounds, it can be difficult to distinguish biofilms from slough. Wound slough has been described as a viscous, yellow, and relatively opaque layer on wound beds, while biofilm found in wounds can appear more gel-like and shiny.[25] There is an important link between biofilms and slough. Biofilms stimulate inflammation, which increases vascular permeability and the production of wound exudate and buildup of fibrin slough.[26] Therefore, slough may indicate that biofilm is present in the wound. Unfortunately, chronic skin wounds are frequently assessed with standard clinical microbiology laboratory assays that are designed to culture single, planktonic bacteria, and they do not adequately measure biofilm bacteria. Currently, the most reliable method to confirm the presence of microbial biofilm is specialized microscopy.[27–31] Recently, an analysis using special cultivation techniques of biopsies from chronic wounds found that 60% of the specimens contained biofilm structures in comparison with only 6% of biopsies from acute wounds.[22]

Management of Biofilms

Antibiotics and antiseptics kill single bacteria very easily, but the biofilm barrier blocks most antibiotics and antiseptics from reaching the bacteria, particularly in the center of the wound matrix. Wound biofilms are resistant

to antibodies, antibiotics, disinfectants, and phagocytic inflammatory cells. Wound biofilms can be effectively treated by a combination of debridement and/or cleansing to remove the biofilms, followed by application of dressings that block new bacteria from reaching the wound and killing bacteria left in the wound bed. These treatments can heal wounds, but patients must comply with the treatment plan because biofilms can re-form within a day and the wound will not heal.

An *ex vivo* porcine skin explant biofilm model that preserves key properties of biofilm attached to skin at different levels of maturity (0–3 days) was used to assess the efficacy of commercially available antimicrobial dressings and topical treatments.[32] Five types of antimicrobial agents (iodine, silver, polyhexamethylene biguanide, honey, and ethanol) and four types of moisture dressings (cotton gauze, sodium carboxymethylcellulose fiber, calcium alginate fiber, and cadexomer beads) were assessed. Time-release silver gel and cadexomer iodine dressings were the most effective in killing mature biofilm, reducing the level of biofilm-protected bacteria between 10^5 and 10^7 colony-forming units (CFUs) for a total of 10^7 CFUs of biofilm bacterial. In contrast, all other dressing formulations reduced biofilm between $10^{0.3}$ and 10^2 log CFUs in 24 or 72 hours with a single exposure. Similar results were found after *24-hour exposure to silver-release dressings using an in vivo pig burn wound model, demonstrating correlation between the ex vivo* and *in vivo* models. These results indicate that commonly used microbicidal wound dressings vary widely in their ability to kill mature biofilm, and the efficacy is influenced by time of exposure, number of applications, moisture level, and the formulation of the microbicide, which sustained release formulations providing improved killing.[32]

Wound Bed Preparation

The **TIME** acronym (**T**issue, **I**nfection/ **I**nflammation, **M**oisture balance, and **E**dge of wound) was first developed more than 10 years ago, by an international group of wound healing experts, to provide a framework for a structured approach to wound bed preparation; a basis for optimizing the management of open chronic wounds healing by secondary intention.[33] The framework was therefore termed "wound bed preparation" and was subsequently published in 2003 by Schultz and colleagues.[34] Since then, the TIME acronym has been widely used as a practical guide for the assessment

and management of chronic wounds. The clinical observations and interventions relating to wound bed preparation are grouped into four areas, all of which need to be addressed at each wound assessment:

- Tissue: assessment and debridement of nonviable or foreign material (including host necrotic tissue, adherent dressing material, multiple organism–related biofilm or slough, exudate, and debris) on the surface of the wound
- Infection/Inflammation: assessment of the etiology of each wound, need for topical antiseptic and/or systemic antibiotic use to control infection, and management of inappropriate inflammation unrelated to infection
- Moisture imbalance: assessment of the etiology and management of wound exudate
- Edge of wound: assessment of nonadvancing or undermined wound edges (and state of the surrounding skin)

However, it should be recognized that the TIME principles are only a part of the systematic and holistic evaluation of each patient at every wound assessment. An article by Leaper et al.[33] examines how new data and evidence generated in the intervening decade impact on the original concepts of TIME, and how they are translated into current best practice (Table 5-3).

Table 5-3 Summary Table of New Developments within the TIME Concept[33]

Clinical Observations	WBP	Developments	Factors to Consider
Tissue	Debridement	**New methods** • Low-frequency noncontact ultrasound (MIST, Sonoca) • Hydrosurgery • Debrisoft wipes **Advances in use of existing methods** • Larvae • Autolytic (honey, hydrogels) • Use of enzymes (collagenase) • Sharp/surgical (new guidelines) • Chemical (antiseptics, i.e., silver, PHMB) **NPWT**—as add-on with existing debridement methods	Use of maintenance debridement Considerations around safe practice • Knowledge • Skills • Competence • Evidence of efficacy
	Wound cleansing	Microbicidal irrigation solutions (Prontosan)	
Infection/ Inflammation	Bacterial balance	**Biofilm** • Improved understanding of biofilms and their role in nonhealing wounds • Management—combination strategy to disrupt biofilm and prevent reconstitution (debridement, antiseptic agents) • Detection of biofilm Use of PCR/pyrosequencing techniques to identify bacteria/fungi in wounds	Increased bacterial tolerance to topical/ systemic agents Mixed flora living synergistically Quiescent state of some bacteria in biofilms reduces effectiveness of antibiotics Diagnostic for biofilm detection needed
	Persistent inflammation	Improved understanding of the role of persistent inflammation in chronic/ stalled wounds • Role of MMPs and other proteases (diagnostics and inhibitors) • Role of biofilms in promoting wound inflammation	
	Managing infection/ inflammation	• Increased use of antiseptic agents • Role of nanocrystalline silver as an anti-inflammatory • Combination of surfactants with antimicrobials—biofilm disruption • NPWT combined with instillation of microbicidal solutions to reduce levels of planktonic and biofilm bacteria • Alternative use of new or existing agents—e.g., using nanocrystalline silver to dampen down inflammation • Improved healing of wounds treated with custom formulations of topical antibiotics/antiseptics based on bacterial profiles	Diagnostic tests—when and how often? Point-of-care detection Review of appropriate antimicrobials Rotation of products Microbial resistance (particularly to antibiotics)

Moisture	Moisture balance	Improved awareness of need to maintain appropriate moisture levels	Dressing selection—what do we need to consider? • Absorption • Retention • Patient comfort • Bacterial pool Skin sensitivity or allergy
	Exudate	Improved understanding of exudate composition—differences between acute and chronic wound fluid • Damaging proteolytic activity of chronic wound fluid Relationship of exudate with bacterial burden and biofilm formation Selection of appropriate dressings or devices for exudate management (i.e., new super absorbers) Greater emphasis on moisture management **NPWT**—for removal and containment of large exudate volumes	
Edge of wound		Epithelial edge advancement Improved state of surrounding skin **Evaluation**—check whether wound is closing Use of NPWT to encourage contraction Adjunct therapies (EMT, laser, ultrasound, systemic oxygen therapy)	Revisiting existing therapies Alternative use of products, e.g., using NPWT to splint wounds Role of diagnostics/ theranostics

A summary of their findings for each element of the TIME principles is as follows[33]:

1. Tissue: A major advance has been the recognition of the value of repetitive and maintenance debridement and wound cleansing, both in time-honored and novel methods (notably using negative pressure wound therapy [NPWT] and hydrosurgery).
2. Infection/Inflammation: Clinical recognition of infection (and noninfective causes of persisting inflammation) is critical. The concept of a bacterial continuum through contamination, colonization, and infection is now widely accepted, together with the understanding of biofilm presence. There has been a return to topical antiseptics to control bioburden in wounds, emphasized by the awareness of increasing antibiotic resistance. Recognition of the importance of biofilms (and the need for a simple diagnostic), use of NPWT, evolution of topical antiseptic therapy as dressings and for wound lavage (notably, silver and polyhexamethylene biguanide), and expanded insight of the role of molecular biological processes in chronic wounds (with emerging diagnostics and theranostics).
3. Moisture: The relevance of excessive or insufficient wound exudate and its molecular components has led to the development and use of a wide range of dressings to regulate moisture balance, to protect

periwound skin, and to optimize healing.

4. Edge of wound: Several treatment modalities are being investigated and introduced to improve epithelial advancement, which can be regarded as the clearest sign of wound healing.

Summary

The molecular and cellular environment of chronic wounds differs substantially from that of acute healing wounds. Specifically, nonhealing wounds have chronically elevated proinflammatory cytokines, which lead to chronically elevated levels of proteases (MMPs and neutrophil elastase) and ROS that degrade the components that are essential to healing, such as ECM components, growth factors, and receptors. Cells in the base of nonhealing wounds often become insensitive to growth factors resulting in senescent cells. Clinical studies using topical application of protease or dressings that bind proteases or use vacuum-assisted closure dressings have shown that reversing these molecular and cellular abnormalities promotes healing of chronic wounds.

PATIENT SCENARIO

Clinical Data

Mr. J is 44 years old. He underwent an open cholecystectomy for removal of gallstones. The patient is generally fit and well and has no underlying health problems. The wound was initially closed by primary intention and appeared to be healing; however, 2 months later, the wound began to show signs of breakdown (dehiscence) (Fig. 5-2A, 2 months postoperatively). After a further 2 months, the wound has now dehisced further (Fig. 5-2B).

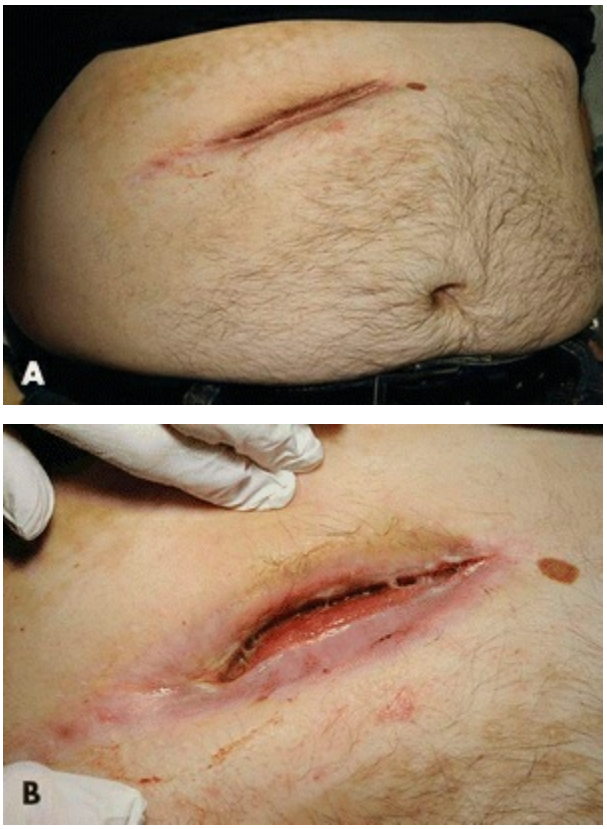


Figure 5.2. **(A)** Wound 2 months after surgery. **(B)** Wound 4 months after surgery.

Case Discussion

The wound initially would be described as an acute wound as it is a surgical wound; however, it is now 10 months since the original surgical procedure, and the wound is showing no signs of improvement. The wound would now be referred to as a chronic wound as it has not progressed through the normal pattern of events that would be expected. In acute wound healing, the inflammatory cells such as neutrophils and macrophages are attracted to the wound site to try and destroy bacteria and remove debris. In combination with cytokines, these cells would normally assist with the wound healing process. Furthermore, the wound should begin to decrease in size through the action of both fibroblasts and myofibroblasts, a process known as wound contraction, which helps to reduce the amount of new tissue that is laid down in the wound. However, there are no indicators that the wound is improving, and in fact, it has deteriorated and increased in size at each follow-up visit. As healing progresses, new tissue should be laid down (known as granulation tissue); this process

relies on an adequate blood supply. In this patient's case, the wound bed and surrounding skin appear to be well perfused, which would indicate that the blood supply to the wound is adequate. At the 2-month visit, a wound sample was obtained despite there being no obvious signs of infection as the clinicians wished to rule out infection as a potential cause of nonhealing. The result indicated the presence of normal skin commensals with no resistant bacteria. In the treating clinician's opinion, it was felt that a biofilm may be present that could be the cause of nonhealing, which was taken into account when choosing the most appropriate treatment. To assist in the healing process, the wound requires a moist environment to ensure the ideal medium for cells to be active; therefore, an appropriate moisture balancing dressing that also had antimicrobial properties was chosen as a strategy to treat any local infection that may be present. It was not possible to undertake any local debridement of the wound bed, which may have helped to remove the biofilm. The surrounding skin was protected with a skin barrier to prevent excoriation from wound exudate. At the 4-month visit, there were still no signs of healing, and the wound had increased in size and depth. In addition, the amount of exudate from the wound had increased, and there were signs of maceration at the wound edges. The patient was sent for an ultrasound exam and referred back to the surgeons as a deep collection was present on the ultrasound. In addition, the team of clinicians considered the potential of *factitious wounding* or self-imposed injury as this is an area of the body that is easily accessible and the clinical course of this case was unusual, in which case the patient would require support from a clinical psychologist as part of the multidisciplinary team.

Show What You Know

1. Immediately following tissue injury, the priority is to:

A. modify the immature scar tissue.
B. achieve rapid hemostasis.
C. rapidly fill the wounded area with granulation tissue.
D. destroy bacteria.

2. The main mechanism by which chronic wounds fail to heal is believed to be:

A. too rapid progress from hemostasis to maturation.
B. a failure of fibroblasts and myofibroblasts to facilitate wound contraction.
C. a dysfunction of collagen remodeling.
D. a prolonged inflammatory phase.

3. During the proliferative phase, the framework that new tissue grows into is commonly called:

A. the extracellular matrix.
B. the complement system.
C. chemotaxis.
D. apoptosis.

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Wound Assessment

6

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Objectives

After completing this chapter, you'll be able to:

- state the reasons for performing a wound assessment
- differentiate between partial- and full-thickness injury
- list the parameters of a complete wound assessment
- describe useful photographic techniques for wound documentation
- discuss wound documentation using an electronic medical record and electronic health record.

The Wound

Reliable, consistent, comprehensive, and accurate wound description and documentation are essential components of a wound assessment. Not only does it provide objective data to confirm wound progress, but it can also serve to alert clinicians about wound deterioration.[1] Wound description and documentation also enhances communication among healthcare providers, patients, and care settings.[1,2] Assessment of wounds is important because several clinical characteristics, such as new or increasing pain, new or increasing cellulitis, new or increasing purulent or nonpurulent drainage, and significant undermining, have been reported to constitute a wound emergency.[2] Appropriate assessment and monitoring of wound healing is founded on scientific principles.[3]

The management of acute and chronic wounds has progressed into a highly focused area of practice, with physicians, nurses, therapists, and

other professionals expanding their practice in this challenging arena. Care plans, treatment interventions, case management, and discharge planning, as well as ongoing patient and wound management, are all based on the initial and subsequent wound assessments. The total patient assessment, inclusive of any comorbid conditions and lifestyle, must also be a part of any comprehensive wound assessment. This chapter addresses the key assessment parameters of a patient with a wound admitted to any healthcare setting, including the importance of a history and physical examination, how to assess a wound, essential practice points, and examples of accurate and thorough documentation tools.

A wound is a disruption of normal anatomic structure and function.[4] Wounds are classified as either acute or chronic. Acute wounds can result from trauma or surgery. According to Larazus and colleagues,[4] acute wounds proceed through an orderly and timely healing process with the eventual return of anatomic and functional integrity. Chronic wounds, on the other hand, fail to proceed through this process and lose the cascade effect of wound healing and sustained anatomic and functional integrity. Stated simply, wounds may be classified as those that repair themselves or can be repaired in an orderly and timely process (acute wounds) and those that don't (chronic wounds).[4] See Chapter 5 for a more detailed description of wound healing. In the United States, the current Center for Medicare & Medicaid Services (CMS) definition of a chronic wound includes a time frame of greater than 30 days' duration for complete healing.[5]

The etiologies or causes of the wound must be determined before appropriate interventions can be implemented. This is especially important as many wounds have mixed etiologies. Ensuring a differential diagnosis right is not always easy, but learning the typical characteristics of a wound type can be helpful. Wounds may have a surgical, traumatic, neuropathic, vascular, or pressure-related etiology. For example, an acute wound caused by a bite (animal, insect, spider, or human) requires a different care plan than a wound caused by a burn. A patient who has an animal bite may require additional testing to rule out damage to nerves, tendons, ligaments, or bone, as well as determination of rabies or rabies vaccination status of the animal and the need for tetanus immunization.[6] The pathologic etiology will provide the basis for additional testing and evaluation to start the wound assessment process and basis for correct classification (see *Practice Point: The nine C's of wound assessment*).

Practice PointThe Nine C's of Wound Assessment

Wound assessment is needed for the following nine reasons:

- Cause(s) of the wound
- Clear picture of what the wound looks like
- Comprehensive picture of the patient
- Contributing factors
- Components of the wound care plan
- Communication to other healthcare providers
- Continuity of care
- Centralized location for wound care information
- Complications from the wound

Initial Patient Assessment

Obtain a thorough history and a complete physical examination on every patient admitted into your care. Obtaining a patient history provides information on relevant disease processes, comorbidities, medications the patient is taking, and family history of conditions that can impact the etiology and potentially the treatment and healing of the wound. In addition, the patient history may reveal information that explains previous wound healing concerns, infection, nutritional status, and other core information needed to develop the plan of care. A detailed patient medical and social history should direct additional questioning on any abnormal lab findings as well as a history of diabetes, vascular conditions, or an immune-compromised state. Assess pain and the patient/family knowledge regarding wounds. Therapies received as part of a prior health condition, such as radiation at the site of a wound, as well as history of a pressure ulcer increases risk to that area, are also important factors that can contribute to impaired healing and delay appropriate management strategies[1] (see "Radiation wounds" in Chapter 23, Palliative Wound Care). Use the assessment data to determine whether the wound is healable, maintenance, or nonhealable.[7]

Family support and patient and family functional abilities should be

evaluated as well. Involving other services and/or departments (e.g., social work, case management, pastoral care) early in the care planning process is crucial to developing a comprehensive plan of care for the wound patient. Case managers can be invaluable in determining the continuity of care across healthcare settings by asking the following questions during the initial assessment:

- What are the patient's values and goals of care?
- Can the patient care for himself or herself?
- Is there a caregiver available to assist with care after discharge?
- Can the patient change his or her own dressings?
- Who will put on and help remove compression stockings?
- Can the patient afford to purchase the necessary wound products/items?
- Does the patient/family know how to care for the stockings or other equipment?

Asking these questions is vital to conducting a comprehensive assessment of a patient with a wound.

Physical Examination

A head-to-toe physical examination should be performed. Evaluation of the skin, including any skin folds, pressure points, healed pressure ulcer sites, old scars or lesions, indications of previous surgeries, and the presence of vascular, neuropathic, or pressure ulcers, should be noted. The appearance of the skin, nails, and hair on the extremities should be assessed. Appraisal of skin color, temperature, capillary refill, pulses, and edema are also important elements of a thorough physical examination (see Chapter 4 Skin: An Essential Organ).

Different types of wounds require different considerations. Dehisced surgical wounds may have opened due to an infection or may heal poorly due to underlying disease processes, current medications (such as steroids), or malnutrition. Hemosiderin staining (reddish-brown color), caused by the chronic leakage of red blood cells into the soft tissue of the lower leg, is a classic sign of venous insufficiency and often seen in a person with a venous ulcer. If not managed with compression, this leakage often leads to venous ulcers. Arterial ulcers often present with the classic signs of hair loss, weak or absent pulse, and very thin, shiny, taut skin. Neuropathic ulcers require intense evaluation to determine the extent of the neuropathy. Patients with

diabetes are prone to callus formations and pressure points even when off-loading interventions are in place. Both are easily noted on examination (Fig. 6-1).

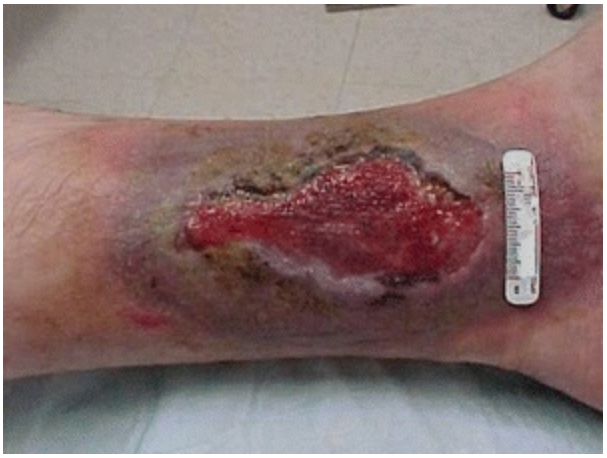


Figure 6-1. Hemosiderin deposit.

In persons with darkly pigmented skin, early detection of ulceration that relies only on visual inspection by the clinician to note erythema and color changes (such as a stage/category I pressure ulcer) remains a clinical challenge. The lack of a tool to help clinicians detect erythema in darkly pigmented skin hampers early detection of tissue injury. In a study in which 28 of 56 subjects had darkly pigmented skin, the authors showed that use of multispectral images of the ulcers resulted in algorithms that enhanced detection of erythema in darkly pigmented skin.[8] Assess for differences in skin temperature at the opposite site, skin tenderness, changes in tissue consistency, and pain.[3]

A comprehensive patient examination will reveal areas of concern for wound development and can pinpoint wound origins as well as why healing is not progressing in some wounds. Based on the comprehensive assessment and the determination as to whether a wound is healable, maintenance, or nonhealable, appropriate goals and treatment plan can be developed[7,9] (see Table 6-1). Developing realistic goals and care plans, performing regular follow-up examinations, and ensuring patient adherence to the plan of care are all key markers for successful outcomes (see chapters on specific wound types for more details).

Table 6-1 Healability of a Wound

"Evaluate the Person's Ability to Heal"	Definition
Healable	"The wound has several attributes including an adequate blood supply; the cause of the wound must be corrected; existing cofactors, conditions, or medications that could potentially delay healing must be optimized or ideally corrected."
Maintenance	"is a wound is a wound that may be healable but that either the healthcare system factors or patient-related issues are preventing the wound from healing."
Nonhealable	"is a wound that does not have adequate blood supply to support healing or the cause cannot be corrected."

Source: Sibbald, R.G., Goodman, L., Woo, K.Y., et al. "Special Considerations in Wound Bed Preparation 2011: An Update," *Advances in Skin and Wound Care* 24(9): 415-36, quiz 436-38, 2011.

Wound Assessment and Classification

Wound assessment—a written record and picture of the current status and progress of a wound—is a cumulative process of observation, data collection, and evaluation. As such, it's an important component of patient care. A wound assessment includes a record of your initial assessment, ongoing changes in the wound bed and periwound area, and treatment interventions. The initial assessment serves as the baseline for future comparisons, with ongoing assessments occurring at least weekly and when significant changes occur throughout the healing process.[3]

Practice Point

Because a wound can change rapidly, it is important to assess wounds for changes that could signal the need to modify

treatment.[3]

Although the frequency of wound assessment is often determined by individual agency or institutional guidelines, treatment modalities, regulatory guidelines, and wound characteristics also play a role in determining assessment frequency.[10] According to the most recent international guidelines, pressure ulcers should be evaluated at a minimum on admission, weekly, and with any signs of deterioration.[3] Frequency of assessment is also determined by wound severity, the patient's overall condition, the patient's environment, and the goals and plan of care.[10] Acute care patients often receive wound assessments daily or with each dressing change. In long-term care facilities, wounds must be assessed on admission, with each dressing change, and at least weekly.[11] Home care assessments are usually based on the frequency of the home visits but often occur weekly and/or with each licensed nurse visit. Regardless of the setting, however, the frequency of assessments should be determined by the wound characteristics observed at the previous dressing change, the significance of wound changes from one assessment to the next, as well as on the physician's or other practitioner's orders. Patient interventions should be implemented based on the baseline and subsequent wound assessment data (see *Practice Point: When to reassess a wound*).

Practice PointWhen to Reassess a Wound

Assessment provides indicators of successful treatment interventions and attainment of achievable outcomes and guides decisions about product changes. Reassess the patient's wound:

- before and after any surgical or specialized procedures
- weekly for a pressure ulcer[3]
- if the wound noticeably deteriorates
- if the wound becomes odorous, has new purulent exudate, or becomes more painful
- upon observing any other significant change in the condition of the wound, including at time of transfer or discharge

- after the patient has returned from another facility.

Although wound assessment needs to be in compliance with the regulatory requirements specific to the care setting, no written standard exists outlining the type and amount of information to include in a wound assessment. Likewise, no single documentation chart, tool, or electronic medical record (EMR) has been designated as the most effective. Banfield and Shuttleworth found that wound assessments were documented significantly more frequently when an assessment chart or form is used and that using a chart or form improves the nurses' assessment skills.[12] The best assessment form is one that is used consistently by the facility's staff. Forms that can be completed easily and quickly are more likely to be used on a regular basis. If the staff finds a form too long or difficult, that form is less likely to be used.

A complete, comprehensive initial assessment of the individual with a wound includes complete medical and social history, patient/significant other's goals of care, factors that may affect healing, a vascular assessment with extremity wounds, laboratory assessments as needed, nutritional status, pain, risk for developing additional wounds, psychological health and cognitive status, social and financial support systems, functional capacity (related to repositioning, posture, need for assistive equipment or personnel), pressure redistributing ability, support surface availability, knowledge of prevention and treatment, as well as ability to adhere to the treatment plan.[3] A minimal wound assessment should include a thorough assessment of the whole patient, identification of the cause of the wound, and wound characteristics such as type of wound, location, size, depth, exudate and tissue type(s) present, and periwound condition.

Wounds can be classified using several different approaches. The partial- versus full-thickness model is used primarily by physicians and clinicians for wounds other than pressure ulcers. Damage to the epidermis and part of the dermis constitutes a partial-thickness wound. Abrasions, skin tears, blisters, and skin-graft donor sites are common examples of partial-thickness wounds. Full-thickness wounds extend through the epidermis and dermis and may extend into the subcutaneous tissue, fascia, and muscle. Partial-thickness wounds heal by resurfacing or reepithelialization. Full-thickness wounds heal by secondary intention through the formation of granulation tissue, contraction, and, finally, reepithelialization, which of course requires a longer time period for healing.[5]

Pressure ulcers and neuropathic ulcers have their own staging and classification systems to indicate the depth of injury and healing methods. Use the specific system for the type of wound (see Chapter 13, Pressure Ulcers, and Chapter 16, Diabetic Foot Ulcers, and Chapter 4 section on skin tears, for more information).

Assessing the severity of a burn is a two-part process. Burn injuries are described by the extent of the body burned using one of several methods for estimating burn size, such as the rule of nines[13] or the Lund and Browder Chart[14] (Fig. 6-2). The depth of a burn injury is described by clinical observation of the anatomic layer of the skin involved (e.g., superficial, partial-thickness, full-thickness, or subdermal burns).

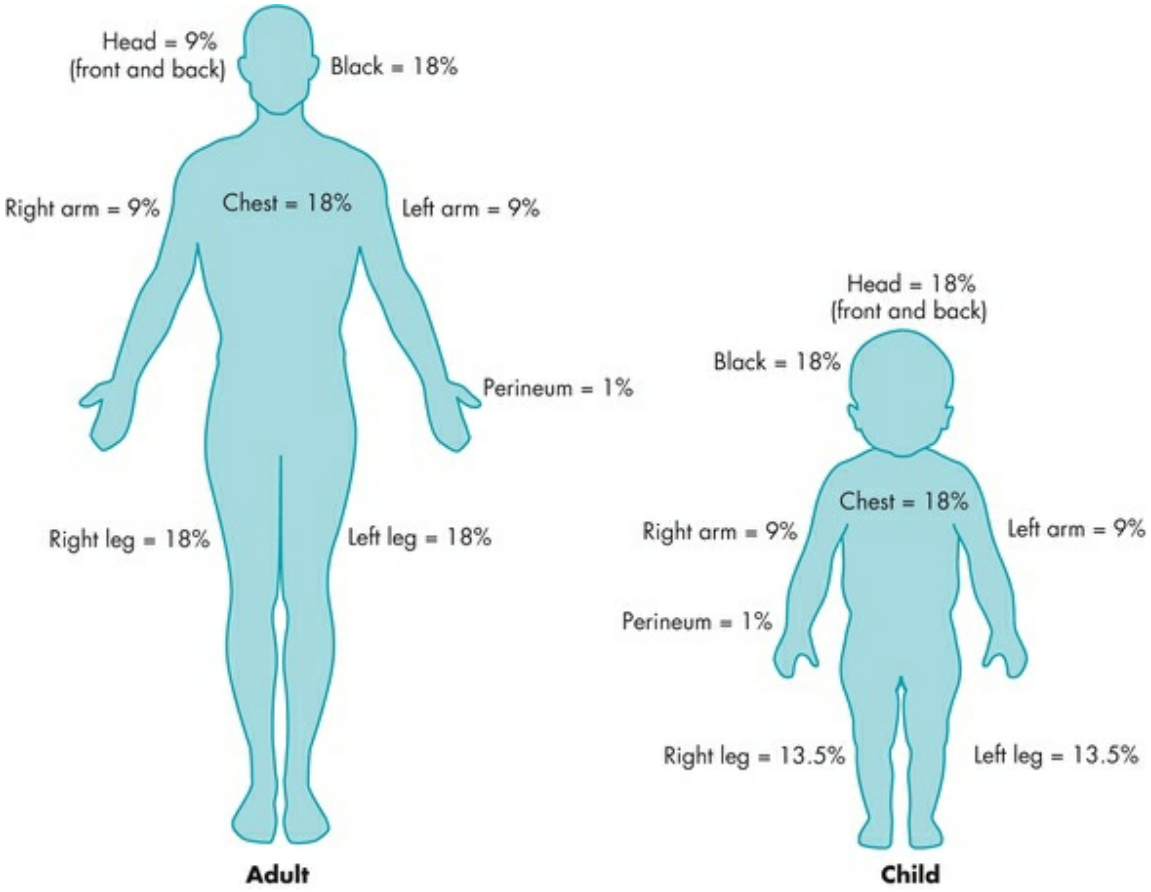


Figure 6-2. The rule of nines. The rule of nines estimates the amount of body surface that has been burned. In adults, the body is divided into sections of 9% or multiples of 9%. The percentages used in the rule of nines differ between adults and children.

Obviously, there are many parameters to consider when performing a comprehensive wound assessment. Each clinical agency needs to develop a protocol that all clinicians should learn and follow to ensure consistency of

assessment and documentation. Whether using stage/category, or partial- and full-thickness terminology, the one constant is clinical assessment. Assessment data give the healthcare provider a mechanism by which to communicate, improve continuity among disciplines, and establish and modify appropriate treatment modalities.

Elements of a Wound Assessment

In 1992, Ayello developed a mnemonic for pressure ulcer assessment and documentation[15] (Boxes 6-1 and 6-2). The mnemonic has been adapted for use with any type of wound to provide a thorough look at the parameters that complete and enhance an assessment. It provides a support structure for clinical decision making regarding ongoing assessment and reassessment and may be used in any practice setting according to the guidelines set up by your facility. This assessment chart may be used daily, weekly, or monthly. It's simple, fast, and can be further adapted to fit individual use.

Box 6-1 Pressure Ulcer ASSESSMENT Chart

PATIENT'S NAME: _____
DATE:_____ TIME:_____

A Anatomic location of wound
☐ Sacrum
☐ Elbow ☐ R ☐ L
☐ Trochanter ☐ R ☐ L ☐ Incisional
☐ Ischium ☐ R ☐ L ☐ Other
☐ Heel ☐ R ☐ L
☐ Lateral malleolus ☐ R ☐ L

Age of wound
_____days or _____months patient has had the pressure ulcer
_____ Date of oldest stage II pressure ulcer (MDS 3.0)

S size
_____cm length _____ cm width _____ cm depth

Shape
☐ Oval ☐ Round
☐ Other _____

Stage/Grade/Category
Pressure ulcer
☐ I ☐ II ☐ III ☐ IV
☐ sDTI
☐ Unstageable—Unable to determine stage; ulcer is necrotic
Wagner ulcer grade for neurotrophic ulcers:
☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5

S sinus tract, tunneling, undermining, fistulas
☐ Sinus tract, tunneling (narrow tracts under the skin) at ____ o'clock ____ cm
☐ Undermining (bigger area [than tunneling] of tissue destruction—area is more like a cave than a tract)

E exudate
Color
☐ Serous ☐ Serosanguineous ☐ Sanguineous
☐ Green ☐ Brown
Amount
☐ Scant ☐ Moderate ☐ Large
Consistency
☐ Clear ☐ Purulent

S sepsis
☐ Local ☐ Systemic ☐ None

S surrounding skin
☐ Dark ☐ Discolored ☐ Erythematous
☐ Intact ☐ Swollen
☐ Other _____

AGE: _____ ☐ M ☐ F
NUMBER OF PRESSURE ULCERS:_____

M margins
☐ Attached (edges are connected to the sides of the wound)
☐ Not attached (edges aren't connected to the sides of the wound)
☐ Rolled (edges appear rounded or rolled over)

Maceration
☐ Present ☐ Not present

E erythema
☐ Present ☐ Not present

Epithelialization
☐ Present ☐ Not present

Eschar (necrotic tissue)
☐ Yellow slough ☐ Black ☐ Soft
☐ Hard ☐ Stringy
Area around eschar is:
☐ Dry ☐ Moist ☐ Reddened

N necrotic tissue
☐ Present ☐ Not present

Nose
☐ Odor present ☐ Odor not present

Neovascularization (blood vessels are visible)
☐ Present ☐ Not present

T tissue bed
☐ Granulation tissue present
☐ Not present

Tenderness to touch
☐ No pain
☐ Pain present
☐ On touch
☐ Anytime
☐ Only when performing ulcer care
Patient getting pain medication:
☐ Yes ☐ No

Tension
☐ Tautness, hardness present
☐ Not present

Temperature
☐ Skin warm to touch
☐ Skin cool to touch
☐ Normal

Box 6-2 Wound ASSESSMENTS Chart

PATIENT'S NAME:_____
ASSESSMENT DATE:_____

Wound etiology:
☐ Surgical ☐ Arterial ☐ Venous
☐ Pressure ulcer ☐ Neurotrophic/DM ulcer
☐ Skin tear ☐ Trauma ☐ Other

A anatomic location of wound
☐ Upper/lower chest ☐ Abdomen
☐ Back ☐ Head ☐ Ear ☐ R ☐ L
☐ Sacrum ☐ Coccyx ☐ Ischium ☐ R ☐ L
☐ Trochanter ☐ R ☐ L
☐ Elbow ☐ R ☐ L ☐ Arm ☐ R ☐ L
☐ Leg ☐ R ☐ L ☐ Foot ☐ R ☐ L
☐ Heel ☐ R ☐ L
☐ Lateral malleolus ☐ R ☐ L
☐ Medial malleolus ☐ R ☐ L

Age of wound
☐ Acute—Date of onset: _____
☐ Chronic—Date of onset: _____

S size, shape, stage
_____ cm L_____cm W _____ cm Depth

Shape
☐ Oval ☐ Round ☐ Irregular ☐ Other _____

Stage
Stage of pressure ulcer
☐ I ☐ II ☐ III ☐ IV ☐ unstageable ☐ sDTI
Wagner ulcer classification
☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5

S sinus tract, tunneling, undermining, fistulas
☐ Sinus tract ☐ Tunneling
☐ Undermining ☐ Fistula ☐ None
Located _____ at _____o'clock, _____ cm depth

E exudate
Amount:
☐ None ☐ Scant ☐ Moderate ☐ Large
Color: ☐ Serous ☐ Serosanguineous ☐ Sanguineous
Consistency: ☐ Clear ☐ Purulent
Odor: ☐ Present

S sepsis
☐ Systemic ☐ Local ☐ Both ☐ None

S surrounding skin
☐ Intact ☐ Erythematous ☐ Edematous
☐ Induration ☐ Warm ☐ Cool
☐ Discolored ☐ Dry ☐ Other _____

M Maceration
☐ Not present
☐ Present: _____ cm, location_____

E edges, epithelialization
☐ Edge attached ☐ Edge not attached
☐ Edges rolled

AGE: _____
REASSESSMENT DUE DATE:_____

☐ Surgical incision approximated
☐ Surgical incision open
☐ Sutures/staples intact
☐ Epithelialization present: _____ cm
☐ Epithelialization not present

N necrotic tissue
☐ Not present ☐ Present
Type
☐ Yellow slough ___% ☐ Black ____%
☐ Soft ☐ Hard ☐ Stringy
Percentage of wound (check closest percentage):
☐ 100% of wound ☐ <75% ☐ >75%
☐ <50% ☐ >50% ☐ <25% ☐ >25%
☐ Other: _____ %

T tissue of wound bed
☐ Granulation not present
☐ Granulation present _____ amount%

Tenderness or pain
(0 being no pain, 10 being intense pain)

Pain scale score
0 1 2 3 4 5 6 7 8 9 10
Circle appropriate number

Pain present:
☐ on touch ☐ anytime
☐ only when performing wound care
☐ during dressing change
☐ other (specify) _____
Pain management: Specify method _____
☐ Not effective ☐ Effective

S status
Wound status: Initial assessment date _____
☐ Improved: date _____ ☐ Unchanged: date _____
☐ Healing: date _____ ☐ Deteriorating: * date _____
*Notify physician
☐ Supportive therapy
☐ compression ☐ off-loading
☐ pressure redistribution devices
☐ other _____
☐ Patient's perception on quality of life ____
☐ Case management/social services needs
☐ Nutrition consultation requested
☐ PT/OT
☐ Referral to other departments _____
Initial assessment:
Signature _____Title _____ Date _____
Reassessment:
Signature _____Title _____ Date _____

Location and Age of Wound

Wound location should be documented using the correct anatomical terms—for example, right greater trochanter rather than right hip. Include an anatomical figure or diagram of the human body, with the wound's location noted in your assessment record to provide complete admission

documentation. If there are two or more wounds near one another, they should be labeled and numbered for clarity (e.g., 1a, 1b, etc.). It is particularly important to note how long the patient has had the wound, especially since CMS now requires that the date of the oldest stage II pressure ulcer be recorded on Minimum Data Set 3.0 (M0300B.3).[16] Are you dealing with a new, acute wound or a wound that has failed to heal for several weeks or months? Time isn't the sole determinant of acute versus chronic wound status. Although 30 days is often used for designation as chronic status, the more important criterion is whether or not the wound is making progress toward healing.[5,16]

In addition to wound duration, documentation of the etiology of the wound, if known, is important. For example, if a patient reported that she spilled hot coffee on her amputated stump, causing a blister that evolved into a full-thickness wound due to trauma and insufficient arterial supply, it would be incorrect to classify the wound as a pressure ulcer. It is not unusual for an individual with diabetes mellitus to have a neuropathic/vascular ulcer or to have multiple ulcers of more than one origin.

Wound Size and Stage/Category

The joint National Pressure Ulcer Advisory Panel (NPUAP)–European Pressure Ulcer Advisory Panel (EPUAP) classification system[3] is only intended for use in staging/categorizing pressure ulcers. It was revised in 2009 to include four numerical stages with two additional categories for use in the United States that incorporate suspected deep tissue injury and unstageable ulcers into their own separate categories.[17] The staging/categorization system addresses the depth of tissue damage in numerical stages/categories I through IV. Any pressure ulcer covered with eschar or necrotic tissue is unstageable, including in long-term care where CMS now requires that it be documented on MDS 3.0 under the unstageable section M0300F in the United States.[16,18] Reverse staging is no longer required in the long-term care setting[16,18] and is never recommended[19] (see Chapter 13, Pressure Ulcers).

Partial-thickness wounds heal fairly quickly (days to weeks) as they involve the epidermis and extend into, but not through, the dermis. Full-thickness wounds penetrate through the fat and involve muscle, tendon, or bone and take longer to heal (Fig. 6-3). Use the correct classification/staging system for the specific wound type, for example,

Meggitt-Wagner for diabetic ulcers (see Chapter 16, Diabetic Foot Ulcers) or International Skin Tear Advisory Panel (ISTAP) for skin tears (see Chapter 4, Skin: An Essential Organ).

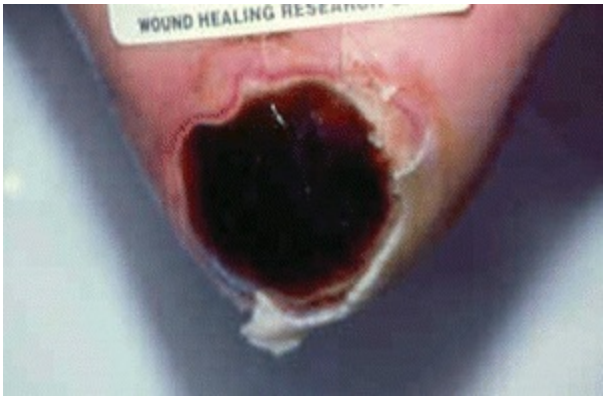


Figure 6-3. Necrotic, unstageable pressure ulcer. Shown here is a pressure ulcer that's unstageable because its base is covered with eschar. Be sure to measure the pressure ulcer's length, width, and percent and type of necrotic tissue. Document your findings.

Practice Point

Even though an ulcer is necrotic and unstageable, you still need to document the wound size for length, width, percent and type of tissue present, exudate, and odor.

Wound Measurement

Measurement of a wound is an important component of wound assessment and provides valuable information on wound progression or nonprogression as well as assessment of the effectiveness of clinical interventions. Wound measurement is particularly important in determining clinical effectiveness for research purposes. Consistency and accuracy in how the wound is measured are important for meaningful comparisons to determine changes in the wound over time and for comparing the effectiveness of various treatments. Consistency is best assured when the agency develops and disseminates a protocol for wound measurement that staff can follow. It is also important to use consistent patient positioning every time a wound is measured.[3]

Wound measurement methods can be simple or sophisticated, two-

dimensional (wound surface area) or three-dimensional (wound volume). A variety of systems are used to measure wounds and assess healing. These include wound tracing, width and length measurements, computerized wound-documenting systems (which can be one, two, or three dimensional), and digital photography.[1,20,21] A new handheld portable device that combines a digital camera with a scanner unit that plugs into a standard personal digital assistant has reportedly been useful in the community setting for assessment and documentation of venous and diabetic ulcers.[1] The WoundVision Scout is a portable, handheld device that can photograph the wound for computerized measurement and documentation.[22] Changes in wound measurements, such as a decrease in size, are used as an indicator of healing. Surgical incisions can be measured using length (e.g., "incision line is 8 cm long"). Wounds should not be measured using objects, such as a dime or half-dollar, but rather should be measured in centimeters or millimeters depending on the size of the wound.

Area

The simplest and most common method of wound measurement is the linear method using a paper or plastic ruler marked in centimeters and millimeters. The NPUAP Position on Wound Area Measurement, outlined in a 2008 study by Langemo and colleagues,[21] is to measure the greatest head-to-toe length and the greatest side-to-side width perpendicular (90-degree angle) to each other.[21,23] If this method is used consistently, then measurements over time should become more reliable and comparable (Fig. 6-4). Linear measurement is inexpensive, readily available, causes little to no discomfort, and is used frequently by most clinicians.[3,21] However, use caution with this method, as it assumes that the wound area is a rectangle or square, which is rarely ever the case, and nearly always overestimates the size of the wound.[24] Regardless of which method is used, what's most important is to have an agency protocol that the staff understands and that is being implemented consistently.

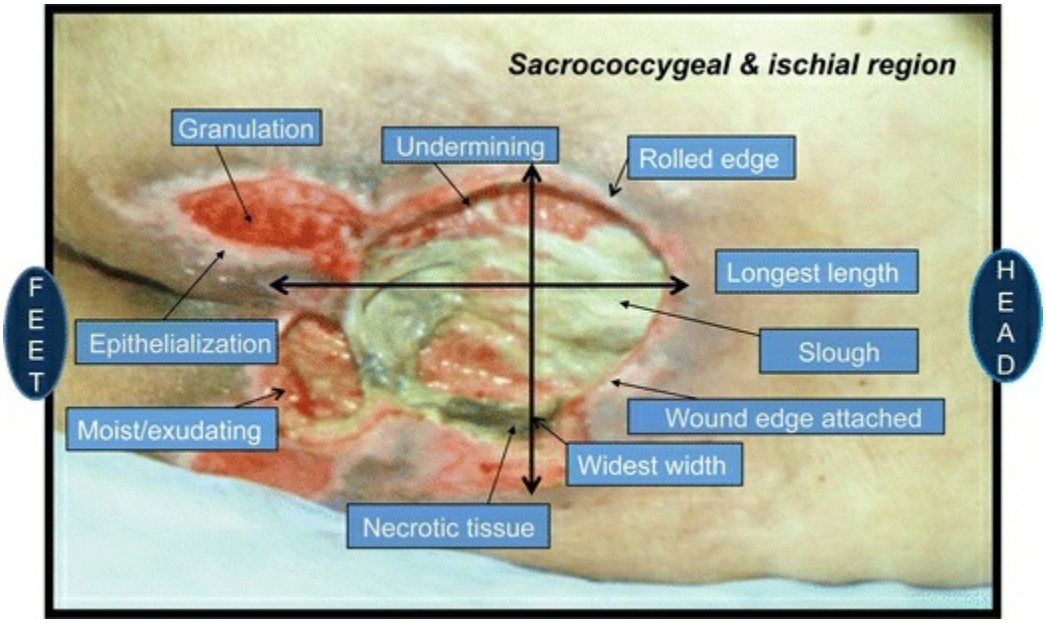
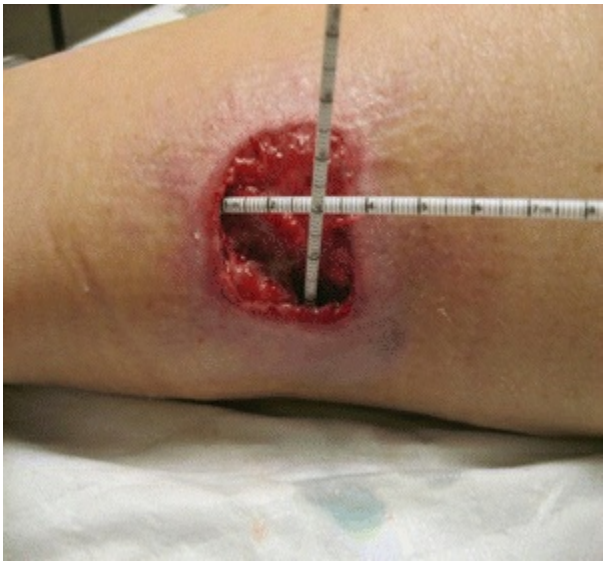


Figure 6-4. Wound terminology. Using current terminology is imperative for accurate assessment. This photograph labels the wound's characteristics as well as its length and width.

Another way to measure area is to multiply length by width in square centimeters (cm^2). This adds a third dimension of depth, which is then added to the linear measurement if desired.[24,25] If the wound is open, depth can be assessed by placing either a clean cotton-tipped applicator or a centimeter measuring device into the deepest part of the wound, marking it, and then measuring it upon removal (Fig. 6-5).



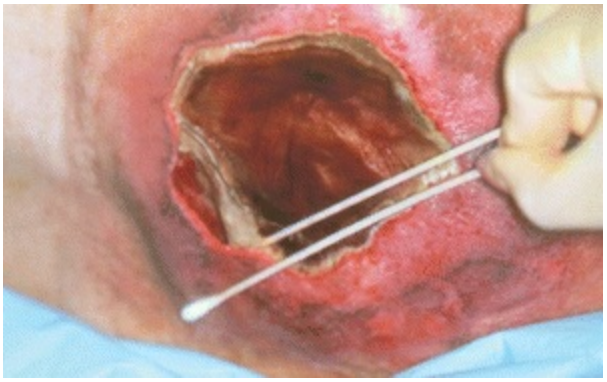


Figure 6-5. Determining wound depth. The depth of a wound can be measured by placing a sterile, premoistened (with normal saline or sterile water) cotton-tipped applicator into the wound and comparing the marked area against a centimeter measuring device.

Planimetry is a method where a wound tracing is made on metric graph paper with a 4-cm or 8-cm grid. The completed squares, within the traced wound edges, are then counted to yield an approximate area in square centimeters.[26] Minimal training is needed to use this method, the acetate tracing medium is inexpensive and disposable, and the wound area can be determined immediately.[24]

The wound area can also be measured noninvasively by stereophotogrammetry (SPG), using a digital camera and computer software. A target plate is placed within the plane of the wound to be photographed. The digital photo is then downloaded to the computer screen where the wound edges are traced, along with the length and width, using a computer-pointing device or mouse. The software automatically calculates the area as well as the length and width.[24] A color picture of the wound along with the measurements taken during each visit can be printed on a chart sheet for the patient record. This method allows for accurate, reproducible measurements of irregular wounds and is noninvasive.[24,27]

The WoundVision Scout, a handheld, portable device, can easily measure wound L and W as well as area thermal (infrared) and visual wound imaging. The Scout is a medical imaging clinical tool designed to photograph and measure area of a wound. It can be used to monitor change in wound size over time.[22]

Evidence-Based Practice

Using SPG to measure wounds is the most accurate and reliable method. Digital planimetry has fairly good reliability.[24,27]

Volume

As most wounds extend below the skin surface, they are three dimensional, generally irregular, and, at times, cone shaped. To that end, volume becomes an important variable and needs to be calculated. The most commonly used technique to assess wound volume is to measure the three dimensions of length, width, and depth and multiply those measurements by one another (L × W × D = volume cm^3).[25] Caution should be used, however, as this equation assumes that the base and surface area are the same size, which is generally not the case. The net effect is overestimation of wound volume.

Other techniques include molds, fluid instillations, the Kundin device, and SPG. Molds and fluid installations are imprecise and time consuming, are uncomfortable for the patient, and can potentially contaminate the wound.[26] The Kundin device is a plastic-coated, disposable, three-dimensional gauge with three arms for measuring length, width, and depth.[28] Wound volume is calculated via a mathematical formula that assumes the shape of the wound lies somewhere between a cylinder and a sphere.[28] Measuring volume using the Kundin device is a convenient, relatively inexpensive, user-friendly technique.[28] As mentioned previously, SPG measures the depth and area of a wound and inputs that information into software that calculates wound volume using the Kundin device formula. In one study, SPG was found to have the greatest reliability and least error of measurement.[25] When the Kundin device and SPG were compared using wound models, SPG was the more accurate method. However, more research is needed. The key is to select and implement a consistent method to measure depth.[3]

Sinus Tracts, Undermining, and Fistulas

Sinus tracts/tunnels, undermining, and fistula formation delay the healing cascade. Intervening early with the appropriate medical and/or surgical and/or nursing actions is paramount to healing these complicated wounds.

Sinus Tracts

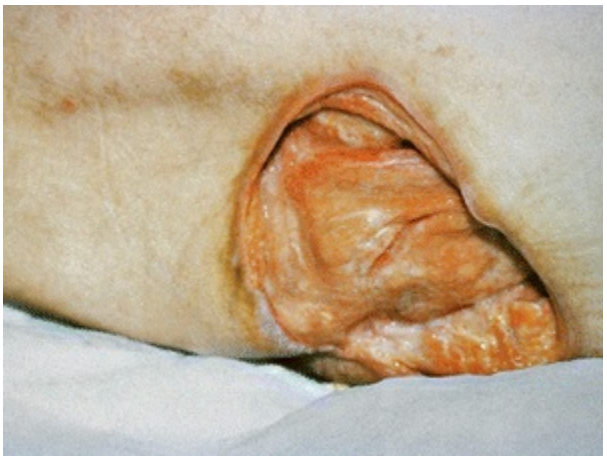
A sinus tract (or tunnel) is a course or path of tissue destruction, sometimes

called a "tunnel," occurring in any direction from the surface or edge of a wound. It results in dead space with a potential for abscess formation. A sinus can be distinguished from undermining in that it involves only a small portion of the wound edge; undermining involves a significant portion of the wound edge.[17] The sinus or tunnel has a potential for abscess formation, further complicating the healing process. Sinus tracts are common in dehisced surgical wounds and may also be present in neuropathic wounds, arterial wounds, and pressure ulcers. Documenting sinus tracts is an important element in assessment because it enables the clinician to evaluate potential treatment interventions and to identify reasons for nonhealing. Treatment interventions involve loosely packing the dead space with an appropriate dressing to stimulate granulation tissue production and the contraction process. Document what goes into the tract to see that it is removed during dressing changes. The goal is to close the sinus tract first, while allowing the outside of the wound to remain open and fully heal.

Measurement of a sinus tract can be made by inserting a sterile, premoistened cotton-tipped applicator, a centimeter measuring device, or a gloved finger into the bottom or end of the tract, marking it, and then measuring it upon removal. This must be done very carefully to avoid injury during measurement (Fig. 6-5).

Undermining

Undermining is tissue destruction that occurs around the wound perimeter underlying intact skin; in these wounds, the edges have pulled away from the wound's base (Fig. 6-6). Pressure ulcers that have been subjected to a shearing force often present, with undermining in the area of the greatest shear. Undermining is also seen when the opening of the wound is smaller than the affected tissue below the dermis and in desiccated wound beds.



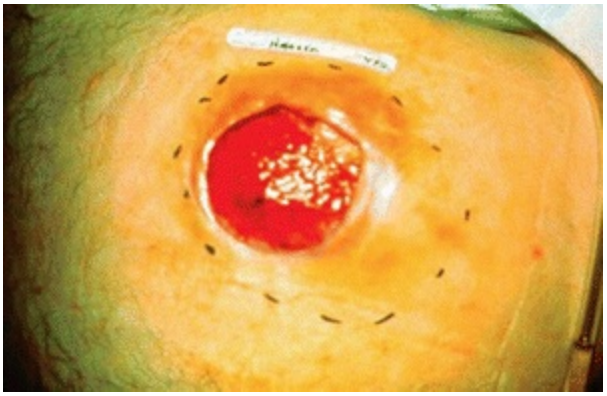


Figure 6-6. Undermining, as shown, is tissue destruction that occurs around the wound perimeter underlying intact skin.

Documentation of the location and amount of undermining is important. Clinicians can document using the clock figure, with the head as the 12 o'clock position (e.g., "undermining from 2 to 6 o'clock, measures 3 cm") or using percentages (e.g., "75% of the wound has undermining measuring 2 cm from 12 to 9 o'clock"). Undermining may also be more extensive in one part of a wound than another. This, too, should be documented appropriately. Interventions include loosely packing or tucking all undermined areas to prevent buildup of debris and dead tissue and applying an appropriate dressing, such as hydrogel, gauze, or alginate dressing.

The degree of undermining can be measured using the same method as for sinus tracts.

Fistulas

Fistulas can develop in surgical wounds and in deep, severe pressure ulcers. A fistula connects viscous organs together (e.g., a rectovaginal fistula) or connects a viscous organ to the skin (e.g., an enterocutaneous fistula).[26] Fistulas are named by using the point of origin, such as the rectum, and the point of exit, such as the vagina. Management of a patient with a fistula is complex and intense and demands critical thinking and technical skills.[26] Fistulas can take weeks or months to heal. The presence of feces in the area of a fistula is a significant concern. In addition, the patient with a fistula is often malnourished and may require weeks of intense nutritional therapy to improve his or her condition (see Chapter 19 for more on fistulas).

Exudate and Odor

Exudate is accumulated fluids in a wound; these fluids may contain serum,

cellular debris, bacteria, and leukocytes. Exudate may appear as dry, dehydrated, dead, or nonviable tissue (nondraining) or be moist and draining. Exudate assessment includes noting the amount (small, moderate, large), color, consistency, and odor.[29] Certain microorganisms, such as *Pseudomonas aeruginosa*, have a characteristic odor (Box 6-3 and Table 6-2).

Table 6-2 LOWE© Skin Barriers for Wound Margins: 20-Second Enablers for Practice

Exudate may indicate that the cause of the wound has not been treated (e.g., edema due to venous insufficiency), congestive heart failure is present (look for bilateral involvement and extension above the knee), low albumin (malnutrition, kidney or liver disease), or infection (check for symptoms or signs).

Periwound skin needs protection from exudate by using absorbent dressings over the wound and protecting the periwound skin. You can choose from four ways to protect the external skin of a wound. Try using this memory jogger to remember them: LOWE© (from Old English, meaning to approve of, prompt, or to humble oneself).

Type	Advantages	Disadvantages
Liquid film Forming acrylate • No sting • Skin preparation, etc.	• Transparent surface that resists removal • Low incidence of reactions	• Some skin sealants may evaporate and dry out • Lack of availability on some institutional formularies
Ointments • Petrolatum • Zinc oxide	• Relatively cheap and easy to apply	• Petrolatum liquefies with heat • Zinc oxide ointment does not allow visualization of underlying wound margin • Ointment vehicle may interfere with the action of ionized silver • Reactions to the adhesive can occur
Windowed dressing • Framing of wound margin with protective adhesive • Hydrocolloid • Film • Acrylate • Silicone, etc.	• Provides a good seal around the wound edge • Some products facilitate visibility of the wound margins	• If seal is compromised, moisture may accumulate under the dressing
External collection devices	• External pouching may help in locations where an external seal is difficult (e.g., perirectal area)	• Devices need to be monitored for external seal *Note: These devices do not replace a search for the cause of the excessive exudate and the need to correct the cause*

Ayello, E.A., Sibbald, R.G. "LOWE© Skin Barriers for Wound Margins: 20 Second Enablers for Practice," *Advances in Skin & Wound Care* 19(5):237, 2006.

Box 6-3 Classifying Exudates

Wound exudate can be classified in two ways—by type or

amount.

Type (color and consistency)

Exudate may exist as a single form or in combinations (e.g., serosanguineous):

- Serous or clear fluid
- Sanguineous for blood
- Purulent pus made up of inflammatory cells and tissue debris that can result from infection or an inflammatory process

Amount

The amount of exudate may indicate that the cause of the wound has not been treated (e.g., edema due to venous insufficiency), that congestive heart failure is present (look for bilateral involvement and extension above the knee), that low albumin levels have occurred (malnutrition, kidney or liver disease), or that infection is present (check for signs or symptoms). Amounts of exudate include:

- none
- small—there's just a detectable discharge when the dressing is removed, less than 33%
- moderate—exudate is covering less than 67% of the dressing surface
- large—exudate is covering more than 67% of the dressing surface.

Exudate may be serous (clear or pale yellow), serosanguineous (serous or blood tinged), sanguineous (bloody), or green brown. The consistency may be thick, milky, or purulent. It can be an important indicator of wound status.

Sepsis

Sepsis or bacteremia is caused by anaerobes and gram-negative bacteria and can occur in any susceptible wound. Assessment for the presence of sepsis should include consideration of erythema, warmth, edema, purulent or increased drainage, induration, increased tenderness or pain, and crepitus

or fluctuance.[3,30–32] Consider local infection in a pressure ulcer in the presence of nonhealing for 2 weeks, friable granulation tissue, malodor, increased pain or heat in the wound or periwound, increased drainage, an ominous change in the nature of wound drainage, an increase in necrotic tissue in the wound bed, and pocketing or bridging in the wound bed.[3] Also be suspicious of infection if the wound is located in an area likely to be contaminated (e.g., anus), in diabetes mellitus, protein–calorie malnutrition, hypoxia or poor tissue perfusion, immune-compromised individuals, or those with autoimmune diseases.[3] If sepsis is present, it's important to determine whether the infection is local or systemic. Interventions are based on accurate assessment and laboratory support.

The best method to culture a wound to determine the presence of sepsis remains controversial. Typically, tissue biopsy and quantitative culture is the gold standard.[3] These options may not be available in all settings, however, and many clinicians lack the skill necessary to perform them. The swab method, in which the wound must be cleaned and thoroughly dried prior to swabbing for a culture, continues to be used in many settings. After first cleansing the wound, culture viable tissue in the wound bed to identify the presence and type of microorganisms.[30] Culturing of wounds is important in determining whether infection is present. Malodorous wounds should also be documented. However, make sure the odor is from the wound —not the dressing change, which is a common mistake. The clinician must first cleanse the wound prior to assessing and documenting malodor. Certain organisms—such as *Pseudomonas*—have a distinct odor that is easily recognized by the trained clinician (see Chapter 7, Wound Bioburden and Infection, for more detailed information on infection and culturing).

Surrounding Skin/Periwound

The periwound is the skin surrounding a wound, and it also provides valuable information to the assessing clinician. Erythema and warmth may indicate inflammation, cellulitis, or infection. Interruptions in periwound skin integrity (denudation, erosion, papules, or pustules) may indicate allergic reactions to tape or dressing adhesive. Moisture-associated skin damage caused by wound drainage can lead to maceration of the periwound skin. Maceration or desiccation may be a sign that the dressing is too moist or too dry for the amount or type of exudate. Ensure that the dressing used can adequately absorb the amount of drainage present without having to be changed frequently. Palpation should be done with the fingertips around a

wound surface. This may reveal induration (hard to touch) or fluctuance (bubbly, fluid wave), which are abnormal fluid accumulations indicative of further tissue damage or abscess. In a study of lower extremity venous ulcers, more rapid closure of the wound bed occurred when treatments were also applied to the periwound area. Although new research provides us with a different understanding of where the wound ends[33] and the surrounding skin begins, assessment of surrounding tissue does provide useful information for the ongoing evaluation and future wound care interventions.

Practice Point

Cleanse the wound prior to assessing for malodor. Remember that not all odor indicates infection; certain dressings develop a distinct odor when exudates interact with them (e.g., alginates). Odor may also indicate the need to change the dressing more often.

Maceration

As stated above, moisture-associated skin damage from wound exudate may cause maceration of the periwound skin. Maceration is a softening of the skin surrounding a wound caused by excess drainage or pooling of fluid on intact skin and appears as a white, waterlogged area. It may be caused by inadequate management of exudate or an increase in exudate due to changes in the wound tissue. Maceration may be prevented by using an appropriate barrier product (cream, ointment) around the wound, changing the dressing more often, or selecting a more absorbent dressing (Fig. 6-7).

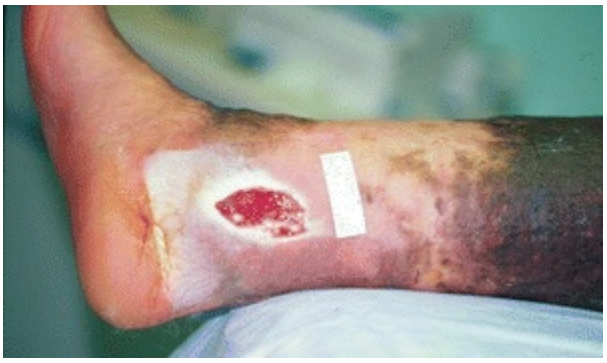


Figure 6-7. Maceration. This photograph shows maceration of the surrounding

skin caused by an overwhelmed dressing.

Edges and Epithelialization

Epithelialization is the regeneration of the epidermis across a wound surface.[26] The epithelial wound edge is continuous and often difficult to see. As wound migration proceeds from the edges toward the center, the portion covered with epithelium appears pearly or silver and shiny. Because it is thin and fragile, this area is easily damaged. The edge of a wound may be attached to the wound bed, unattached (undermining), or rolled inward (Figs. 6-8 and 6-9). Wound edges should be assessed as part of a thorough evaluation of the wound.

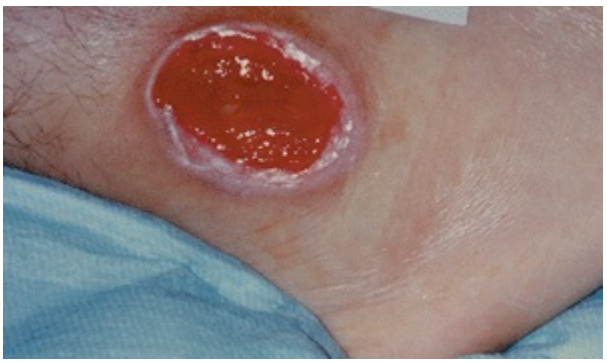


Figure 6-8. Wound edges with epithelialization. In this photograph, wound edges are attached and epithelialization is present.

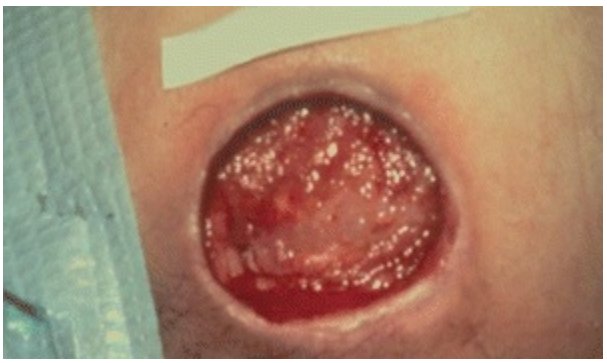


Figure 6-9. Rolled edges. Wound edges rolled inward.

Examining the wound edges may reveal whether the wound is acute or chronic and can often provide clues as to the wound's etiology. For example, a wound with inflamed edges or violaceous with undermined borders may indicate pyoderma gangrenosum. A wound with edges rolled inward may be too dry, causing the wound edges to seek more moisture from

the wound bed. A wound that is covered in necrotic tissue, desiccated, or deprived of oxygenation will exhibit poorly defined wound margins.[26]

Epithelialization can also occur in the middle of a wound bed if hair follicles or new cell growth is present. The appearance of new tissue at the wound edge can be measured in centimeters or by the percentage of wound coverage (e.g., "0.3 cm of epithelial tissue surrounds the wound" or "wound is 25% epithelialized"). The degree of epithelialization is often overlooked.

Necrotic Tissue

Necrotic tissue is dead, devitalized, avascular tissue that provides an ideal medium for bacterial proliferation and may inhibit healing. It's a well-known theory that wound healing is optimized when all necrotic tissue is removed from the wound bed. Necrotic tissue may present as yellow, gray, brown, or black. As it becomes dry, it presents as thick, hard, leathery black eschar.[26] Yellow, stringy necrosed tissue is referred to as slough.[26] Document the type and percent of necrotic tissue in the wound bed. For example, the wound bed may be 100% necrotic or 25% granular with 75% necrotic tissue. Slough is not present in stage I and II pressure ulcers[3] (Fig. 6-4). Biofilm can also be found in chronic wounds. A biofilm is "an aggregate of microorganisms known to cause chronic inflammation … they have enhanced resistance to destruction by endogenous antibodies and phagocytic cells, as well as by exogenous antibiotics and antiseptics. Biofilms play an important role in maintaining a chronic inflammation state ultimately leading to the failure to heal of skin wounds."[3,34]

Wound Bed Tissue

The wound bed tissue reveals the phase and progress of wound healing through observation of its color, degree of moisture—a moist wound bed facilitates movement of fibroblasts and macrophages, as well as collagenase, and other chemicals, across the wound bed, resulting in healing —and amount of epithelialization.[26,35] The wound bed may be pale pink, pink, red, yellow, or black. Clean, granular wounds are typically described as red, and wounds with devitalized slough are described as yellow. Brown and black wounds are typically those with necrotic tissue or eschar or desiccated tissue; these wounds need to be debrided because this type of tissue slows the healing process.[26]

Is the wound bed moist or dry? The presence of moisture or dry tissue

will guide you in selecting the right dressing to create an environment that supports healing. Do you see new tissue growth—epithelialization at the wound edges or within the wound bed? Is granulation tissue present—that is, beefy red tissue with a granular or gritty appearance?

Documentation should be based on your observations.[36] Is the wound 100% granular tissue, or is it 25% filled with slough (yellow tissue) or necrotic (dead) tissue? All three tissue types can be found in the same wound, and assessing the amount of each type of tissue will help you determine the appropriate treatment and document the outcome of care based on improvement or deterioration, as indicated by wound tissue characteristics. Outcomes can then be tracked by percentage of improvement toward a clean granular wound bed (e.g., "the wound progressed from 75% necrotic tissue to 100% granular tissue").

Tenderness to touch or the amount of pain the patient reports—both in the wound itself and in the surrounding tissue—are also essential parts of your assessment. Wound pain is one of the secondary signs of infection. It's important to differentiate between constant and episodic pain (such as pain that occurs only with dressing changes). Use a validated pain assessment scale accepted by your facility[3] (see Chapter 7, Wound Bioburden and Infection, and Chapter 12, Pain Management and Wounds).

A variety of proteins are present in the wound bed. In a longitudinal study, a difference in 21 levels of wound proteins in different areas of wound tissue (peripheral vs. internal) was identified between chronic pressure ulcers and those that healed.[37]

Assessing and Measuring Healing

Although wound assessment and measurement are important, so is documenting wound healing. There is growing research on the best ways to determine the healing rates of different types of chronic wounds (pressure, venous, and diabetic neuropathic ulcers) as well as expected healing rates at 4 weeks.[38,39] The 2014 International Pressure Ulcer Prevention and Treatment Guidelines state that, for a pressure ulcer, one should "expect some signs of healing in most individuals within 2 weeks."[3] A variety of tools are available to assess and document healing in clinical practice, including the Pressure Sore Status Tool (PSST),[40,41] the Pressure Ulcer Scale for Healing (PUSH),[42] the Toronto Symptom Assessment System for Wounds (TSAS-W),[43] and the DESIGN-R from Japan.[44]

Originally developed for pressure ulcers, the PSST has been revised by the author, Dr. Barbara Bates-Jensen, to be used for all wounds and is now referred to as the Bates-Jensen Wound Assessment Tool (BWAT).[45] The BWAT includes 13 wound factors to be tracked over time, each of which is scored numerically. Wound location and shape are not scored. The total of the 13 factors reflects overall wound status.[45]

The PUSH tool, developed and revised by the NPUAP,[42] has been validated by research. This tool allows for the quick and reliable assessments necessary to monitor pressure ulcer healing over time and should be used at least weekly.[42,46] Three scores are developed: one is for surface area (L × W), one indicates drainage amount, and the third is for tissue type. The total score, which is the sum of these three factors, is plotted on a healing record (or graph) to depict healing over time. Although originally developed by the NPUAP to quantify healing in pressure ulcers, a study by Hon and colleagues[47] provides evidence that this tool is valid for monitoring and evaluating the progress of venous and diabetic ulcers.

The TSAS-W, which can be completed by either the patient or caregiver,[43] quantifies 10 wound-related symptoms. The patient or the caregiver ranks each symptom from 0 to 10. In a test on 531 patients, this tool was found to be useful for all wound types.

Another tool is the DESIGN from Japan, which is used to classify severity and monitor healing of pressure ulcers.[48] The tool uses six factors to classify and assess healing: *d*epth, *e*xudate, *s*ize, *i*nfection, *g*ranulation, and *n*ecrosis. Reported interrater reliability is good with both patients and photos, and there is very good correlation with the BWAT.[48]

Wound Documentation Essentials

Documentation is an essential component of wound assessment. Every wound assessment should be documented thoroughly, accurately, and legibly, with an accompanying signature as well as the date and time of the assessment. Wounds should be documented on the patient's admission, weekly, with each dressing change, upon any significant change in the wound, and upon discharge.

As mentioned earlier, the initial assessment and documentation become a baseline comparison for all future assessments. It is recommended that each clinical agency have a consistent chart form and format for wound documentation. All facilities should follow the wound assessment policy as

determined by their setting-specific regulations, but to identify ulcers/wounds present on admission, assessment and documentation should take place at least within the first 24 hours. These include the mandated Outcome and Assessment Information Set (OASIS)[49] for home care or the MDS 3.0 for long-term care[16] (see Chapter 2, Reimbursement Regulations Impacting Wound Care).

Practice Point

It's important to remember that in the United States, the OASIS and MDS are regulatory documentation tools—neither tool is considered a comprehensive wound assessment.

Wound Photography

Photographs can provide a visual record of the wound. When done correctly, they can assist the clinician in clinical care decisions and provide documentation support in the case of litigation[50] but should not replace bedside assessments. Standardization of when and how to photograph wounds is paramount. It is important to remember, however, that photographs do not replace bedside wound assessments.

When using photography, it's necessary to permanently mark on the photograph the date and time it was taken as well as patient identification information. The photo should also include a sample measure in each frame, such as a 10-cm strip of paper tape. For the best clarity when using digital photography, a camera no smaller than 1.5 megapixels is recommended. However, because digital photos can be altered, the identifying data must be encoded permanently. When periodic photos are taken, distortion of the photos can be a problem due to body contour, angle of the body in a bed, angle and distance of the camera from the patient, and lighting. Some photo software packages use a target plate in the photo and the software; once the photo is downloaded, it can automatically calculate wound area and volume as well as length and width. Wound photographs can quickly and accurately represent a wound's appearance if the proper equipment is used in the correct way.[51,52] Clinicians need appropriate training and must consistently follow a wound photography protocol. The angle at which the camera is held to the wound makes a difference. Using the wrong camera angle can

result in an improperly photographed wound that appears larger than it truly is (Fig. 6-10). Other strategies to enhance wound photos have been described elsewhere in the literature.[20,53]

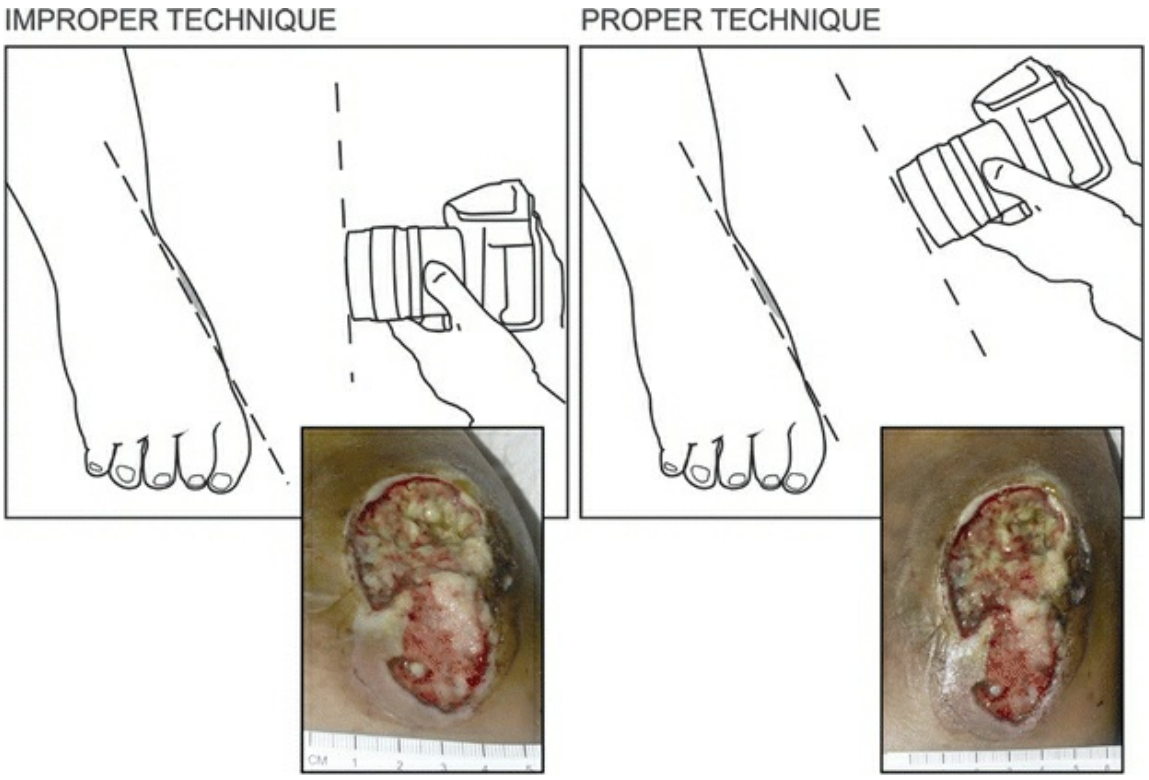


Figure 6-10. Proper photo technique. Used correctly, photography can be a great tool in documenting the appearance of a wound. Holding the camera at the appropriate angle is imperative to accurately document wound size. (Reprinted with permission from Rennert, R., Golinko, M., Kaplan, D., et al. "Standardization of Wound Photograph Using the Wound Electronic Medical Record," *Advances in Skin & Wound Care* 22(1):32-8, 2009. Copyright © 2009 Lippincott, Williams and Wilkins.)

Electronic Medical Records and Electronic Health Records

The EMR is the legal record created in hospitals and healthcare environments that is the source of data for the electronic health record (EHR). The EHR represents the ability to easily share medical information among stakeholders and to have a patient's information follow him or her through the various modalities of care in which he or she engages. EHRs are reliant on EMRs being in place, and EMRs will never reach their full potential without interoperable EHRs in place.[54]

A system of dedicated wound EMRs supports comprehensive and consistent care in patients with wounds. It promotes patient safety, facilitates evidence-based care,[52] and helps eliminate disparities in care regardless of care setting because the interdisciplinary team has access to objective wound assessment data.[2] In one large urban center, the use of an objective wound EMR decreased chronic wound emergencies.[2]

Telemedicine

Teleassessment and telemanagement are terms used to describe the "assessment and management of a wound at a distance using electronic information and communication technologies."[20] Telemedicine can enhance wound treatment in the home, at out-lying clinics, and in inclement weather when travel is impossible. In one randomized controlled study of 103 participants with 160 pressure ulcers or nonhealing surgical wounds receiving home care in conjunction with telemedicine, the average healing or improvement rate was 51 days for pressure ulcers and 34 days for surgical wounds.[55] The role of telemedicine in wound healing continues to evolve. In another descriptive comparative study of home care nurses with 43 adult patients, the use of digital photography rather than simply reporting resulted in a decreased chance of under- or overtreating the patients' wounds.[56] Use of digital images in telemedicine also resulted in the unexpected finding of more information than just what was revealed by conventional inspection, such as the identification of other factors in the patient's environment beyond the local wound bed that may have inhibited wound healing.[56] These included, for example, the patient's bed, wheelchair, redistribution devices, and shoes.[56] In areas of the country where weather can prohibit travel to a wound care center or the patient is unable to be transported, telemedicine has been an immense facilitator of timely and appropriate wound assessment and treatment. Truly this was an example of the importance of treating the "whole patient and not just the hole in the patient."

Summary

Wound assessment—an appraisal of a patient's condition based on clinical signs and symptoms, laboratory data, and medical history—is an integral part of wound management. Assessment has become a highly specialized

area of care, requiring well-developed observational skills and current knowledge. The use of current terminology is vital to accurate assessment and communication. Use of the Wound ASSESSMENTS[15] chart or WOUND PICTURE[36] mnemonic can provide a fast, ongoing, and accurate assessment for patients with wounds[39] (Box 6-4). Other tools that quantify wound characteristics, such as the BWAT,[45] PUSH,[42] TSAS-W,[43] DESIGN,[48] or DESIGN-R,[44] can help the clinician track wound healing. Proper assessment of wound parameters provides clinicians with the information they need to make the decisions that guide the wound care team to suitable interventions, management, and care strategies. Technology will continue to help clinicians with documentation. Unless standardized techniques and protocols are followed, wound photos can hinder, rather than help with treatment care decisions. EMRs and their important role in EHRs are the wave of the future. These advances, coupled with the use of telemedicine, ensure that the future of wound assessment and documentation looks promising.

Box 6-4 Wound Picture

When assessing wounds in your patient, use the mnemonic, **WOUND PICTURE**, for a fast and accurate assessment.

Wound or ulcer location

Odor Assess before and during all dressing changes

Ulcer category, stage (for pressure ulcer) or classification (for diabetic ulcer), and depth (partial thickness or full thickness)

Necrotic tissue

Dimension of wound (shape, length, width, depth); drainage color, consistency, and amount (scant, moderate, large)

Pain (when it occurs, what relieves it, patient's description, patient's rating on scale of 0 to 10)

Induration (surrounding tissue hard or soft)

Color of wound bed (red-yellow-black or combination)

Tunneling (record length and direction—toward patient's right, left, head, feet)

Undermining (record length and direction, using clock references to describe)

Redness or other discoloration in surrounding skin

Edge of skin loose or tightly adhered? Edges flat or rolled under

From *Wound Care Made Incredibly Easy*, 2nd ed. Philadelphia, PA: Lippincott, Williams & Wilkins, 2006.

PATIENT SCENARIO

Clinical Data

Mr. X is a 54-year-old gentleman with a painful stage IV pressure ulcer on the right trochanter. There is a large amount of light beige exudate that has some blood in it. There is a small amount of black eschar in the wound bed, while the rest of the wound bed is red and granular.

Case Discussion

After the nurse removes the dressings and cleanses the pressure ulcer, he or she completes the wound assessment chart documentation (see Box 6-1).

Wound etiology: Pressure ulcer

Anatomic location: right trochanter; date of onset: chronic, 6 weeks ago

Stage/category: IV; size: 7.5 × 6.2 × 2.5 cm (L × W × D); shape: circular

Sinus tract/tunneling: none present; undermining: 0.75 cm from 1 o'clock to 4 o'clock

Exudate: large amount, thin, tannish/serosanguineous; malodorous drainage

Sepsis: local

Surrounding skin: erythematous, intact, slightly swollen; temperature: skin warm to touch

Maceration: not present

Edges/epithelialization: attached and slightly rolled;

epithelialization: small amount present, 0.05 cm

Necrotic tissue: present; black, <10%

Tissue wound bed: granulation, present 90%; tenderness to touch: pain present; anytime; pain scale 9/10

Status: initial assessment date, 6/23/10; nutritional, PT consult ordered 6/23/10

Signature: A Nurse RN_____

Show What You Know

1. Initial wound assessment involves all of the following except:

A. observation.
B. data collection.
C. evaluation.
D. surgical debridement.

2. A wound that has tissue damage through the epidermis and partially into the dermis would be classified as:

A. superficial.
B. partial thickness.
C. full thickness.
D. subdermal.

3. In assessing a wound, you find an area of tissue destruction under the edge of the patient's wound. This is best described as:

A. a sinus tract.
B. maceration.
C. fistula.
D. undermining.

4. Which of the following wound photography techniques is <u>incorrect</u>?

A. Include a measuring device in the photo.
B. Permanently mark the date and time of the photo in the picture.
C. Hold the camera at a 90-degree angle to the wound.
D. Digital photos should be at least 1.5 megapixels.

5. Which of the following statements about electronic health records is

false? They are:

A. the same as electronic medical records.
B. reliant on electronic medical records being in place.
C. an important way of sharing medical information across stakeholders.
D. a modality of care that holds promise to promote patient safety across care settings.

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Bioburden Infection

7

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The contributions of Sue E. Gardner PHD, RN, and Rita A. Frantz, PHD, RN, FAAN, to the previous editions of this chapter are greatly appreciated.

Objectives

After completing this chapter, you'll be able to:

- compare and contrast the various phases of the wound bioburden cycle (contamination, colonization, critical colonization/biofilm, and infection)
- propose how to identify infection using signs and symptoms and diagnostic tools
- describe biofilm, how it is formed and its role in wound healing
- propose treatment for increased bioburden, biofilm, and infection.

Bioburden in Wounds

Microorganisms and man normally live in harmony.[1] Gene sequencing of the skin microbiome has shown that organisms living on the skin are associated with moist, dry, and oil microenvironments.[2] Thus, the microorganisms at one wound site are not necessarily like those at another site on the same person nor is the microbiome of one person like that of another.

Practice Point

The microbes that inhabit one body site may not be the same as

found in another site on the same person.

The skin is a mechanical barrier to organisms, and skin oils and sweat are chemicals that help prevent their entrance into tissue.[1] When a wound occurs, organisms from the skin, gastrointestinal track, and mucous membranes as well as the external environment have free access to tissue. Bioburden is a measure of the number of viable microbes on a tissue surface or device.

Practice Point

Bioburden is a measure of the viable organisms in a wound or on a tissue surface.

Until recently, scientists and clinicians understood that infection was due to planktonic organisms, those who float free when placed in water.[3] These organisms are individual cells that have a predictable life cycle and work independently. There is a good understanding of planktonic organisms, as seen by the fact, that antibiotics have been developed that target-specific sites to kill or immobilize them. Our growing understanding of biofilm has radically changed our perspective on microorganisms, how they function, and their effect on wound infection.[4]

Practice Point

Antibiotic treatment is based on knowledge of planktonic organisms.

Planktonic organisms of all types float freely in the air and exist on the skin and surfaces in the environment. With injury, organisms contaminate the wound surface. The organisms attach to the surface, change their genetic code (become biofilm genotype) and exude the extracellular polymeric substance (EPS) that provides the structure for the biofilm. Biofilm is defined as communities of microorganisms attached to a surface (living or

not) embedded within a hydrated matrix of extracellular polymeric substance (EPS).[5,6] It is composed of polysaccharide-based substance, although it includes proteins, lipids, and DNA. Biofilm provides protection against antimicrobials and host defenses. The communities of organisms that form are genetically different from each other as well as their planktonic predecessors.[7] The organisms, in the biofilm community, work synergistically, communicate with each other and other communities (through a process called quorum sensing), are able to change their metabolism, and are resistant to antibiotics.[3,8]

Biofilm communities are usually composed of multiple organisms. They form fluid-filled channels that allow oxygen and nutrients to get to the cells and carry away waste. Their ability to reduce their metabolism allows these organisms to use little oxygen and nutrients and become dormant, only later to be reactivated. Biofilms impair healing as the EPS helps the organisms evade the immune system and contributes to perpetuating the inflammatory phase so the wound turns into a chronic nonhealing wound.[5,8]

Practice Point

Biofilm protects organisms from antibiotics.

At some point, the biofilm communities come to exceed their nutrient/oxygen capacity and send off organisms into the wound environment (seed the environment).[3] Essentially, the cells released are planktonic organisms that float freely in the moisture of the wound fluid and go on to repeat the process of setting up a new biofilm. The newly released planktonic organisms also stimulate a continuation of the inflammatory response. Meanwhile, the EPS prevents antibiotics from reaching the organisms; in addition, the organisms constantly change their genetic code, making it difficult for antibiotics to kill them.

Practice Point

Scientific discoveries have radically changed the way we think of infection. Evidence-based practice requires current scientific

knowledge about biofilm as the basis for safe care.

Biofilm can form throughout the body, for example, plaque on teeth, thick secretions with cystic fibrosis.

The biofilm generally is not visible because of its location, except perhaps in wounds. Some authors suggest that biofilm in wounds may be observed by expert clinicians as translucent films or opaque yellow-green material.[6] Others do not believe it is visible.[3,5] For the most part, clinicians can only infer the presence of biofilm genotype organisms based on signs and symptoms. Sophisticated diagnostic tools needed to identify biofilms are not currently available in clinical practice.[3,5]

Seminal work showed that biofilms occur more frequently in chronic wounds than acute wounds (60% of chronic wounds and 6% of acute wounds).[9] Thus, in this chapter where appropriate, the differentiation between acute and chronic wounds is presented.

Practice Point

Biofilm occurs much more frequently in chronic wounds (every 3 out of 5 wounds) than in acute wounds (1 in 12 wounds). The physical characteristics of biofilm have not been identified; the presence of biofilm is inferred from signs and symptoms.

Wound Bioburden Cycle

Predictable processes occur that help us understand how exposure to bioburden can lead to infection or eradication of invading organisms in wounds. Risk of wound infection involves the interaction of the individual's immune system, the environment, and the organisms.[3,10] Increased risk of infection occurs in patients who are immunocompromised as occurs with therapeutic chemotherapy for cancer and persons receiving prednisone. Infection risk also is increased when the environment is moist, warm, lacks appropriate pH, and sufficient oxygen and when there is a large load of bioburden present. The nature of the organisms also affects the risk of infection and depends on their constitutional needs such as oxygen requirement (aerobic/anaerobic) and inherent virulence, for example, high

virulence as seen in beta hemolytic *Streptococcus*.

The wound bioburden cycle describes the processes associated with the increasing invasion of a wound by organisms (Table 7-1). The first phase of this cycle is contamination, and it begins with injury that disrupts the skin. Examples of tissue injury are a surgical incision, trauma, or a bite. Microorganisms present in the environment contaminate the wound. The full range of microorganisms may be included (bacteria, fungi, viruses), and they may be endogenous, that is, from the patient's skin or gastrointestinal track, mucous membranes or exogenous, that is from external contamination, for example, healthcare workers, trauma. Contamination cannot be seen nor does it generate clinical signs and symptoms. The organisms are planktonic genotype (free floating) and are normally managed by host defenses and the bioburden cycle terminated at this phase.[3]

Table 7-1 Phases of the Wound Bioburden Cycle

Phase	Definition	Signs and Symptoms
Contamination	Organisms present on wound surface.	No signs or symptoms.
Colonization	Organisms multiply on wound surface. Early reversible adherence, later irreversible adherence of organisms to the tissue.	No signs or symptoms.
Critical colonization/biofilm	Organisms form biofilm on wound surface.	Delayed healing. Local infection.
Infection	Organisms invade tissue and systemic response occurs.	Fever, leukocytosis, inflammation (redness, warmth, increased exudate, pain, fever, leukocytosis).

Practice Point

Contamination and colonization cannot be seen and produce no signs or symptoms. This phase of the wound bioburden cycle offers the best opportunity for removal of organisms from the wound, before the organisms become attached to the wound surface.

If the microorganisms are not removed, they assess the environment with receptors on their cell wall and attach to the surface. At this point, the organisms' attachment is reversible, that is, the organism can be washed away with irrigation.[3] Once lightly attached, the organisms use the wound bed as a source of nutrition, oxygen, etc. The organisms soon become

irreversibly attached, and early stages of biofilm are formed. Over time, the biofilm genotype develops, and the organisms begin to modify their genetic profile and multiply. Colonization cannot be seen and does not produce signs and symptoms.

Within 24 hours of contamination, the organisms begin to develop a thin layer of EPS, and the layer becomes thicker as the biofilm organisms continue to lay down layers of ESP.[6] Biofilm forms during the critical colonization/biofilm phase, and this phase is characterized by local infection and delayed healing.[5,10] In fact, it has been proposed that the concept of critical colonization represents the effects of the biofilm,[11] so in this chapter, critical colonization is termed critical colonization/biofilm.

Practice Point

The critical colonization/biofilm phase occurs when organisms create local communities in the biofilm on the wound. Local infection and delayed healing are visible to the clinician and characterize this phase.

Infection occurs when the organisms invade the tissue and produce systemic signs. Infection is characterized by inflammation (redness, heat, edema, pain, and increased exudate.[12,13] It is treated with antibiotics, but they are effective only when the organisms are metabolically active.

Practice Point

Infection occurs when organisms invade the tissue and produce systemic signs. Antibiotics used to treat wound infection function only when organisms are metabolically active.

Table 7-1 summarizes the phases of the wound bioburden cycle and their associated signs and symptoms. It should be noted that with the increasing availability of DNA-based diagnostic protocols, a change in this paradigm may be needed that alters the phases of the cycle. One such

proposed paradigm is contamination, attachment, proliferation, critical concentration of signaling molecules, adaptation of the biofilm phenotype, and polymicrobial biofilm.[14]

Defenses Against Infection

Injury mobilizes a predictable, albeit complex, sequence of events that are designed to lead to healing (see chapter 5, Acute and chronic wound healing). These processes also provide a network of protection against wound invasion by microorganisms. With initiation of the inflammatory response to tissue injury, there is an influx of polymorphonucleocytes (PMNs) and monocytes. Of the PMNs, neutrophils are the most common and arrive immediately after injury and predominate for the first 48 hours.[15] The monocytes enter the wound space immediate after the PMNs and are transformed into macrophages, reaching their peak within 24 hours of injury. Both the leukocytes and macrophages are important in quickly infiltrating the area of tissue damage and removing microorganisms by phagocytosis. These WBCs work best in a well-perfused oxygen-rich environment as phagocytes require oxygen (oxygen burst).[16]

Proinflammatory mediators, reactive oxygen species, proteases, and antimicrobial peptides are released into the wound and result in microbial killing as well as tissue damage. Normally, the processes stimulated by the proinflammatory mediators that lead to inflammation and immune cell activation are reversed when the neutrophils die (apoptosis) and are engulfed by macrophages.[15] In acute wounds, this occurs by about day 5, and wound infection is circumvented.

Practice Point

Inflammation is important to fighting infection early after injury. Continued inflammation over time characterizes chronic nonhealing wounds.

What was a positive response to injury becomes a negative one when the inflammatory cycle continues beyond its normal duration. Specifically, chronic wounds inflammatory cells continue to be recruited; generate reactive oxygen species, proteases, and antimicrobial peptides that kill

microorganisms; and result in tissue damage. The organisms themselves may participate in protective processes, for example, *Pseudomonas aeruginosa* may contribute to inflammation by producing rhamnolipids that protect them from phagocytosis.[17]

Proteases are especially problematic as they cause degradation of the extracellular matrix and tissue damage. A negative spiral is set up where damage caused by neutrophil generated proteases and chronic inflammation delays wound healing (delayed reepithelialization, impaired collagen generation, cells at the edge of the wound).[15,18] The biofilm protective mechanisms are in place, and wound infection occurs when the microorganisms overtake the individual's immune system.

From a clinical perspective, it is important to recognize that the body's defense against micro-organisms works effectively in most acute wounds as seen by the low rate (<2%) of surgical site infection (SSI), recognizing that even at this rate, SSI is the most common hospital acquired complication.[19] Comparatively, chronic wounds present a grim picture with the prevalence of wound infection estimated to be 53%.[20] With more than 40 million inpatient surgeries each year and the increasing incidence of chronic diseases that often involve chronic wounds, for example, diabetes, venous disease, it would behoove us to be vigilant in applying evidence-based care to mitigate wound infection and its sequelae. While the etiology of various types of chronic wounds differ (e.g., diabetic ulcers are due to arteriosclerotic peripheral vascular disease often complicated with peripheral neuropathy, pressure ulcers are caused by unrelieved pressure), there is agreement in the field that the pathway for chronic wound infection is applicable across the chronic wound types.[3]

Defining Infection

Wound infection is the invasion and multiplication of microorganisms in wound tissue resulting in pathophysiologic effects or tissue injury[21] or the presence on any beta-hemolytic *Streptococcus*.[22] The definition sets out the criteria for a wound infection: tissue must be viable, organisms invade tissue, organisms multiple, patient defenses are overwhelmed, and signs and symptoms of infection are visible. Also, beta-hemolytic *Streptococcus* needs to be identified early because of their well-recognized virulence. Necrotic tissue and slough by definition are dead so infection cannot be present in them; however, dead tissue does support proliferation of

organisms and so needs to be removed.[18] Also, pus is the product of phagocytosis and digestion of dead microorganisms by WBCs; pus is not indicative of infection.

Practice Point

Infection occurs in living tissue so cannot occur in debris; however, debris (slough, necrotic tissue, old sutures) does support proliferation of organisms and so needs to be removed.

The definition of infection was established based on acute wounds and the existing understanding of planktonic organisms. Since then, our understanding of biofilm in chronic wounds and biofilm genotype organism behavior has grown. In addition, the National Institutes of Health (NIH) has declared that 80% of infections are due to biofilm.[23] As invasion during critical colonization/biofilm results in delayed healing, the question must be posed whether wounds in the critical colonization/biofilm phase of the wound bioburden cycle are infected and should be treated as infected. The expectation is that in the near future, the availability of bedside molecular diagnostic techniques will allow better definition of bioburden and biofilm and development of more effective treatment of offending organisms.[14]

Wound infection currently is diagnosed based on its signs and symptoms.[5,13] Classic characteristics of wound infection are erythema, heat, swelling, and pain.[3,12] One of the challenges in clinical practice is differentiating infection from inflammation. Inflammation normally is present for 3 to 5 days after injury and is characterized by the same signs.[5,13] The question becomes how to differentiate these two processes and especially in chronic wounds where the inflammatory response often is more muted.

The most common acute wounds are surgical and the criteria for SSI are used for acute wounds. The definition for SSI was established more than 20 years ago[24] and continue to be the criteria used by the Centers for Disease Control (CDC).

Chronic wounds, on the other hand, are characterized by a chronic inflammation and biofilm. However, the identification of infection in chronic wounds is tricky as the signs and symptoms may be more subtle than

in acute wounds and indicators of moving from critical colonization to infection may not be recognized.

To date, there are not universal criteria for infection in chronic wounds. Lack of criteria contributes to inconsistent diagnosis of chronic wound infection and potentially to inappropriate treatment (under or overtreatment). The problem of lack of widely accepted criteria is compounded by the lack of clinical diagnostic tools to evaluate the contribution of the biofilm to infection in chronic wounds.[4,5]

Signs and Symptoms of Wound Infection

SSI is divided into incisional and organ/space. Incisional SSI is further divided into superficial and deep SSI.[24] Superficial SSI sites include the skin and subcutaneous tissue while deep SSI designates infection in soft tissues, fascia, and muscle layers of the incision. Organ/space SSIs involve any part of the anatomy other than the incision or that manipulated during the operation (Table 7-2).

Table 7-2 Diagnostic Criteria for Incisional and Organ/Space Surgical Site Infection[24]

	Incisional		Organ/Space
	Superficial[a]	Deep	
Purulent drainage	From superficial incision.	From deep incision but not organ/space.	From a drain placed through stab wound into organ/space.
Culture	Positive from aseptically obtained fluid or incision from superficial incision.		Positive from aseptically obtained culture of fluid or tissue in organ/space.
Signs and symptoms	AT LEAST ONE: pain or tenderness, localized swelling, redness, heat AND incision is deliberately opened UNLESS incision culture is negative.		
Dehiscence		Spontaneously or is deliberately opened when patient has AT LEAST one: fever (>38°C), localized pain, or tenderness, UNLESS culture is negative.	
Abscess		Found on direct examination, reoperation or histopathology or radiology.	Found on direct examination, reoperation or histopathology or radiology.
MD or surgeon diagnosis	Of superficial incisional SSI.	Of deep incisional SSI.	Of organ/space SSI.

[a]Not superficial SSI: stitch abscess, episiotomy or neonate circumcision, infected burn, incisional SSI that extends into fascia and muscle.

SSI refers to infection at the site of an incision. All SSI occurs within 30 days of surgery, except deep SSI that involves an implant. The timeframe for these may extend up to 1 year if an implant is in place, the SSI appears related to the implant surgery, and the infection meets the anatomic criteria for deep SSI.

Practice Point

Diagnosis of wound infection in acute wounds is based on the depth of the infection. In addition, the criteria for infection in acute and chronic wounds are not the same.

A number of researchers have examined the signs and symptoms of infection in chronic wounds. Cutting and Harding[12] identified traditional infection criteria (abscess, cellulitis, discharge [serous exudate with inflammation, seropurulent, hemopurulent, pus]) as well as the additional criteria for granulating tissues: delayed healing, discoloration, friable granulation tissue that bleeds easily, unexpected pain/tenderness, pocketing at the base of the wound, bridging of epithelium or soft tissue, abnormal smell, and wound breakdown.[12] The validity of these criteria was tested on ward nurses (n = 20) working in pairs, who were asked to identify if each of four wounds was infected. Findings were compared with wound infection diagnosis by the principal investigator (an MD) and swab culture results. Descriptive statistics were reported that showed the nurses had less than chance agreement with the MD (47.5%), and the MD had nearly perfect agreement (97.5%) with culture findings.[25] The relationship of the nurses' infection classification with the culture data was not reported. These data indicate that the assessment of the presence of wound infection is not consistent among healthcare providers. The author concluded that the nurses needed more education to improve their performance.

Practice Point

Practitioners responsible for identifying wound infection need knowledge about the criteria and practice in applying them.

Subsequently, a landmark study by Gardner et al.[13] tested the Cutting and Harding.[12] They studied signs and symptoms of infection in patients with chronic wounds (n = 36), 11 of whom were infected based on biopsy with both aerobic and anaerobic analyses.[13] Classic signs of infection (pain, erythema, edema, heat, purulence) were compared with signs of infection in secondary wounds (serous drainage with inflammation, delayed healing, discolored granulation tissue, friable granulation tissue, pocketing at the base of the wound, foul odor, and wound breakdown). The Clinical Signs and Symptoms Checklist,[13] comprised the signs and symptoms with operational definitions and mutually exclusive evaluative categories, was used to measure the signs and symptoms (Box 7-1). Findings showed that the signs and symptoms of chronic wounds were better indicators of infection in chronic wounds than were the classic signs (sensitivity 0.62, specificity 0.38). Valid indicators of infection in chronic wounds were based on a combination of the sensitivity, specificity, discrimination power, and positive predictive power and were increasing pain, friable granulation tissue, foul odor, and wound breakdown. Specificity of increasing pain (1.0) and wound breakdown (1.0) were robust enough to be considered signs of infection.

Box 7-1 Clinical Signs and Symptoms Checklist[13a]

Clinical signs and symptoms checklist

Signs and symptoms	Check (+) if present
Increasing pain in the ulcer area The patient reports increased level of peri-ulcer pain since the ulcer developed. Ask him to select the most appropriate statement for current level of ulcer pain from the following choices: 1. I can't detect pain in ulcer area. 2. I have less ulcer pain now than I had in the past. 3. The intensity of the ulcer pain has remained the same since the ulcer developed. 4. I have more ulcer pain now than I had in the past. If the patient selects number 4, his pain is increasing. Write n/a if the patient can't respond to the question.	☐
Erythema The presence of bright or dark red skin or darkening of normal ethnic skin color immediately adjacent to the ulcer opening indicates erythema.	☐
Edema The presence of shiny, taut skin or pitting impressions in the skin adjacent to the ulcer but within 4 cm from the ulcer margin indicates edema. Assess pitting edema by firmly pressing the skin within 4 cm of ulcer margin with a finger, release and waiting 5 seconds to observe indentation.	☐
Heat A detectable increase in temperature of the skin adjacent to the ulcer but within 4 cm of the ulcer margin as compared with the skin 10 cm proximal to the wound indicates heat. Assess differences in skin temperature using the back of your hand or your wrist.	☐
Purulent exudate Tan, creamy, yellow, or green thick fluid that's present on a dry gauze dressing removed from the ulcer 1 hour after the wound was cleaned and dressed indicates purulent exudate.	☐
Sanguineous exudate Bloody fluid that's present on a dry gauze dressing removed from the ulcer 1 hour after the wound was cleaned and dressed indicates sanguineous exudate.	☐
Serous exudate Thin, watery fluid that's present on a dry gauze dressing removed from the ulcer 1 hour after the wound was cleaned and dressed indicates serous exudate.	☐
Delayed healing of the ulcer The patient reporting no change, or an increase in the volume or surface area of the ulcer, over the preceding 4 weeks indicates delayed healing. Ask the patient if the ulcer has filled with tissue or is smaller around than it was 4 weeks ago.	☐
Discoloration of granulation tissue Granulation tissue that is pale, dusky, or dull in color compared with surrounding, healthy tissue. Note variations of normal, beefy red appearance of granulation tissue.	☐
Friable granulation tissue Bleeding of granulation tissue when gently manipulated with a sterile cotton-tipped applicator indicates friable tissue. **Pocketing at base of wound** The presence of smooth, nongranulating pockets of ulcer tissue surrounded by beefy red granulation tissue indicates pocketing.	☐
Foul odor The ulcer may have a putrid or distinctively unpleasant smell.	☐
Wound breakdown Small open areas in newly formed epithelial tissue not caused by reinjury or trauma indicate wound breakdown.	☐

Practice Point

Signs of wound infection in chronic wounds are increasing pain, friable granulation tissue, foul odor, and wound breakdown; the strongest of these indicators are increasing pain and wound breakdown.

The Gardner[26] research team then went on to examine individually the same signs and symptoms in chronic diabetic foot ulcers (DFUs), those of the Guidelines of the Infection Disease Society of America (IDSA),[27] and a composite predictor based on all the signs and symptoms of infection.[26] Of the 64 participants, 25 had a DFU with a high microbial load ($>10^6$). Using the same methods as in the 2001 study, researchers evaluated whether the IDSA predicted ulcers. Findings showed no individual sign was a significant predictor of high microbial load nor was the IDSA combination. The composite predictor performed moderately well (concordance = 0.645 with 95% confidence interval 0.559 to 0.732).

At the same time, Sibbald and colleagues[10] explored ways to translate existing research findings, so they could be used in clinical practice. They proposed diagnostic criteria using the acronym NERDS for critical colonization (Fig. 7-1) and STONEES for infection (Fig. 7-2). With NERDS acronym, critical colonization/biofilm is present in chronic wounds when three or more of the signs/symptoms are present. With the STONEES criteria, infection was present when any three of the STONEES criteria are observed.

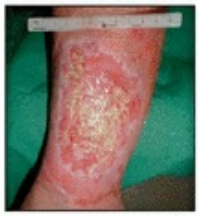
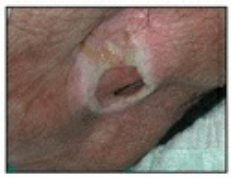
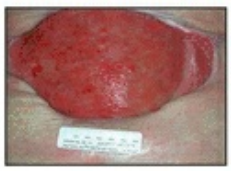
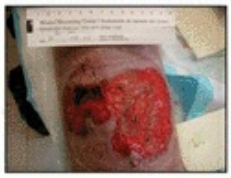
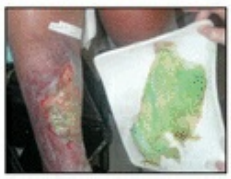
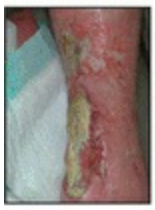
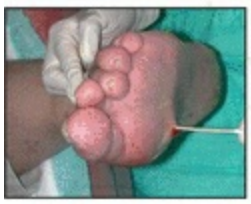
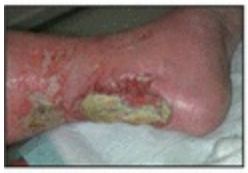
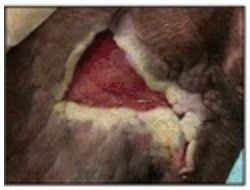
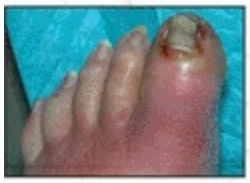
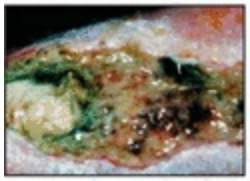
Letter	Key information to know	
NONHEALING WOUND 	• The wound is nonhealing despite appropriate interventions (healable wound with the cause treated and patient-centered concerns addressed). • Bacterial damage has caused an increased metabolic load in the chronic wound, creating a proinflammatory wound environment that delays healing.	• To determine a healing trajectory, the wound size should decrease 20% to 40% after 4 weeks of appropriate treatment to heal by week 12. • If the wound does not respond to topical antimicrobial therapy, consider a biopsy after 4 to 12 weeks to rule out an unsuspected diagnosis, such as vasculitis, pyoderma gangrenosum, or malignancy
EXUDATIVE WOUND 	• An increase in wound exudates can be indicative of bacterial imbalance and leads to periwound maceration. • Exudate is often clear before it becomes purulent or sanguineous.	• Increased exudates needs to trigger the clinician to assess for subtle sign of infections. • Protect periwound area using the LOWE© memory jogger (Liquid film-forming acrylate; Ointments; Windowed dressings; External collection devices) for skin barrier to wound margins.
RED AND BLEEDING WOUND 	• When the wound bed tissue is bright red with exuberant granulation tissues and bleeds easily, bacterial imbalance can be suspected.	• Granulation tissue should be pink and firm. The exuberant granulation tissue that is loose and bleeds easily reflects bacterial damage to the forming collagen matrix and an increased vasculature of the tissue.
DEBRIS IN THE WOUND 	• Necrotic tissue and debris in the wound is a food source for bacteria and can encourage a bacterial imbalance.	• Necrotic tissue in the wound bed will require debridement in the presence of a dequate circulation. • Debridement choice needs to be determined based on wound type, clinician skill, and resources.
SMELL FROM THE WOUND 	• Smell from bacterial by products caused by tissue necrosis associated with the inflammatory response is indicative of wound-related bacterial damage. *Pseudomonas* has a characteristic sweet smell/ green color; anaerobes have a putrid odor due to the breakdown of tissue.	• Clinicians need to differentiate the smell of bacterial damage from the odor associated with the interaction of exudates with different dressing materials, particularly some hydrocolloids. Odor may come from superficial or deep tissue damage, and this should not be relied on along with exudates alone as the only signs of increased superficial bacterial burden.

Figure 7-1. NERDS©: Superficial increased bacterial burden.

Letter	Key information to know
SIZE IS BIGGER 	• Size as measured by the longest length and widest width at right angles to the longest length. Only very deep wounds need to have depth measured with a probe. • An increased size may be due to deeper and surrounding tissue damage by bacteria or alternately because the cause has not been treated or there is a systemic or local host factor impairing healing.
TEMPERATURE INCREASED 	• With surrounding tissue infection, temperature is increased. This may be performed crudely by touch with a gloved hand or by using an infrared thermometer or scanning device. There should be a high index of suspicion for infection if >3°F difference in temperature exists between two mirror-image sites.
OS (PROBES TO OR EXPOSED BONE) 	• There is a high incidence of osteomyelitis if bone is exposed or if the clinician can probe to the bone in a person with a neurotrophic foot ulcer. • An MRI is probably the most discriminating diagnostic test and considered necessary for diagnostic dilemmas.
NEW AREAS OF BREAKDOWN 	• Note the satellite areas of skin breakdown that are separated from the main ulcer. • It is important to remember this may be due to the cause of the wound, infection, or local damage being left uncorrected.
EXUDATE 	• All of the features here are due to the inflammatory response. With increased bacterial burden, exudates often increases in quantity and transforms a clear or serous texture to frank purulence and may have a hemorrhagic component.
ERYTHEMA, EDEMA 	• The inflammation leads to vasodilatation (erythema), and the leakage of fluid into the tissue will result in edema.
SMELL 	• Bacteria that invade tissue have a "foul" odor. There is an unpleasant sweet odor from *Pseudomonas* gram-negative organisms, and anaerobe organisms can cause a putrid smell from the associated tissue damage.

Comments

- Clinicians need to have a consistent approach to measurement.
- An increased size from bacterial damage is due to the bacteria spreading from the surface to the surrounding skin and the deeper compartment. This indicates that the combination of bacterial number and virulence has overwhelmed the host resistance.

It is important to distinguish between infection and the other two potential causes of temperature change:

- A difference in vascular skin supply (decreased circulation; is colder).
- Inflammatory conditions are not usually as warm, but they can demonstrate a marked increase temperature with extensive deep tissue destruction (acute Charcot joint).

- Radiographs and bone scans are less reliable for diagnosis of osteomyelitis with loss of bone mass that occurs with neuropathy. Radiographs of well-calcified bone, such as pressure ulcers of the pelvis, may be more reliable. The majority of ulcers that probe to bone in other locations are less likely to be associated with osteomyelitis.

- Search for the cause of the satellite areas of breakdown and the need to correct the cause.
- Check for local damage and consider infection, increased exudates, or other sources of trauma.

- For exudates control, determine the cause and then match the absorbency of the dressing (non, low, moderate, heavy) to the amount of exudates from the wound.
- Assess surrounding skin to evaluate for maceration. Again, use the LOWE© memory jogger (Liquid film-forming acrylate; Ointments; Windowed dressings; External collection devices) for skin barrier for wound margins.

- For erythema and edema control, the cause or the tissue infection needs to be treated.

- Make sure the smell is from organisms and not from the normal distinct odor from the interaction of exudates with some of the dressing material.
- Systemic antimicrobial agents are indicated that will treat the causative organisms, and devitalized tissue should be aggressively debrided in wounds with the ability to heal.

Figure 7-2. STONEES©: Deep compartment infection.

A cross-sectional validation study of patients with leg and foot ulcers conducted by Woo and colleagues[28] found that the presence of any three NERDS signs had a sensitivity of 73% and a specificity of 80.5% when compared with semiquantitative swab cultures with scan or light growth. They also found that any three STONEES signs had a sensitivity of 90% and a specificity of 69.4% when compared with semiquantitative swab cultures with moderate-to-heavy growth.

Practice Point

Any three NERDS signs are indicative of critical colonization/biofilm, and any three STONES signs are indicative of wound infection.

Cutting and colleagues[29] sought expert opinion from international interdisciplinary wound experts to determine the clinical signs and symptoms of infection present in chronic wounds as well as those specific to a particular type of wound. Data showed that signs and symptoms common to all chronic (not acute wounds or burns) were cellulitis, malodor, pain, delayed healing, wound deterioration or breakdown, and an increased amount of exudate. Signs or symptom-specific criterion for infection in various chronic wounds are crepitus for pressure ulcers; phlegmon for neuropathic/diabetic ulcers; increase in local skin temperature for venous ulcers; and dry necrosis (that may become moist and boggy at the edges of the necrotic tissue) for arterial disease–associated tissue breakdown.

Practice Point

Expert opinion of international wound experts using the Delphi technique showed that signs and symptoms of infection in chronic wounds were cellulitis, malodor, pain, delayed healing, wound deterioration or breakdown, and an increased exudate.

A review by Reddy et al.[20] summarizes the literature that examines the relationship between deep tissue biopsy (gold standard) and signs and

symptoms of wound infection in patients with chronic wounds. Likelihood ratios (LRs) were used to identify the presence of infection. A positive LR predicts the presence of infection while negative values indicate that wound infection is not present. The likelihood of infection increases when an ulcer causes increasing pain (LR range 11 to 20); however, absence of decreasing pain is not as helpful in identifying the absence of pain (LR 0.64 to 0.88).[13,26] Classic signs of wound infection were not helpful in diagnosing chronic wound infection (LR small and including one).[13,26]

Practice Point

Classic signs and symptoms of wound infection are not helpful in identifying infection in chronic wounds.

When Reddy et al.[20] examined the literature, they found few studies that systematically examined signs and symptoms and compared them with tissue biopsy culture. Lack of using the gold standard was a major issue. For example, the often cited work of Cutting and Harding[12] proposed signs and symptoms of wound infection based on their clinical experience. In their subsequent work that examined the ability of nurses to identify wound infection based on their list of signs and symptoms, the standard of comparison was MD evaluation; swab cultures were used to test MD diagnosis based on the signs and symptoms from the list he had generated.[25] In the Delphi study that assessed the opinion of experts on signs and symptoms of chronic wound infection,[29] the signs and symptoms elicited were not tested against a standard criterion.

Reddy's team noted that the majority of studies had examined a single sign or symptom and its relationship with criteria whose validity had not been established. They also acknowledged that many of the signs are quite subjective (e.g., friable granulation tissue; odor) and interrater reliability had not been reported. Reddy et al.[20] concluded that data from the Gardner research team provides the strongest evidence for the relationship between signs and symptoms and tissue biopsy cultures. Data from the Sibbald[10] paradigm and the validation study of Woo and colleagues[28] were not considered in the review because they did not compare the signs and symptoms with tissue biopsy.

Diagnostic Tests

Wound Cultures

Tissue biopsy is the gold standard for wound infection. Those with greater than 10^5 CFU have been recognized as diagnostic of wound infection.[30] Krizek et al.[31] showed that skin grafts did not "take" when there were more than 10^5 organisms per gram of tissue. Similarly, Robson et al.[32] found that bacteria that exceeded 10^5 per gram of tissue inhibited successful secondary closure of wounds. Also at this same time, Mack[33] explored the effect of topical antibiotic on skin flora. Classic work of Robson and Heggers[30] using a sample of wounds (n = 41) of mixed etiology showed that the quantitative tissue biopsy method had a sensitivity of 100%, a specificity of 93.5%, and an accuracy of 95.1% in predicting the success of delayed closure laid the foundation for biopsy as the gold standard for infection.

Subsequently, studies used 10^6 as the criterion for wound infection. Krizek and Davis[34] found that fatal sepsis was associated with visceral or blood cultures greater than 10^6 or 10^7 organisms per gram of tissue or milliliter of blood. These researchers also demonstrated that fatal wound sepsis was related to the number of bacteria in the wound.[35] In addition, Noyes and colleagues[36] found that wound exudates with greater than 10^6 bacteria per milliliter were associated with invasive infection. The U.S. Army Surgical Research Unit provided a series of studies that found burn wound sepsis was associated with bacterial levels exceeding 10^5 organisms per gram of tissue.[37,38]

Part of the issue is how to interpret greater than 10^5 CFU. Does it mean 100,000 or does it mean 1,000,000? References to greater than 10^5 have been interpreted as 100,000 or 1,000,000[39] organisms per gram of tissue. Some research[30,40] has used greater than 10^5, while others[41] have used 10^6. In addition, gram stain criteria for infection is the presence of 10^6 or more organisms.[42] While there is not universal agreement on the number, use of 10^6 to define infection is recommended.

Wound cultures measure planktonic organisms and underestimate the number of organisms present. In addition, recent understanding of bioburden raises the question of whether culture findings can accurately capture the

bioburden in a wound, especially in chronic wounds.[4] However, for the moment, until strategies to measure bioburden in biofilm and ways to interpret those data are more universally available, culture will remain the gold standard for wound infection.

Practice Point

Tissue biopsy with 10^6 or greater organisms or the presence of beta-hemolytic *Staphylococcus* is recommended as the criterion for wound infection.

Three approaches to culturing are available: biopsy, aspiration, and swab. Table 7-3 describes important dimensions of the procedure.

Table 7-3 Comparison of Three Techniques for Wound Culture

	Biopsy	Aspiration	Swab with Levine Technique
Performed by	MD or provider with appropriate authority and preparation in biopsy.	MD or provider with special education in technique and understanding of anatomy.	Nurse or physical therapist that have been taught how to collect specimen.
Location of specimen	Clean granulation tissue in wound bed.	Intact skin adjoining wound.	Clean granulation tissue in wound bed.
Cleansing before sampling	Best taken immediately after debridement; if not rinse with saline that has no preservative.	Intact skin where need will be inserted is cleansed with antibacterial solution.	Rinse with saline that has no preservative.
Use of local anesthetic	Contraindicated.	Contraindicated.	Not needed.
Obtain sample	Tissue sample (at least 0.25 g) removed aseptically with scalpel or punch biopsy. No local anesthetic.	Using a 10-mL syringe with #22 safety needle, insert into tissue and aspirate, moving the needle tip at 2–3 angles for directions to obtain 0.5 mL of fluid.	Rotate sterile culture tip in 1 cm^2 area for 5 s until wound fluid is elicited.
Specimen care	Insert into sterile container, insuring appropriate approach for aerobic/ anaerobic analyses.	Remove excess air, remove needle with hemostat and after disposing of it in sharps container, cap syringe with sterile blood gas cap.	Insert culture tip into sterile container, insuring that portion in the container is sterile. Insure media is appropriate for aerobic/ anaerobic analyses.
Transport	Transport immediately. Delay in transport may lead to inaccurate data as organisms can grow in the culture container.		

Regardless of the culture technique employed, preparation of the lab

slip that accompanies the specimen is critical to accurate interpretation of the culture findings. The lab needs to know the site on the body where the specimen was obtained, the type of wound, the time the specimen was obtained, the age and gender of the patient, whether receiving antibiotics (name, dose, started when) as well as any special information, for example, occupation (farmer vs. ocean diver), anticoagulated. Prompt delivery of the specimen also is essential so organisms do not have time to grow. Failure to complete these seemingly mundane aspects of wound culture can result in false-positive or false-negative results.[43]

Practice Point

Accurate wound culture data depend on aseptic collection of the specimen, essential data about the wound and patient that are incorporated into the lab slip, and timely transport of the specimen to the lab.

Specimens can be processed qualitatively, quantitatively, or semiquantitatively. Qualitative processing, results in isolation and identification of the organisms present. The quantitative processing allows the specific number of organisms grown in media to be identified and counted. The number of organisms is expressed as number per gram of tissue, milliliter of fluid, or swab. The number of organisms present provides information regarding the rate of microorganism multiplication. Few facilities are equipped to perform quantitative analyses, and most are restricted to research studies. The majority of cultures are processed using semiquantitative technique where the cultures are inoculated onto media using a sterile loop that dilutes the specimen from the first to the fourth quadrant. Results are reported as 1+, 2+, 3+, and 4+ (trace, few, moderate, abundant).[41,43] With semiqualitative analyses, authors[10,44] suggest that 4+ should be used as the cutoff for diagnosing infection. The sensitivity of the organisms isolated to antibiotics can be obtained, regardless of which processing technique is used. Normally, results from a culture are available in 24 to 48 hours. Also, analyses for anaerobic organisms and fungi are normally not performed unless requested.

Each of these wound culturing techniques has strengths and limitations. Tissue biopsy is the gold standard and so is the standard against which the

other approaches are compared. However, it is a labor-intensive process where the tissue must be weighed, homogenized, and serially diluted prior to inoculating media, and then plating it and growing it in aerobic and anaerobic conditions.[43] Biopsy also is limited because it causes patients pain when performed, many facilities do not have the resources to perform the laboratory analysis, and it requires that a MD or specially educated provider with appropriate education perform it.[41,43]

Few studies have been performed that address aspiration wound culturing. A study by Lee et al.[45] examined aspiration but did not compare it with biopsy. Another found that aspiration underestimated infection identified by biopsy but that the organisms obtained were not consistent with the organisms identified with deep tissue biopsy.[46] Currently, there are insufficient data about the use of aspiration wound cultures to recommend it for culturing wounds, although it is appropriate for draining abscesses.

Swab cultures have been extensively studied in an effort to identify a less invasive but accurate method of culturing wounds. Early work by Levine et al.[42] showed a high concordance ($R^2 = 0.76$) between their specific method of obtaining a swab culture (1 cm^2 area, 5-second culture, obtain bloody fluid) and biopsies of the same sites (Fig. 7-3). Other approaches to surface cultures have been used, most recently the Z-stroke where the tissue is swabbed across the wound from edge to edge in a Z pattern. The Levine technique sensitivity was 90% and specificity 57%, while that from the Z technique at a sensitivity of 90%, specificity was 27%. The concordance of the Levine technique with tissue biopsy was identical (78%), and concordance with respect to recovering the specific organisms *Staphylococcus aureus*, *P. aeruginosa*, and beta-hemolytic *Streptococcus* was 96%, 96%, and 99%, respectively.[41]

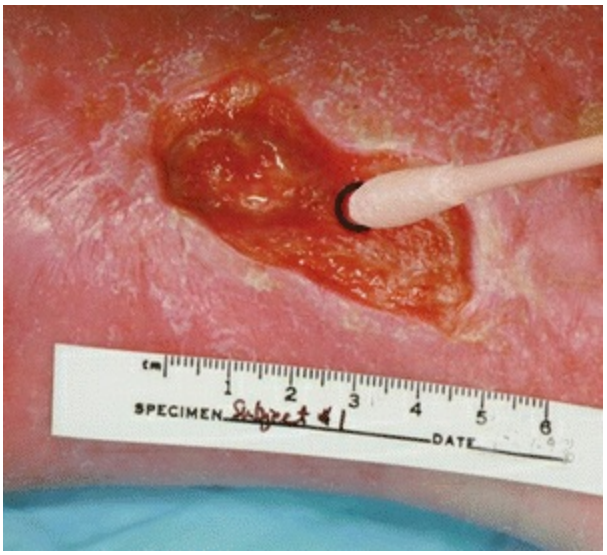


Figure 7-3. The Levine technique. (Photo courtesy of Dr. Rita Frantz.)

Swab culture has the advantage of being easy to use and require minimal training to ensure appropriate sampling. However, it has been criticized because it only accesses wound surface organisms as well as contaminants. It also does not sample organisms deep in the tissue, including anaerobic organisms.[47] An addition threat to accuracy is the possibility of dry swabs or swabs with insufficient specimen.[43] One approach proposed to overcome the concern of contaminants is wound cleansing, recommended to remove exudate, topical therapies, and loose nonviable tissue prior to obtaining the culture.[47,48] Moistening the swab with normal saline or transport medium is recommended prior to specimen collection to address the potential dryness issue.[46,48,49]

Although the accuracy of swab cultures as compared with biopsy cultures has been studied, methodological issues interfere with our ability to obtain little information as the basis for clinical practice.[40,42,46,50] The most serious methodological problem presented by these studies is that the specific swabbing techniques employed were not described. Swabbing techniques vary greatly according to wound preparation, area of the wound sampled, duration of sampling, and even the type of swab employed (e.g., alginate).[51]

To address this problem, Gardner et al.[41] compared culture findings from swab specimens obtained using wound exudate, the Z-stroke technique, and Levine's technique with culture findings from viable wound specimens. Culture findings based on swab specimens obtained using Levine's technique were more accurate and concordant with culture findings based

on tissue specimens than swabs taken with either wound exudate or the Z-stroke technique.

At a critical threshold of 37,000 organisms per swab, the Levine technique had a sensitivity of 90% and specificity of 57% when the true infection status of the wound was defined as 1,000,000 or more organisms per gram of tissue. The Z technique using the critical threshold of 62,500 organisms had a sensitivity of 90% but only a 27% specificity. In terms of recovering any and all organisms, swab specimens obtained using Levine's technique were 78% concordant with tissue specimens. Gardner et al.[41] as well as others[52] recommend the Levine technique. The researchers, however, note that further study is needed to identify the optimal critical threshold for practice.

Evidence-Based Practice

Swab cultures should be obtained using the Levine technique as it obtains fluid from the wound tissue, not just surface organisms.

A review by Reddy et al.[20] was conducted to determine the preferred noninvasive method of culturing chronic wounds. Few (four) studies met the inclusion criteria that included comparison of the techniques with tissue biopsy, and they calculated likelihood ratios (LR) to identify predictors of infection (positive LR) and predictors of no infection (negative LR). The Gardner study[41] that examined nonarterial chronic wound ulcers using the Levine technique (when compared with biopsy) had a likelihood ratio (LR) of 6.3 (95% CI 2.5 to 15), showing its strength in identifying infection. Similarly, a negative wound culture (LR 0.47 with 95% CI 0.31 to 0.73) indicated that infection was less likely. In contrast, the Z-technique (superficial surface culture of entire wound surface moving from side to side while rotating the swab between the thumb and forefinger) neither predicted nor excluded wound infection. In a later study by Gardner et al.[26] on patients with nonarterial diabetic ulcers, Levine technique did not predict infection, but negative cultures predicted the absence of infection. The third study[40] in patients with mixed chronic wounds, compared quantitative swab culture with the Levine technique with tissue biopsy, did not predict infection while the negative culture predicted the absence of

wound infection (LR 0.35; 95% CI 0.15 to 0.85). A serious limitation of the study is the lack of information about the exact number of organisms that were defined infection, that is, does greater than 10^5 actually mean 10^6? Other concerns were lack of information about who the participants were; age and gender of participants were not reported.

In many ways, this review summarizes the existing literature about noninvasive wound culture in patients with chronic wounds. First, the prevalence of chronic wounds is high, especially in spinal cord–injured patients, long-term care residents, and diabetics. Wound infection in the chronic wound populations is high, the range depending to some extent on the quality of the research.[20] Yet, even the high prevalence of infection in patients with chronic wounds may be an underestimation as patients with infected wounds often are excluded from many research studies. This review shows that the identification of wound infection remains challenging. Clinical signs and symptoms of infection are important in wound assessment. Inflammation as markers are especially important in acute wounds.

However, the signs and symptoms associated with inflammation may not be present in some patients with acute wounds or in patients with chronic wounds. The signs specific to secondary wounds continue to require ongoing attention. Wound pain and wound breakdown need to be routinely incorporated into assessment. Consideration also needs to be given to routine evaluation of a set of signs and symptoms of infection in chronic wounds such as is provided by the Clinical Symptom Assessment List[13] and the Sibbald et al.[10] NERDS and STONEES approach. The use of criteria that have established operational definitions will aid providers in evaluating chronic wounds consistently.

Wounds suspected of infection, especially those with delayed healing, are often cultured to confirm the diagnosis. While qualitative cultures provide useful information in wounds that are demonstrating obvious clinical signs of infection, they may not be as useful in diagnosing infection in the absence of signs and symptoms unless certain pathogens are isolated. In the absence of clinical signs and symptoms, quantitative cultures remain the gold standard method for diagnosing localized wound infection. Newer technique on the horizon hold hope for a revolution in point of care diagnostics for wound infection.

Newer Technique to Identify Bacteria in

Wounds

Advanced techniques have been used in research to evaluate the presence of biofilm and the nature of organisms in it. Early on, microscopic approaches were used to identify the biofilm EPS. Initially light microscopy was used and later confocal scanning microscopy. At times, the microscopy was complemented with various staining techniques.

More recently, various molecular methods have been developed and utilized. Polymer chain reaction (PCR) identification of bacteria and fungi has been used to sequence the DNA in biofilm organisms. Sequencing specific regions of bacteria and fungi present in chronic wounds has been used but is expensive and not widely available to clinicians. The goal is to develop large databases to identify patterns of organisms that correlate with nonhealing.[14,53]

The recognition that most chronic wounds have high levels of proteases has led to the development of point of care detectors for use with wound fluid. While approved for use in some European countries, these sensors are not currently available in the United States.[53]

The goal of this diagnostic work is to identify specific organisms so focused care can be provided. At the moment, personalized care seems to be needed that addresses the specific organisms identified in a specific wound in a given patient.[14,53] Such an approach will require improved diagnostics and pharmacies able to prepare combination of drugs that can be applied in the immediate postdebridement period to kill biofilm organisms. Much research is focused on this area, and clinicians will need to continue to update their knowledge to provide evidence-based care.

Practice Point

A bedside test for identifying microorganisms in biofilm does not currently exist. Ongoing research continues to develop and test this needed diagnostic tool.

Treatment of Bioburden, Biofilm, and Infected Wounds

The goals of treatment for wounds with increased levels of bioburden are

to:

- maximize host resistance
- minimize bioburden
- prevent and disrupt biofilm
- eliminate organisms that have invaded tissues and caused infection

Major interventions are addressed to treat bioburden, biofilm, and infect. A model for holistic care that is essential to managing patients with varying levels of bioburden is provided by various authors.[3,10,54] They focus on the patient as a whole as well as therapies specific for the wound.

Maximize Host Resistance

Host factors are the most important factor in the prevention of infection.[3,10] A robust immune system that is able to respond to invading organisms is fundamental to prevention of infection and resisting organisms. When that is not present as is seen in severe immunosuppressed patients (e.g., bone marrow transplant patients), extra vigilance is needed to protect that patient from external contamination, for example, masks, hand washing, sterile gloves, etc.

Each patient is evaluated independently to determine that factors fundamental to the wound healing are present.[10,54] An adequate blood supply is required as infection occurs more often in ischemic tissues. Local edema needs to be minimized. Oxygen is needed for WBC function. Glucose levels need to be normalized as hyperglycemia inhibits the ability of WBCs to function. Nutritional intake needs to be maintained, so the patient is not catabolic. Factors specific to the type of wound need to be managed to maximize available tissue perfusion, for example, reduction of edema in venous ulcers, unloading tissues in DFUs, prevention of excess load in pressure ulcers. Failure to address these basic host factors may contribute to continued proliferation of microorganisms, despite initiation of bioburden/infection-specific treatment.

Practice Point

Care must be focused on placing the patient in the best physical and mental condition to healing.

Minimize Bioburden/Disrupt Biofilm

Bioburden in wounds can be reduced through cleansing, irrigation, debridement, negative pressure therapy, antiseptics, and antibiotics. Cleansing, irrigation, debridement, and negative pressure therapy seek to dislodge organisms from the wound surface/wound tissue. Antiseptics and antibiotics are used to kill or inactive microorganisms.

Cleansing

Cleansing of wounds is performed to reduce planktonic bacteria, foreign debris, contaminants, and inflammatory exudates present on the wound surface that may create tissue damage. Wound cleaning is a process that removes these less adherent inflammatory contaminants from the wound surface and renders the wound less conducive to microbial growth. On the other hand, cleansing itself may create tissue damage, depending on the pressure used for cleansing and the cleansing agent. Host factors such as foreign debris and contaminants on the surface of the wound can harbor microorganisms or provide nutrients for their growth. Cleansing with each dressing change is recommended.[3,55]

A Cochrane meta-analysis of patients with either acute or chronic wounds examined the effect of cleansing of the wound surface on infection and healing. They compared normal saline versus tap water and tap water versus no cleansing.[56] Data showed no difference in infection rates with normal saline versus tap water; no difference with cleansing with tap water versus no cleansing. Healing rates also did not differ. The authors noted the lack of consistent definition for infection was important in this meta-analysis. They also emphasized that tap water had to be safe when used. Theoretically, water should not be used because of its deleterious effects on open tissue; however, because the fluid is only briefly in contact with the wound surface, it isn't crucial that the solution be isotonic (0.9% sodium chloride). If an isotonic saline solution is used, however, it can be prepared inexpensively by combining one teaspoon of noniodized salt in one quart of water.

Patient TeachingPreparing Saline Solution at Home

To prepare saline solution at home, tell the patient to combine 1 teaspoon of noniodized salt with 1 quart of distilled water, stirring until the salt is completely dissolved. The solution can be stored for up to 1 week, at room temperature, in a tightly covered glass or plastic container.

When open wounds are exudative or full of debris, cleaning with a commercially available wound cleanser should be considered. Many of these products have a surfactant in them to help reduce surface tension and aid in the removal of contaminants that have the potential to cause local tissue damage and inhibit healing. With that said, manufacturers of commercially available wound cleansers are challenged to create a product that has the cytotoxicity level needed to kill microorganisms but without damaging cells needed for normal healing. Studies have examined the toxicity of various wound cleaner and skin cleaners on various cells using a toxicity index, a measure of the product's ability to kill microorganisms without damaging cells needed for repair. To date, an optimal toxicity level has not been proposed. Table 7-4 presents published data from one classic study and two newer studies on the toxicity index of several wound cleaners. Data show that toxicity is variable by product and type of cell tested. Also skin cleaners (data not shown) have a higher toxicity index than skin cleansers[57–59] and are harmful if used to cleanse wounds. Data in Table 7-4 provides data by product to allow facilities and clinician to made decisions about which products best need the needs of the patients seen at their institution.

Table 7-4 Comparison of the Toxicity Index of Wound Cleansers in Three Studies

	Foresman et al.[57]	Wilson et al.[58]		Rani et al.[59]
Cells used as basis for toxicity index	WBCs	Fibroblasts	Epidermal keratinocytes	Fibroblasts
Shur-Clens	10	0	0	0
Biolex	100	10	0	10
SAF-Clens	100	0	10	100
Cara-Klenz	100	10	10	10
Dermal wound cleanser	10,000	10	100	10
Puri-Clens	10,000	10	100	X
Restore	X	100	100	0

X, not tested.

A subsequent review[60] examined literature on 10 generic categories of cleansers. They explored whether each product was effective (vs. not effective), toxic (vs. low toxicity), and improved wound healing (vs. not). Their data are illustrated in Figure 7-4.

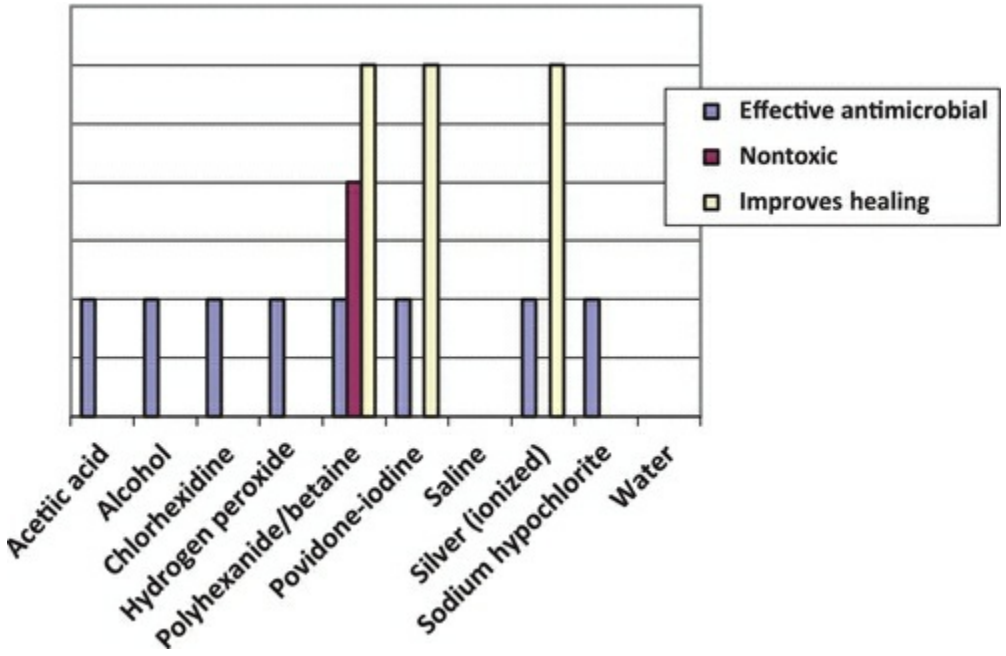


Figure 7-4. Ten generic cleansers: their antimicrobial effectiveness, nontoxicity, and improvement in healing. (Reprinted with permission from Wilkins, R.G., Unverdorben, M. "Wound Cleaning and Wound Healing: A Concise Review," *Advances in Skin & Wound Care* 26(4):160-63, April 2013. Copyright © 2013, Lippincott, Williams and Wilkins.)

Data from this review[60] showed that saline and water do not kill organisms nor do they enhance healing. All of the other cleansers were effective against common contaminants, although their effectiveness in supporting healing is uncertain due to the irregular quality of the available studies. Only polyhexanide–betaine had low toxicity, was effective against microorganisms, and improved healing. Povidone–iodine and silver (ionized) were more toxic but still improved wound healing. A paucity of good quality research limits the positive conclusions that can be reached from this review except for polyhexanide–betaine and possibly povidone–iodine and silver.

Practice Point

Cleansing with each dressing change is recommended. The best agent to use for cleansing has not been established.

Irrigation

Irrigation is utilized as a strategy to remove planktonic phenotype organisms before they become attached to the EPS. Wound irrigation creates hydraulic forces generated by the fluid stream that are effective in cleaning the wound when the force of the irrigation stream is greater than the adhesion forces that hold the organisms and debris to the wound surface. An ideal pressure for wound cleansing is 5 to 15 psi.[61–63]

A pressure less than 5 psi is ineffective in cleansing. Research on experimental wounds in the animal model found that irrigation pressures of 1 and 5 psi removed significantly less wound debris than did pressures of 10 and 15 psi.[64] A bulb syringe (0.05 psi) used to treat trauma wounds within 24 hours of injury was less effective than pressures of 13 psi created with a needle and a syringe.[65] Significantly more inflammation and wound infection was seen in those wounds cleaned with the bulb syringe.

Evidence-Based Practice

Research indicates that the optimum pressure for wound cleansing is between 5 and 15 psi.

The use of a needle and syringe to deliver fluid to wound tissue is generally regarded as a convenient method of providing effective irrigation pressure. A 35-mL syringe and a 19-gauge needle or angiocatheter has been shown to deliver an irrigation stream at 8 psi[66] and was more effective than a bulb syringe in removing wound bacteria and preventing the development of infection in experimental wounds.[65]

A variety of needle and syringe combinations can be used to achieve the desired range of irrigation pressure. The size of the syringe and the needle gauge determine the amount of pressure of the fluid stream. Since the force depressing the plunger is distributed over a larger surface area, the larger the syringe, the less the force. With a 19-gauge needle, 6-, 12-, and 35-mL syringes will produce pressures of 30, 20, and 8 psi, respectively. The

opposite effect occurs by increasing the size of the needle. Since the larger the lumen of the needle, the greater will be the flow of fluid, needles of 25-, 21-, and 19-gauge will create pressures of 4, 6, and 8 psi, respectively, when used with a 35-mL syringe.[67]

It is tempting to use higher fluid stream pressures for irrigation as they enhance removal of bacteria and debris from the wound.[64,68] Studies of mechanical irrigation devices used on crushing trauma wounds confirmed that cleaning with a pressure of 70 psi produces significantly more effective removal of bacteria and debris than cleaning with 25 or 50 psi.[69–71] Yet lower pressures are generally desirable in chronic wounds because of the increased risk of dispersing fluid into adjacent wound tissue or along tissue planes with higher pressures.[61–63]

Practice Point

Irrigation at 5 to 15 psi is recommended for heavily exudative wounds. Wound irrigation can be accomplished with a variety of medical tools and specially made devices. A 19-gauge Angiocath and a 35-mL syringe is an inexpensive and easy to use approach to irrigation.

The wound irrigation fluid stream can be delivered in either a pulsatile or continuous flow pattern. The benefit of delivering wound irrigation with a pulsatile as compared with a continuous fluid stream hasn't been substantiated in experimental studies.[68,70,71] Although several commercially available battery-powered, disposable irrigation systems (Davol, Inc., Cranston, RI; Stryker Instruments, Kalamazo, MI; Zimmer, Inc., Dover, OH) deliver pulsatile fluid streams with different spray patterns and remove the fluid and wound debris with suction, their wound healing efficacy in comparison with other irrigation methods remains to be established.[72,73] In a randomized clinical trial, pulsed lavage was compared with conventional debridement and betadine dressing. Findings showed the pulse lavage resulted in smaller size, less bioburden, and faster healing.[74] Lack of detail as to the nature of "conventional debridement" limits the validity. At the present time, their primary benefit appears to be their portability and capability to serve as an alternative to whirlpool

therapy for patients with chronic wounds, which aren't amenable to whirlpool, or for patients when whirlpool therapy isn't accessible.

An alternate approach to wound irrigation is the whirlpool bath. It cleans the wound by exposing the entire wound bed and surrounding skin to agitating water generated by jets in the sides of the whirlpool tub. Only two studies have investigated the cleaning effectiveness of whirlpool, and these are methodologically confounded with wound irrigation, which was provided at the end of the whirlpool therapy.[75,76] The whirlpool is best suited for use with chronic wounds containing thick slough or necrotic tissue. Caution must also be taken to ensure that the wound doesn't come in close contact with the water jets, since the high pressure they generate could cause further tissue injury.

A more recently developed technology that assists with removing fluids from a wound is negative-pressure wound therapy (NPWT). Fluid removal is accomplished by placing foam or gauze dressing into the wound, sealing it with a semiocclusive covering, and applying subatmospheric pressure through an evacuation tube coupled to a computer-modulated pump. The suction action created by the subatmospheric pressure facilitates removal of stagnant fluid from a wound. A recent systematic review examined seven small randomized clinical trials and heterogeneous studies that examined the relationship between NPWT use, bacterial load, and type of bacteria present.[77] Theoretically, NPWT should remove organisms to reduce bioburden due to the negative pressure. Because of the small number of studies and methodological limitations, the authors suggest that no definitive conclusion about the effect of NPWT use on the number and type of organisms when status prior to and after NPWT is compared. Although widely used, further work is needed to establish NPWT as effective in reducing bioburden.

Debridement

At the stage of critical colonization/biofilm, either sharp/aggressive debridement or ultrasonic debridement is needed. The goal of debridement in this situation is to remove all devitalized tissue as it provides an excellent medium for growth of microorganisms. In addition, it is critical to remove existing biofilm and with biofilm genotype organisms. Sharp debridement is needed to open tunnels and to address undermined areas, should they exist.[11] With the viable tissue exposed and the biofilm disrupted, topical agents can be used between debridement to kill the

planktonic organisms before they become attached to the wound surface. Repeated debridement is recommended, at no more than 7-day intervals.

Practice Point

Sharp debridement repeated at weekly intervals is the first step in antibiofilm treatment.

Enzymatic debridement has been be used to remove devitalized tissue, but its value on biofilm has yet to be established.[3] Some authors recommend the use of biodebridement with maggots. While limited by the number of available studies, divergent control products, and varying outcomes, a recent systematic review by Sun and colleagues[78] concluded that the use of maggots increased wound healing and decreased time to healing.

An important issue in the recommendation for surgical/sharp or ultrasonic debridement is the availability of trained personnel to perform adequate debridement to interrupt the wound-bioburden cycle. The provider who performs the debridement must have the knowledge, skill, and authority to safely perform sharp debridement.[11] Referral of the patient to a wound center or outside consultant may be needed in some situations, for example, provider with limited experience with wounds, long-term care. Details on methods of wound debridement are addressed in chapter 8, Wound debridement.

Topical Agents

Topical antiseptics and antibiotics were out of favor for a period of time because of their toxic effect on cells essential to healing.[79–81] With appreciation of the large population of patients with chronic wounds, the role of biofilm in delayed healing, and the growing number of antibiotic resistant organisms, it became apparent that treatment perspectives needed to change and they have.

Topical agents are currently viewed as essential treatment of critical colonization/biofilm as well as wound infection.[3,14,82] In fact, antibiofilm therapy has been identified as a treatment category for chronic wounds.[83–85] There are many biofilm treatments, but all of the approaches

begin with removal of the existing biofilm with debridement. Topical agents then are used to prevent the planktonic organisms that are released with debridement from reattaching and to suppress biofilm reformation.

One developing treatment principle is to change products when the wound gets stalled.[83] Such an approach requires meticulous record keeping, ongoing evaluation of events other than the topical agent that might contribute to the nonhealing, for example, brief flu, poor glycemic control, trauma to the wound site.

Another principle is to use a bundle of treatments. Wolcott and Rhodes[85] evaluated the frequency of complete healing in patients with chronic wounds who had critical limb ischemia. Patients were individually managed to address systematic threats to healing. Wound treatment was guided by the physical appearance of the wound, and care was individualized to include the entire patient. Treatment was focused on specific goals and included block biofilm attachment (lactoferrin, EDTA), kill bacteria (antibiotics, silver, iodine), inhibit quorum sensing (farnesol), interfere with EPS (farnesol, xylitol), and address false metabolites (gallium, xylitol). In this retrospective study, 77% healed, a much higher rate than other studies. The authors conclude that treating biofilm with multiple treatments simultaneously is needed. However, there is a need for prospective research to confirm these findings.

The lack of valid indicators of chronic wound infection has complicated the initiation of topical therapy. Given the current state of ambiguity regarding valid clinical signs and symptoms of chronic wound infection, the decision to initiate antimicrobial therapy is best guided by the failure of a wound to make progress toward healing or local infection.[10,54,84] although some authors recommend careful examination for secondary signs of infection.[85,86]

The ideal antiseptic has low toxicity, is effective in killing microbes, but does not disrupt wound healing. Antiseptics are not site specific and so have a broader impact, affecting both microorganisms and host cells needed for healing and fighting infection.[3,55] An additional consideration is whether the product is effective on biofilm.[14,82,84,87] Box 7-2 lists antiseptics recommended to treat biofilm.

Box 7-2 Antiseptics Used to Control Bioburden and Biofilm

Acetic acid[3,82]

Chlorhexidine[3,82]

EDTA[3,85]

Faresl[3,85]

Honey[3,14,82,85]

Iodine[3,14,82,85]

Lactoferrin[3,85]

Methylene blue–gentian violet[3,14]

Mupirocin[3,14]

Polyhexamethyl biguanide (PHMB)[3,82]

Potassium permanganate[3,82]

Silver[3,14,82,85]

Xytol[3,85]

Overall, topical antimicrobials are formulated from elements such as silver, copper, gold, or zinc. The specific formulation of the antimicrobial agent is crucial to its effectiveness in reducing bacterial burden without destroying cells essential to healing. Iodine is an example of an agent that, in its elemental form, is toxic to cells that promote healing. However, when formulated as *cadexomer iodine* gel, it slowly releases iodine from its microspheres while absorbing bacteria.[88,89] An in vitro study of varying concentrations of cadexomer iodine demonstrated that it is nontoxic to human fibroblasts in culture in concentrations of up to 0.45%; the study also showed that chronic wounds treated with cadexomer iodine revealed reepithelialization on biopsy.

Topical agents formulated with silver have long been a part of the antibacterial armamentarium. Using the ionized form of silver (Ag+), these agents exert bacteriostatic properties through the action of the silver cation on proteins. The Ag+ binds to proteins in the bacterial cell wall, disrupting its integrity and resulting in death of the cell. Silver-containing wound care products include pure silver, creams, and sustained-release dressings. Research evidence regarding the efficacy of these agents is most substantial for the cream formulations (silver sulfadiazine 2% or 7%). Topical cream formulations have been shown to reduce bacterial density, vascular

margination, and migration of inflammatory cells in chronic leg ulcers.[90] The efficacy of silver sulfadiazine cream in reducing bacterial burden in chronic wounds was substantiated in a randomized trial of 45 patients with a single infected pressure ulcer who were randomly assigned to receive silver sulfadiazine cream, povidone–iodine solution, or saline gauze dressings.[91] Standard care consisting of debridement, pressure reduction, and nutritional support was provided to all subjects. All ulcers (100%) were treated with silver sulfadiazine cream; the bacterial levels were reduced to 10^5 or less per gram of tissue during the 3-week study period, while only 78.6% of the povidone–iodine solution treated ulcers, and 63.6% of the saline gauze–treated ulcers achieved these reductions in bacterial levels. Overall, ulcers treated with silver sulfadiazine responded more rapidly, with one-third achieving bacterial levels of less than 10^5 within 3 days and half of the ulcers reaching this level within 1 week. These data support treatment with silver and limiting silver-based creams to no more than 2 weeks. Limiting the use of effective topical agents to short durations with high doses can minimize the potential for selection or development of more resistant bacteria. If clinical evidence of improvement has not occurred with 2 weeks of treatment, other host factors that may be contributing to decreased bacterial resistance should be explored.

Practice Point

Topical antimicrobial therapy is a critical second step in antibiofilm treatment.

Silver and other biofilm treatments also can be delivered with a silver-impregnated dressing.[92] However, many questions remain regarding the dosage of silver released into the wound and the potential for delays in healing if the silver cation binds to fibroblasts and epithelial cells. Reports of industry-conducted in vitro studies demonstrate that different silver-containing dressings release different amounts of silver over time.[93] There are preliminary indications that the toxic dosage of silver differs depending on whether the fibroblasts and epithelial cells are in a monolayer or in a three-dimensional matrix.[94] The potential adverse consequences in epithelializing wounds were demonstrated in a controlled study of matched

pair skin graft donor sites treated with a nonantimicrobial foam dressing or a nanocrystalline silver dressing. Although there were no differences in bacterial counts between the two treatment groups, reepithelialization was significantly slower in the wounds treated with the silver dressing.[95] At present, in vivo evidence to establish the safety and efficacy of silver-containing dressings is lacking. Recent reviews of the research literature confirm that clinical evidence of benefit for silver-containing dressings is lacking.[96–98] An evaluation of multiple elements of dressing performance, including silver content, rate of silver release, and antibacterial activity, concluded that the dressing should be selected based on standard clinical parameters rather than on a dressing's silver content or release activity.[99] Therefore, discretion should be exercised in using them to reduce microorganisms on wound surfaces. The treatment periods should be limited to 4 weeks duration and discontinued when wound reepithelialization is observed.

Although clinical studies provide evidence supporting the utility of topical antibiotics in reducing bacterial burden in chronic wounds, these agents can cause adverse reactions in some patients. Reports of permanent hearing loss with topical 1% neomycin solution and acute anaphylactic reactions with topically applied bacitracin suggest that careful monitoring is indicated when using these agents.[100,101] Additionally, since there's a risk of selecting out resistant strains of bacteria, antibiotics that are used to treat infections systemically shouldn't be used in a topical form on chronic wounds. Furthermore, since topical antibiotics are limited in the range of species they are effective against, it is important to judiciously select the specific antibiotic based on the organism present in the wound.

In contrast to topical antiseptics, topical antibiotics exert their antimicrobial effects through selective binding to chemical targets on the bacterial cell wall.[3] Since human cell membranes lack these chemical targets, topical antibiotics have negligible effect on cells that promote wound healing. Antibiotics were developed to treat planktonic organisms, and the concern in their use is that organisms may develop resistance. Topical antibiotics result in side effects to host cells and, while infrequent, they may have serious consequences. Antibiotics are effective only when organisms are metabolically active, so when biofilm organisms become dormant, antibiotics are ineffective. Also topical antibiotics are inactivated by debris in wounds and so are effective for short periods of time.[3]

Systemic Antibiotics

While topical antibiotics have demonstrated effectiveness in reducing bacterial burden when the area of involvement is localized, they're generally regarded as inadequate to control more extensive tissue involvement, such as advancing cellulitis. In these instances, systemic antibiotic therapy is indicated. Since the type of organisms and degree of invasiveness will vary, the choice of antimicrobial therapy will need to be individualized. Unfortunately, little research evidence exists to guide selection of antibiotics to treat chronic wound infections. Generally, chronic wound infections are treated empirically with antibiotics that have a narrow but sufficient spectrum of coverage. Care should be taken to avoid routine or extended periods of treatment. Less acute forms of chronic wound infection are treated with oral antibiotics. However, parenteral therapy may be indicated when the infection involves deeper tissue and is accompanied by systemic signs, such as fever, chills, and elevated WBC count. Regardless of the route, the effectiveness of any systemic antibiotic in reducing bacterial burden will be dependent on the adequacy of the patient's peripheral circulation. In those instances where peripheral vascular disease compromises blood flow to the infected tissue, systemic antimicrobial therapy may produce no clinical improvement in the wound.

Practice Point

The effectiveness of systemic antibiotics is dependent on an adequate blood supply to the wound.

Adjunctive Therapies

Several adjuvant therapies have shown potential as interventions for reducing bacterial burden in chronic wounds. Hyperbaric oxygen therapy, administered by intermittent inhalation of pure oxygen at a pressure greater than 1 atmosphere, has been shown to promote PMN microbicidal efficacy in DFUs.[102] Ultraviolet (UV) light has long been recognized for its bactericidal effects derived from in vitro studies. The effect of UV light has been demonstrated in a clinical study of 22 patients with chronic wounds containing high bacteria levels as determined by quantitative swab culture.[103] Following one 180-second UV-C treatment session, cultures

showed a statistically significant reduction in predominant bacteria ($P = 0.001$) and significant reductions in MRSA ($P < 0.05$) and *S. aureus* ($P < 0.01$).[104]

Additionally, exposure of wound tissue to electrical current using an electrical stimulation device has been shown in animal studies to exert bacteriostatic and bactericidal effects on microorganisms known to infect chronic wounds. In a study of 20 patients with burn wounds that had been unresponsive to conventional therapy for 3 months to 2 years, application of direct current stimulation for 10 minutes twice weekly produced a quantitatively lower level of microorganisms.[104] However, the antibacterial effect of pulsed current, the more commonly used electrical stimulation modality in current practice, remains unclear. There is evidence that the voltage that would be required to produce an antibacterial effect would create profound muscle contractions, making it not applicable in clinical practice.[105–107]

Summary

Most of our understanding of wound infection has been derived from the study of acute wounds. As wound healing science has evolved, it has become clear that chronic wounds are distinctly different environments where host resistance has been overwhelmed by bacterial burden. The classic signs and symptoms of infection are well recognized. However, they're based on assessments made of acute wounds and aren't valid in the chronic wound. While indicators of chronic wound infection remain ambiguous, substantial evidence shows that debridement of necrotic tissue and biofilm in the wound bed is an essential first step to reducing bacterial burden. Regular wound cleaning with a noncytotoxic solution, using sufficient force to remove surface contaminants and debris while minimizing trauma, is an important adjunct to reduce surface contaminants. In those instances where these measures aren't sufficient to restore a balance between host resistance and bacterial burden, antiseptics and antibiotics that act directly on the bacteria are indicated.

PATIENT SCENARIO

Clinical Data

Mrs. H is a 47-year-old well-developed, well-nourished African American female who presents today with a nonhealing malodorous wound on the lateral aspect of the gaiter area of her left leg. She has been managed by her primary care physician who refers her today for evaluation and treatment.

Mrs. H reports that the wound was the result of being kicked in the leg by her horse and that she nursed it at home for a month. When the wound pain and edema did not resolve, she sought care from her physician. She has been treated with compression for 6 months and is compliant with treatment.

Clinical examination of the wound show the wound is 4 × 4.5 × 0.6 cm. About half is covered with yellow slough and heavy exudate. The ulcer is malodorous and painful. No edema is appreciated. The area is not warm, and there are no satellite wounds. According to the record from the referring physician, the ulcer has not increased in size and her ABI is 0.9. Her history is negative for diabetes, pulmonary disease, and cardiac disease. She takes no prescription medications, only a daily multivitamin.

Case Discussion

Analysis of the history, the signs and symptoms, and NERDS criteria show Mrs. H's wound is experiencing critical colonization/biofilm. Having confirmed the presence of adequate circulation and a normal nutritional state, the wound was sharply debrided and cadexomer iodine applied topically, covered with a foam dressing. Local treatment prescribed included daily cleansing with normal saline using a 35-mL syringe and 19-gauge angiocath followed by topical application of the cadexomer covered with a foam dressing. The patient was seen in a week, and the odor and exudate had abated. She again underwent sharp debridement, and an ionized silver dressing was applied with the plan to cleans the wound and change the dressing every 3 days. When seen in clinic 3 weeks after her initial visit, the wound bed was granulating, and the wound size had decreased.

Show What You Know

1. **How can you tell when a wound is infected, rather than in the**

critically colonized/biofilm phase?

A. An infected wound will increase in size, while one that is critically colonized/biofilm will have delay in healing.

B. An infected wound will have pus present, while a critically colonized/biofilm wound will not.

C. An infected wound will always have necrotic tissue; a colonized wound won't.

D. An infected wound will be malodorous, while a critically colonized biofilm wound will not.

2. Which of the following most accurately indicates wound infection?

A. A tissue biopsy showing large amounts of *Staphylococcus aureus*.

B. A quantitative culture of tissue showing 1,000 organisms/g of tissue.

C. A semiquantitative culture showing 4+ organisms present.

D. A tissue biopsy with less than 10^5 CFU organisms present.

3. Antiseptics are used for chronic nonhealing wounds because they

A. target specific intracellular sites.

B. have a broad mechanism of action.

C. never result in organisms developing resistance to them.

D. are not harmful to fibroblasts and other healing cells.

4. Scientific data indicate that the most effective debridement of chronic wound is as follows:

A. Use of topical enzymes for up to 4 weeks.

B. Continuous use of autolytic debridement with agents such as hydrogels.

C. Weekly sharp debridement.

D. None of the above.

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Wound Debridement

8

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The contribution of Janet E. Cuddigan PhD, RN, CWCN, in previous additions is gratefully acknowledged.

Objectives

After completing this chapter, you'll be able to:

- assess the purpose of debriding a wound
- evaluate criteria for *not* debriding a necrotic wound
- describe types of debridement, including sharp/surgical, conservative surgical, mechanical, maggot, enzymatic, and autolytic
- compare the advantages and disadvantages of each type of debridement
- select the most appropriate method of debridement depending on patient preference, clinician expertise, and healthcare system resources.

Speeding the Healing Process

Debridement is an important component of the wound bed preparation (WBP) management model.[1–4] After considering the cause of the wound and patient-centered concerns, debridement is a necessary step in local wound care.[1,2,4] Debridement is the removal of necrotic tissue, exudate, bacteria, and metabolic waste from a wound in order to improve or facilitate the healing process.[3,4] Accumulation of necrotic tissue usually results from poor blood supply, a prolonged inflammatory process, bacterial

damage, or an untreated cause of the wound (e.g., uncontrolled edema leading to increased interstitial pressure or other mechanical, chemical, or traumatic injury). In otherwise healthy people, natural debridement often keeps pace with the accumulation of dying tissue in a wound. If the host resistance is impaired by poor nutrition, continued pressure damage, or other comorbidities such as uncontrolled high blood sugar in a person with diabetes, medical intervention is required to facilitate wound healing.

The primary purpose of debridement is to reduce or remove dead and necrotic tissue in a healable wound because this tissue is a proinflammatory stimulus and a culture medium for bacterial growth.[1,3,4] The removal of dead and necrotic tissue is necessary to reduce the biological burden of the wound in order to control critical colonization on the wound surface and prevent deep and surrounding wound infection, especially in deteriorating wounds.[4] Debridement allows the practitioner to visualize the sides and base of a wound more accurately to determine whether viable tissue is present. (Keep in mind that a pressure ulcer that is covered with necrotic tissue cannot be numerically staged until debridement is completed.). Necrotic tissue that is not removed not only impedes wound healing but also can result in spread of bacterial damage to deeper tissue, causing surrounding cellulitis, osteomyelitis, and the possibility of septicemia, preventable limb amputation, or death. By removing necrotic tissue, debridement creates an acute wound within a chronic wound, restoring nutrient supply from the underlying circulation and facilitating optimal available oxygen delivery to the wound site.[4,5]

For a wound to heal, it must have a microenvironment free from the nonviable tissue that serves as a bacterial culture medium to increase bacterial metabolism and proliferation.[6] Oxygen is a primary requirement for this energy-dependent metabolic process to occur. The production of free radicals crucial to wound healing facilitates this process by killing bacteria and promoting the proliferation of both fibroblasts and epithelial cells. However, bacteria that are present in hypoxic conditions (anaerobes, facultative aerobes, and bacteria in biofilms) compete with healing tissue for nutrients and produce exotoxins and endotoxins that damage newly generated, mature cells. This setting of hypoxia and bacteria interrupts the fundamental wound healing process of fibroblast migration into the extracellular matrix. Fibroblasts excrete procollagen fibers that are assembled in the extracellular space with the aid of vitamin C into a collagen matrix that forms the building blocks of granulation tissue. This process allows a chronic wound stuck in the destructive abnormally

prolonged inflammatory stage to move to the proliferative stage, promoting new tissue formation and laying the foundation for healing with the necessary recruitment of fibroblasts to deposit collagen.[6]

Leukocytes—primarily polymorphonuclear cells—are the primary cells of the acute inflammatory process of wound healing. They enter the wound and aid in the removal of devitalized tissue and foreign material. Collaboration of local enzymes (metalloproteases, elastases, or collagenases) also helps to dissolve and remove devitalized tissue. Because collagen comprises approximately 75% of the skin's dry weight, the overall endogenous collagenase is considered to be one of the main regulators of tissue remodeling in the process of wound healing. Remodeling is part of the healing process in which the wound restructures into its final functional image.

Tissue injury is followed by a clotting stage and then the inflammatory stage to remove tissue debris. After wound debris is removed, macrophages are recruited; they, in turn, recruit fibroblasts, which deposit collagen and fill the wound space with scar tissue. An acute wound of reasonable size with adequate blood supply for healing and essential nutrients generally results in a "healed" closed wound within 14 days—but this doesn't represent the total healing process. Remodeling, or maturation, typically takes another 4 weeks, making the total healing process about 6 weeks, but the further increase in tensile strength can take a year or even longer. Other factors may be involved in the healing process as well. For example, a wound that appears in an area of rich blood supply (e.g., scalp) will heal faster than a wound in an area with a lesser blood supply (e.g., sacrum). Abnormal collagen breakdown and new collagen buildup occur in a balanced growth pattern resulting in a normal-appearing scar. However, excess collagen can form a hypertrophic scar or keloid. A hypertrophic scar represents disordered collagen within the scar tissue margins of a healed wound. A keloid extends beyond the wound scar margin with a greater disorganization of collagen fibers. (Chapter 9, Table 9-8 and Figure 9-29.)

Identifying Necrotic Tissue

Dead or necrotic tissue may be loose and moist or dry and firm. This tissue is identified by its soft loose moist, yellow, green, or gray appearance and may become hard thick and leathery with a dry black eschar (Fig. 8-1). Oxygen and nutrients can't penetrate the portion of a wound composed of necrotic tissue. Dead tissue is the breeding ground for bacteria, and the eschar may mask an underlying abscess.[7] Necrotic tissue that is soft, moist,

stringy, and yellow is referred to as slough (devitalized/avascular) tissue.[8–11] While some clinical experts have questioned whether slough is in fact tissue or an inflammatory by-product,[10] the National Pressure Ulcer Advisory Panel (NPUAP)/European Pressure Ulcer Advisory Panel (EPUAP) and Pan Pacific Pressure Injury Alliance (PPPIA) 2014 clinical practice guideline did define slough as "soft, moist, devitalized (non-viable) tissue. It may be white, yellow, tan, or green and it may be loose or firmly adherent" (p. 287)[11] (Fig. 8-2). Regardless of whether slough is or is not tissue, clinicians agree that it must be removed in acute and chronic wounds where the goal of care is healing.[4,8,9,11–13]

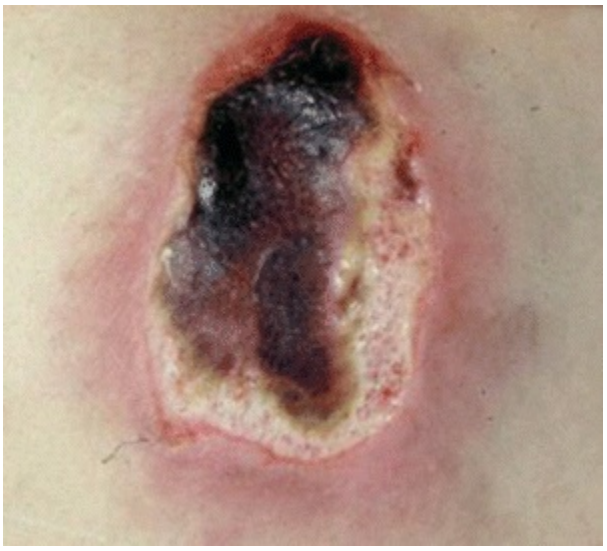


Figure 8-1. Eschar. In a wound that has become dehydrated, necrotic tissue turns thick, leathery, and black. This tissue is referred to as *eschar*.

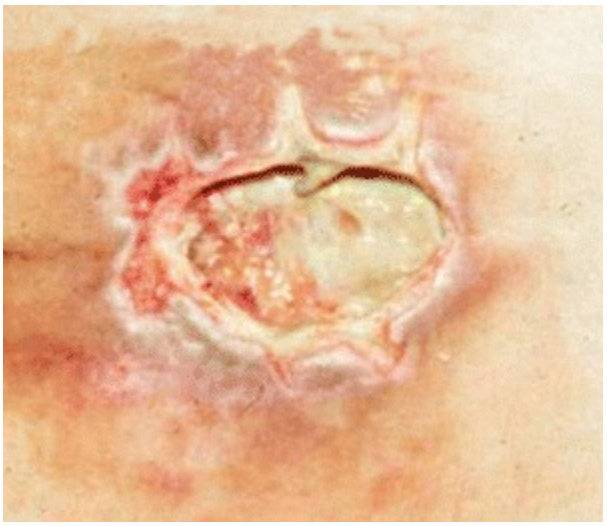


Figure 8-2. Slough. Necrotic tissue that's moist, stringy, and yellow (devitalized

tissue) is referred to as *slough*.

In general, removing necrotic tissue restores the local vascular supply to the wound and improves healing. However, debriding too much viable tissue can destroy the collagen structural framework for healing. Some wounds shouldn't be debrided at all. Exercise caution, for example, in dealing with necrotic ulcers in persons with compromised vascular supply or immunocompromised.[11] Debriding heel ulcers remains controversial because of problems with perfusion and the small amount of tissue that covers the calcaneus bone.[8,11] There is consensus that a vascular assessment needs to be performed to ascertain if there is an adequate vascular supply to heal prior to debridement of ulcers in the lower extremities.[1,8,11] Controversy revolves around whether to debride stable, adherent eschar with no exudate or signs of infection.[1,8,11] Some clinicians believe that although caution is indicated, all necrotic heels should be debrided.[9,12] The 2014 NPUAP/EPUAP/PPPIA pressure ulcer guideline state that stable, hard dry eschar should be assessed for signs of infection (erythema, tenderness, edema, purulence, fluctuance, crepitus, and/or malodor) and then debridement in the presence of adequate blood supply.[11] Wounds become larger with debridement, something that should be discussed with the patient/family.

Pyoderma gangrenosum is a neutrophilic disorder that has a raised translucent border that rapidly expands the central ulcer. This condition may be idiopathic or associated with underlying inflammatory bowel disease, rheumatoid arthritis, or myeloproliferative disease (leukemia/lymphomas). When the raised peripheral border is active, pyoderma gangrenosum is one example of a wound that should not be debrided[2]. A raised active border indicates an acute inflammatory reaction, and debridement would stimulate the infiltrate even more through a process called *pathergy*.[2] Septicemia is another condition that requires serious caution before initiating debridement. The wounds of patients with septicemia should not be debrided unless the patient is receiving adequate systemic antibacterial coverage.[2]

Practice Point

Monitor stable necrotic heels for odor and signs of edema, erythema, tenderness, fluid wave, purulence, crepitus, malodor, or drainage that may signal the need for debridement.[11]

Practice Point

It's often important to watch the line of demarcation between viable and necrotic tissue for signs of further tissue breakdown or softening eschar.

Chronic wound care begins with treating the cause and patient-centered concerns, including pain and activities of daily living. Assess individual patients to determine whether the wound is healable, maintenance, or nonhealable. To assess healability, an adequate blood supply needs to be present.[1,4,11] Palpable pulses in the foot indicate a pressure in excess of 80 mm Hg and enough blood supply for healing to occur. This finding could equate to an ankle-brachial pressure index of 0.6 or higher. If there is a sufficient blood supply and the cause has been corrected (compression for venous ulcers and pressure offloading for diabetic neurotrophic foot ulcers), the ulcer is healable. For pressure ulcers, the source of tissue damage needs to be corrected, including high blood pressure, poor nutrition, friction and shear, decreased mobility, or excess local moisture. Active debridement can be coupled with moisture and bacterial-balanced dressings.

A maintenance wound is one that has a sufficient blood supply for healing but is unable to heal due to patient or health delivery system factors.[4] Debridement and local wound care should then be conservative for maintenance wounds. This often includes the removal of nonviable slough but not an active debridement with removal of healthy tissue to create bleeding tissue (an acute wound in a chronic wound). A nonhealable or palliative wound does not have enough blood supply to heal; therefore, debridement should be conservative and limited to easy to remove soft slough with a local antimicrobial (such as povidone–iodine or chlorhexidine) and moisture reduction to reduce local bacteria.[4]

Although it is acknowledged that debridement is a critical first step in preparing the wound bed for healing, all of the recommendations on debridement from the international NPUAP/EPUAP/PPPIA 2014 clinical

practice guideline are at the C level of knowledge[11] (Table 8-1).

Table 8-1 Debridement Recommendations: NPUAP/EPUAP/PPPIA 2014 Pressure Ulcer Clinical Practice Guideline

1. Debride devitalized tissue within the wound bed or edge of pressure ulcers when appropriate to the individual's condition and consistent with overall goals of care. (Strength of Evidence = C; Strength of Recommendation 👍👍) CAUTION: Debridement should only be performed when there is adequate perfusion to the wound (refer to recommendation 11).
2. Debride the wound bed when the presence of biofilm is suspected or confirmed. (Strength of Evidence = C; Strength of Recommendation = 👍) **3.** Select the debridement method(s) most appropriate to the individual, the wound bed, and the clinical setting. (Strength of Evidence = C; Strength of Recommendation = 👍👍)
4. Use mechanical, autolytic, enzymatic, and/or biological l methods of debridement when there is no urgent clinical need for drainage or removal of devitalized tissue. (Strength of Evidence = C; Strength of Recommendation = 👍)
5. Surgical/sharp debridement is recommended in the presence of extensive necrosis, advancing cellulitis, crepitus, fluctuance, and/or sepsis secondary to ulcer-related infection. (Strength of Evidence = C; Strength of Recommendation = 👍)
6. Conservative sharp debridement and surgical/sharp debridement must be performed by specially trained, competent, qualified, and licensed healthcare professionals consistent with local legal and regulatory statutes. (Strength of Evidence = C; Strength of Recommendation = 👍👍)
7. Use sterile instruments for conservative sharp and surgically/sharp debridement. (Strength of Evidence = C; Strength of Recommendation = 👍👍)
8. Use conservative sharp debridement with caution in the presence of immune incompetence, compromised vascular supply, or lack of antibacterial coverage in systemic sepsis. (Strength of Evidence = C; Strength of Recommendation = 👍) CAUTION: Relative contraindications include anticoagulant therapy and bleeding disorders.
9. Refer individuals with category/stage III or IV pressure ulcers with undermining, tunneling/sinus tracts, and/or extensive necrotic tissue that cannot be easily removed by the other debridement methods for surgical evaluation as appropriate to the individual's condition and goals of care. (Strength of Evidence = C; Strength of Recommendation = 👍👍)
10. Manage pain associated with debridement. (Strength of Evidence = C; Strength of Recommendation = 👍👍)
11. Perform a through vascular assessment prior to debridement of lower extremity pressure ulcer to determine whether arterial status/supply is sufficient to support healing of the debrided wound. (Strength of Evidence = C; Strength of Recommendation = 👍👍)
12. Do not debride stable, hard dry eschar in ischemic limbs. (Strength of Evidence = C; Strength of Recommendation = 👍 1)
12.1 Assess stable, hard, dry eschar at each dressing change and as clinically indicated (Strength of Evidence = C; Strength of Recommendation = 👍👍). Assessment of an ulcer covered with dry, stable eschar should be performed at each dressing change and as clinically indicated to detect the first signs of any developing infection. Clinical indications that the dry, stable eschar requires assessment and intervention include signs of erythema, tenderness, edema, purulence, fluctuance, crepitus, and/or malodor (i.e., signs of infection) in the area around the dressing.
12.2 Consult a medical practitioner/vascular surgeon urgently in the presence of the above symptoms. (Strength of Evidence = C) (Strength of Recommendation = 👍)
12.3 Debride the pressure ulcer urgently in the presence of the above symptoms (i.e., erythema, tenderness, edema, purulence, fluctuance, crepitus, and/or malodor) (Strength of Evidence = C; Strength of Recommendation = 👍)
13. Perform maintenance debridement on a pressure ulcer until the wound bed is free of devitalized tissue and covered with granulation tissue. (Strength of Evidence = C; Strength of Recommendation = 👍) NOTE: NPUAP, EPUAP, PPPIA definition of Strength of Recommendation. "A consensus voting process (GRADE) involving all the experts formally engaged in the guideline development was used to assign a 'strength of recommendation' that indicates the confidence the health professional can have that the recommended practice will improve patient outcomes (i.e., do more good than harm). The overall aim of the 'strength of recommendation' is to help health professionals to prioritize interventions." (p. 3) Strength of Recommendation is noted by the symbols of thumbs in the guideline: 👍 = Probably Do It, weak recommendation for using an intervention 👍👍 = Do It, strong recommendation for using an intervention

Emily Haesler (ed)., National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel, and Pan Pacific Pressure Injury Alliance. *Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline*. ; Perth, Australia:

Cambridge Media, 2014:154-160.

WBP is the management of a wound to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic measures.[1,3,4,12] The original TIME model—**T**issue nonviable or deficient, **I**nfection or inflammation, **M**oisture imbalance, **E**pidermal margin nonadvancing or undermining[3]—has been reconceptualized to the **DIME**© model (Fig. 8-3).[1,4,12] As mentioned previously, you should first identify and treat the cause of the wound, address patient-centered concerns, and finally provide local wound care (Table 8-2).[4,12]

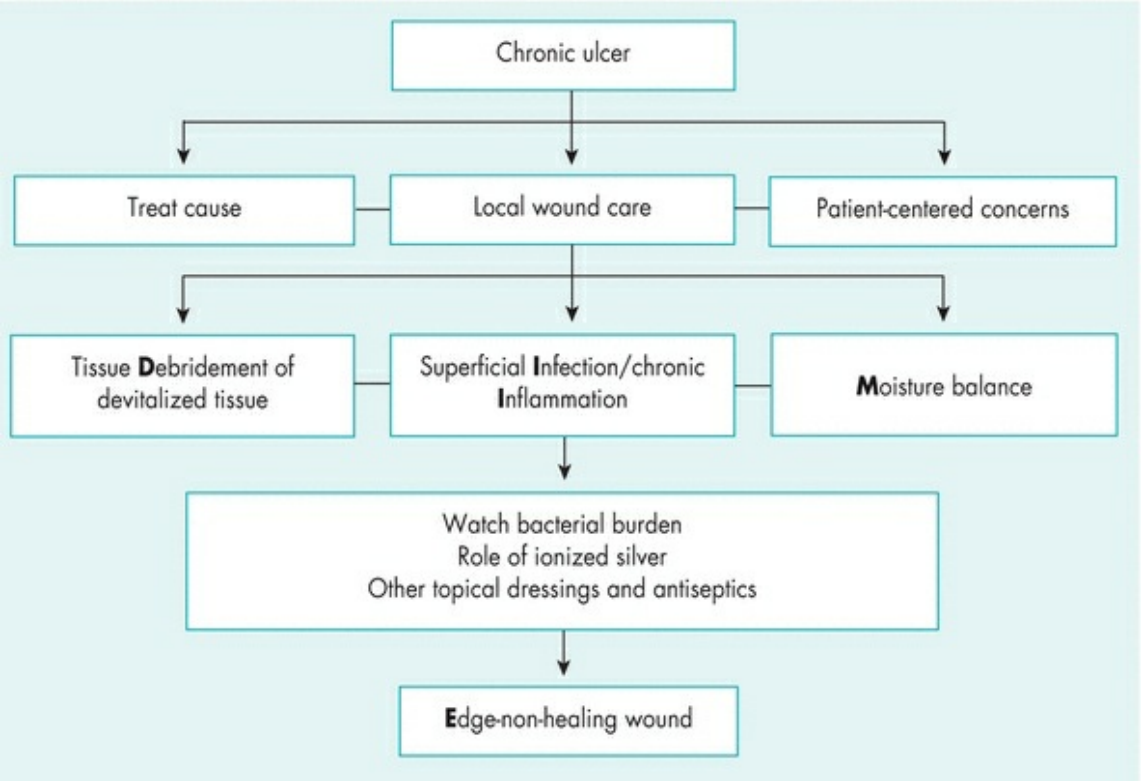


Figure 8-3. TIME is now DIME: Wound bed preparation DIME© model. (Reprinted with permission of Wound Care Canada, the official publication of the Canadian Association of Wound Care. Available at: http://www.cawc.net. ©2006.)

Table 8-2 TIME is Now DIME: Wound Bed Preparation DIME Model

	Chronic ulcer	
Treat cause	Local wound care	Patient-centered concerns
Tissue **D**ebridement of devitalized tissue	Superficial **I**nfection/ chronic **I**nflammation	**M**oisture balance
	Watch bacterial burden Role of ionized silver Other topical dressings and antiseptics	
	Edge—nonhealing wound	

Reprinted with permission of *Wound Care Canada*, the official publication of the Canadian Association of Wound Care. Available at: http://www.cawc.net. © 2006. Keast, D.H., Parslow, N., Houghton, P.E., et al. *Best Practice Recommendations for the Prevention and Treatment of Pressure Ulcers: Update 2006.* Canadian Association of Wound Care. Available at http://www.cawc.net/images/uploads/wcc/4-1-vol4no1-BP-PU.pdf.

Practice Point

Save time and use the DIME[1,3,4,12] acronym in preparing the wound bed for healing.

- **D**ebridement
- **I**nfection or inflammation
- **M**oisture imbalance
- **E**dge—nonhealing

Predebridement Teaching

Patient-centered care should include teaching about the purpose and usual expectations of the debriding process. Before initiating as a treatment, the process needs to be explained and understood by the patient, their family, and their circle of care. Circle of care is an expression, which includes the individuals and activities related to the care and treatment of the patient. Thus, it covers the healthcare providers who deliver care and services for the primary benefit of the patient. Include in your teaching the debridement options that could be used and the method chosen should be acceptable to

the patient and clinician with the desired outcome and potential complications. It is vital that the patient and family understand why the necrotic tissue is being removed. The importance of educating patients and their families and obtaining written consent prior to debridement is one of the ten recommendations for conservative sharp wound debridement published by the Canadian Association for Enterostomal Therapy (CAET).[14] Some laypersons mistakenly believe that necrotic tissue is a "scab" (eschar) and is a sign of healing. They need to know that epithelium needs a firm pink surface of granulation tissue base at the wound edge to migrate optimally toward the center of a wound. Delayed healing will result if epithelial margins need to migrate down valleys under eschar or over hypertrophic or unhealthy bright red friable granulation tissue. Similarly, patients and families need to know that the wound will change during the debridement process because an acute wound is created within a chronic wound to stimulate the healing process. For example, tell them to expect the wound to become larger in size.

Another patient-centered concern within the WBP model is pain. (See chapter 12, Pain management and wounds for an extensive discussion on pain). Adequate wound debridement needs to consider how debridement pain will be managed. In an outpatient setting, for example, a wound clinic or practitioner's office, application of a topical local anesthetic agent prior to debridement may be adequate for small-size wounds. For larger wounds requiring more extensive debridement, surgical debridement may need to take place in the operating room where an anesthesiologist or nurse anesthetist can administer regional or general anesthesia. As with any surgery, a preoperative evaluation of the patient is performed by the anesthesiologist or nurse anesthetist to assess individual patient needs[15]. Wound care patients may have more comorbidities that need to be identified and evaluated prior to debridement. In one medical center, there were higher operative risks identified through higher American Society of Anesthesiologists (ASA) scores for wound patients (3.09) compared to other surgical patients (2.09).[15] Another safety example is a patient with a cardiac pacemaker or internal defibrillator will need a comprehensive assessment including knowing the specific type and manufacturer of any implanted devices and getting the cardiac device interrogation performed *before* the patient arrives on the operating table.[16] Some pacemakers (mostly older models) may malfunction with electrocautery devices used for hemostasis of bleeders.

If a patient is unable to express their pain experience, a family member

may be advocating for them so that they do not experience pain.[17] One such patient population includes patients with the locked-in syndrome (LIS). LIS can occur from a basilar artery thrombosis that results in a ventral pontine infarction leaving the patient without motor function. Although the patient cannot move or speak, and even though the patient's eyes may be closed, the patient may be awake and conscious and thus able to experience pain.[17] Special monitoring in the OR using bispectral (BIS) monitoring will facilitate adequate pain management during debridement.[17] Patients, their family, and circle of care need to understand that either regional or general anesthesia may be appropriate choices for debridement pain management in the OR.[18]

Debridement Methods

Mechanical, sharp/surgical, enzymatic, and autolytic are the common methods of debridement.[1,2,4,8,11] However, a resurgence in the use of older methods, such as maggots (biological or larval therapy), has become accepted practice in some wound care centers (Table 8-3).[2,4]

Table 8-3 Best Practices for Preparing the Wound Bed

Identify and Treat the Cause
- Diagnose and correct or modify treatable causes of tissue damage.
- Differentiate the wound's ability to heal: healable, maintenance, or nonhealable wound.

Address Patient-centered Concerns
- Assess and support the management of patient-centered concerns to enable healing.
- Provide patient education and support to increase adherence to the treatment plan.

Provide Local Wound Care
- Assess and monitor the wound history and physical characteristics (location and measurements).
- Debride healable wounds with adequate vascular supply, removing nonviable, contaminated, or infected tissue (surgical, autolytic, enzymatic, mechanical, or maggot).
- Clean wounds with low-toxicity solutions (such as normal saline or potable water); reserve topical antiseptic solutions for wounds that are nonhealable or those in which the bacterial burden is of greater concern than the stimulation of healing.
- Assess and treat the wound for increased bacterial burden or infection; distinguish from persistent inflammation of nonbacterial origin.
- Select a dressing that is appropriate for the needs of the wound, the patient, and the caregiver or clinical setting.
- Monitor the quantity and quality of wound exudate to prevent periwound maceration.
- Evaluate expected rate of wound healing; if suboptimal, reassess patient.

Provide Organizational Support
- For improved outcomes, education and evidence base must be tied to interprofessional teams with cooperation of healthcare systems

 Secondary source: Sibbald, R.G., et al. "Best Practice

Recommendations for Preparing the Wound Bed: Update 2006," *Wound Care Canada* 4(1):15-29, 2006.

Mechanical Debridement

Methods of mechanical debridement include wet-to-dry dressings, hydrotherapy (whirlpool), and wound irrigation (pulsed lavage).[1,4,3] Mechanical debridement may be more painful than other debridement methods, and the healthcare provider should always consider patient premedication for pain. Both whirlpool and pulsed lavage (see Chapter 9, Wound Treatment Options) require special equipment and skill. All of the mechanical methods are considered nonselective debridement; that is, they don't always discriminate between viable and nonviable tissue. Mechanical methods may be harmful to healthy granulation tissue on the surface of the wound and lead to bleeding, trauma, and disruption of the collagen matrix along with the necrotic tissue.

Practice Point

Mechanical debridement may be painful; consider patient premedication for pain.

Wet-to-Dry Dressings

Despite the drawbacks, such as pain and the necessary application up to three times per day, the use of wet-to-dry wound debridement dressings unfortunately remains a common treatment in some healthcare settings. This method involves placing a moist saline gauze dressing on the wound surface and removing it when it's dry. The removal of the dried gauze dressing facilitates removal of devitalized tissue and debris from the wound bed. However, newly formed granulation tissue and new cell growth are also removed. To prevent pain and to help remove the dry gauze, clinicians often wet the dressing before removal. However, this defeats the purpose of aggressively removing dead tissue. This type of debridement requires significant nursing time, and although the materials may be relatively inexpensive, the overall cost can often be greater than other techniques.

A wet-to-dry dressing can be used when a moderate to large amount of necrotic tissue is present and surgical intervention is not an immediate

option. Because of pain and the removal of viable tissue, the Centers for Medicare and Medicaid Services (CMS) has stated in its revised surveyors guidance on pressure ulcers in long-term care that use of wet-to-dry dressings should be limited.[19] CMS also states that wet-to-dry dressings should not be used in a clean, granulating wound. Instead, they recommend use of moist wound therapy dressings, which are discussed in chapter 9, Wound treatment options.

Hydrotherapy

Hydrotherapy (or whirlpool) debridement may be indicated for patients with large wounds that need aggressive cleaning or softening of necrotic tissue. It is contraindicated in granulating wounds because it can macerate and injure the wound bed. Hydrotherapy should be discontinued after necrotic tissue has been removed from the wound bed.

Hydrotherapy is performed by putting the patient's wound in a whirlpool bath and letting the swirling waters soften and loosen dead tissue. This procedure is usually performed in the physical therapy department, with an average treatment duration of 10 to 20 minutes up to twice per day. Operators should carefully monitor the water temperature to prevent burns. The water should be tepid (80°F to 92°F [26.7°C to 33.3°C]) or close to body temperature (92°F to 96°F [33.3°C to 35.5°C]).

This type of debridement may cause periwound maceration, traumatize the wound bed, and put the patient at risk for waterborne infections such as *Pseudomonas aeruginosa*. The potential for cross-contamination between patients is also a concern. Both patients and healthcare workers may be exposed to health risks associated with aerosolization. To minimize infection risks, the whirlpool tank must be cleaned thoroughly with an appropriate disinfectant after each use.

Pulsed Lavage

Pulsed lavage debridement is often indicated for patients with large amounts of necrotic tissue and for those in whom other debridement methods are not an option. It is accomplished by using specialized equipment that combines a pulsating irrigation fluid with suction.[20] Utilizing pulsed lavage, you can clean and debride a wound at variable irrigation pressures (measured in pounds per square inch [psi]). The pulsatile action and effective wound bed debridement may improve granulation tissue growth. This treatment takes 15 to 30 minutes and should be performed twice daily if more than half of the

wound contains necrotic tissue.

Patients may need to be premedicated for comfort before beginning the procedure. Safe and effective ulcer irrigation pressures range from 4 to 15 psi.[21] Water pressure control is critical during this procedure. Because fluid is being forced directly at the wound, the risk of driving organisms deep into the wound tissue is a concern. In addition, inhalation of contaminated water droplets or mist is possible for both the clinician and the patient.

Practice Point

Always use appropriate equipment to prevent excess lavage pressure. Wear personal protective equipment to prevent splash injury, including eye and face protectors as well as an impervious gown. Remember to administer pain medication to the patient before the procedure.

Sharp/Surgical Debridement

Sharp/surgical debridement includes the use of a scalpel, forceps, scissors, hydrosurgery devices, or lasers to remove dead tissue.[2,4,22] Sharp debridement is considered by many clinicians to be the gold standard of debridement.[23] It can cause pain, so a topical anesthetic, such as lidocaine cream or gels, may be required.[24] Patients may also need follow-up appointments for serial debridement.

Because viable tissue may also be removed inadvertently with this method, excellent judgment must be used when performing sharp debridement.[23–25] The clinician must be able to differentiate where and what to cut, identifying, for example, a tendon versus slough because both are yellow in color[23–26] (Fig. 8-4). Clinicians need guidance in discerning the line of demarcation between viable and nonviable keratinocytes at the wound edge. As shown in Figure 8-5, most clinicians can identify the stalled edge of a chronic wound (location A), where the keratinocytes, which are the cells that close the surface of the wound, cannot migrate across the wound bed. Research suggests that the wound edge really extends to location B[27,28] and that debriding only to this boundary may be

inadequate as it leaves behind impaired cells.[27,28] Therefore, adequate debridement of a chronic wound needs to extend beyond the point at which the keratinocytes have lost the ability to move (edge of the callus tissue at location B) to where the cells can move to heal the wound (location C). Clinicians may need to rethink the distance from the wound edge (location A) that they need to debride. Research focuses on an easy way for clinicians to identify how far from the wound edge to perform debridement, based on the pathology of the abnormal keratinocytes at the wound edge.[27,28] While this technique is being adapted for clinical use, Endara and Attinger have described a simple clinical technique to stain the tissues with methylene blue dye as a color guide to aid the clinician to do a complete wound debridement.[26]

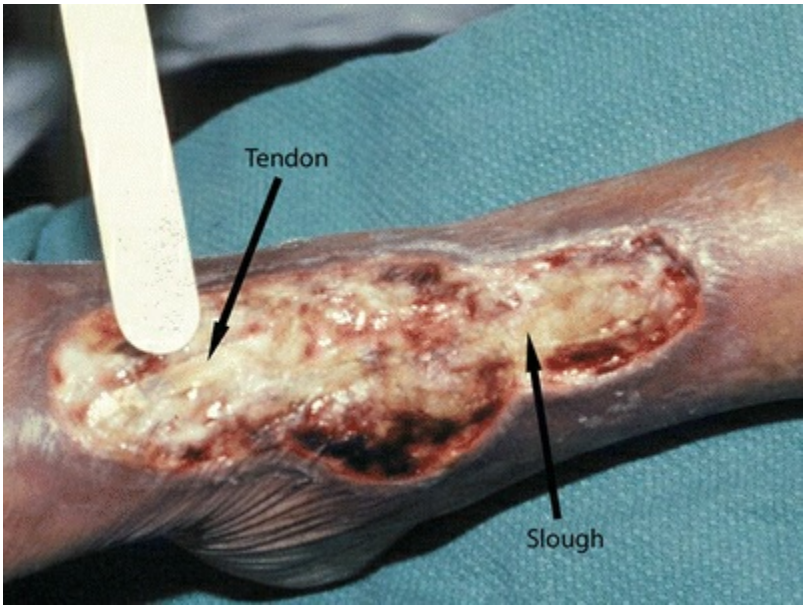


Figure 8-4. Differentiating tendon from slough. Performing debridement requires knowing where and what to cut. For example, tendon and slough both are *yellow*—the clinician must be able to distinguish between them.

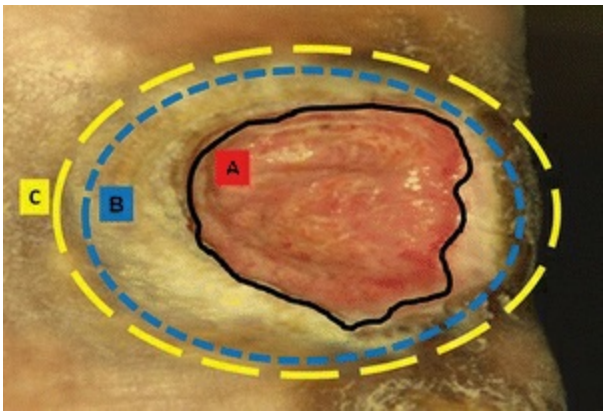


Figure 8-5. Margin of debridement. The *solid black line* **(A)** indicates a nonhealing edge. The *outer blue and yellow broken lines* **(B and C)** indicate two possible margins of debridement. Location B is the edge of the wound, whereas location C is the presumed location of the healing edge of the wound where keratinocytes have the ability to migrate and participate in the wound healing process. Therefore, debride to location C. (Adapted from Tomic-Canic, M., Ayello, E.A., Stojadinovic, O., et al. "Using Gene Transcription Patterns [Bar Coding Scans] to Guide Wound Debridement and Healing," *Advances in Skin & Wound Care* 21(10):487-92, 2008.)

Nonviable surgical debridement must be distinguished from sharp/surgical debridement, which may remove viable tissue on the wound surface to a bleeding base and thus create an acute wound within a chronic wound.[1–3,12,23] This procedure is usually performed by experienced physicians or surgeons. In the United States, only physicians can perform surgical debridement in the operating room with hydrosurgery devices. Individual states may allow nurses, physical therapists, and physician assistants with appropriate licensing and training to perform some sharp debridement procedures with a scalpel, forceps, or scissors. Recommendation for conservative sharp debridement for nurses in the USA is written by WOCN[29] and for Canada.[14]

The use of sharp debridement is based on expert opinion and clinical data. Steed et al.[30] reanalyzed data from a six-site random controlled trial that tested the use of recombinant human platelet–derived growth factor (rhPDGF) versus placebo in neuropathic diabetic foot ulcers. These authors found significantly higher healing rates in treatment facilities in which more frequent and complete surgical debridement to bleeding tissue was performed rather than simply removal of the periwound callus. The removal of loose bright friable granulation tissue from the surface of an ulcer removes senescent fibroblasts as well as bacteria that may be arranged in

biofilms. The wound surface bacteria and callus around the perimeter of the ulcer lead to underlying tissue damage. Surgical debridement is used for adherent eschar and devitalized or dead slough on the wound surface. This method can be selected for acute infected wounds and should be the first choice for wounds demonstrating signs of advancing cellulitis or sepsis. Small wounds may be debrided at the bedside, but extensive wounds—for example, a stage IV pressure ulcer—may require debridement in the operating room. Physicians have reported that the use of hydrosurgery devices has decreased the number of times that maintenance debridement was needed[22] (Fig. 8-6). Other authors have described the use of hydrosurgery devices in combination with undiluted Betadine and a bilayer porcine collagen dressing to successfully debride burns.[31] Surgical/sharp debridement must be performed with extreme caution in patients taking anticoagulant medications. The medication may need to be held for a short period of time prior to the procedure. Patients with prolonged bleeding may be best treated with other methods of debridement (Figs. 8-7 and 8-8).

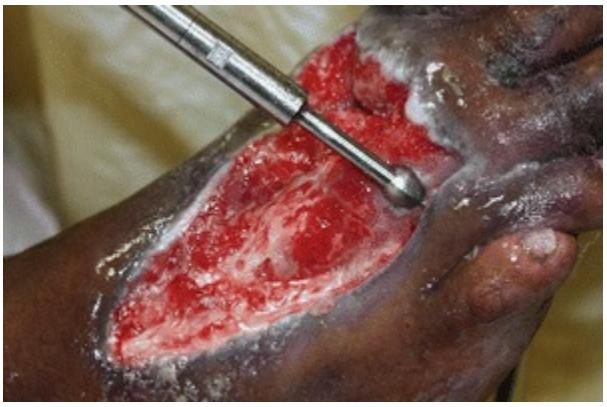
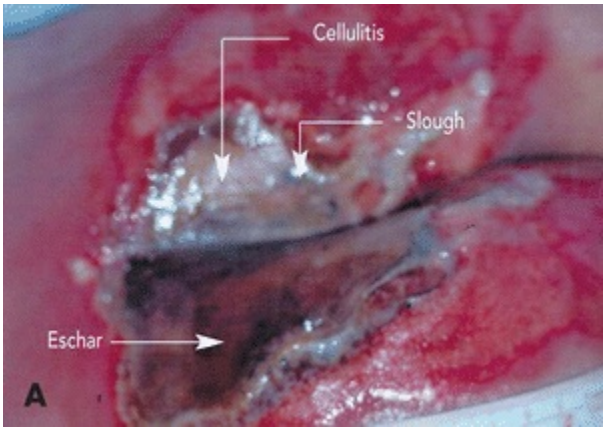


Figure 8-6. Hydrosurgery device. Example of one type of device used to remove necrotic tissue. (Photo courtesy of Jeffrey Niezgoda, MD.)



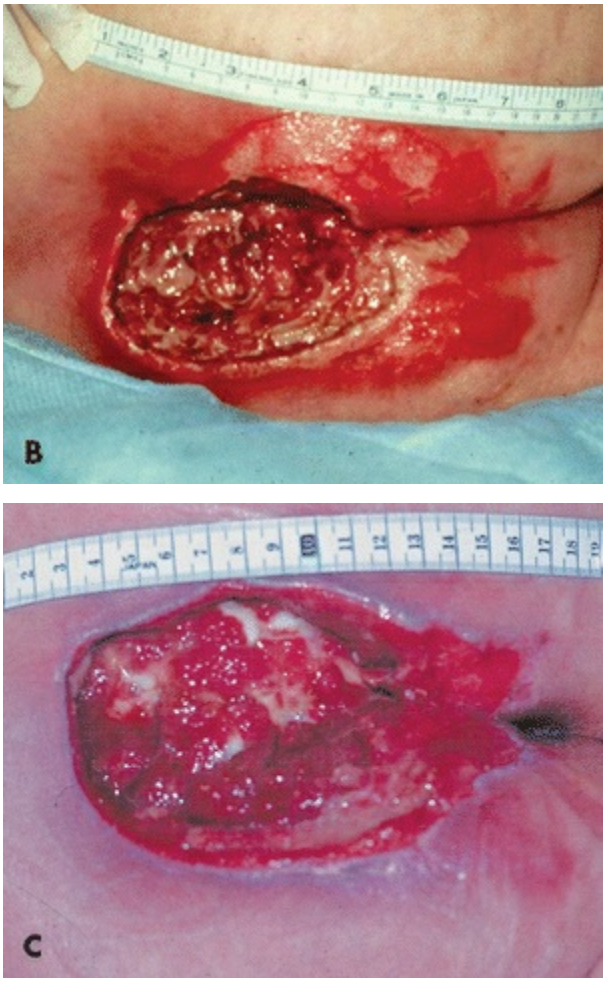
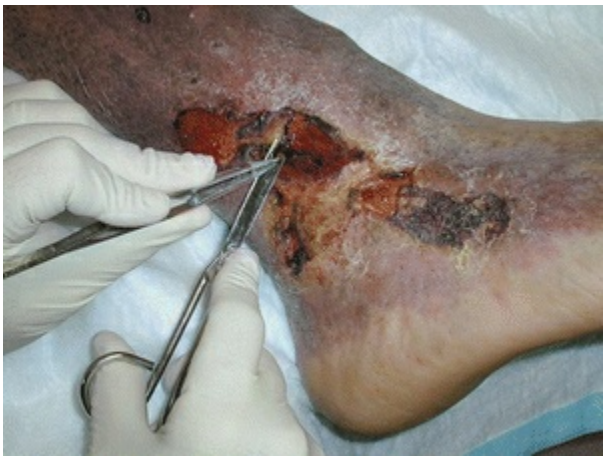


Figure 8-7. Surgical wound debridement case series. **(A)** This pressure ulcer with slough and eschar requires surgical debridement because of advancing cellulitis. **(B)** The same pressure ulcer after surgical debridement. Note the absence of eschar. Cellulitis is still present. (**C**) The same pressure ulcer after 7 days of treatment shows minimal necrotic tissue and significant amounts of granulation tissue. Note the change in the cellulitis surrounding the wound.



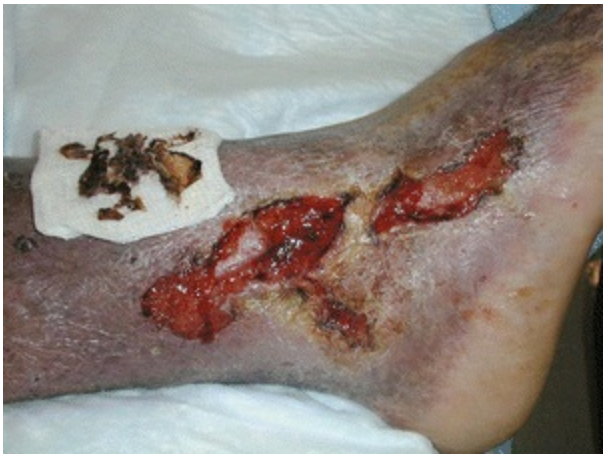


Figure 8-8. Sharp debridement at the bedside. Small wounds may be debrided at the bedside, as shown here. Notice the increased size of this ulcer after debridement. (Photos courtesy of Steven Black, MD.)

Sharp debridement can also disrupt biofilm. Wolcott et al. used both sharp debridement and antibiotic treatment to demonstrate a ninefold reduction in bacterial concentration compared to patients without debridement and only antibiotics were used.[32]

Practice Point

Exercise caution when performing surgical/sharp debridement on any patient who has been on a prolonged course of anticoagulant therapy.

Enzymatic Debridement

Enzymatic debridement is considered safe, effective, and easy to perform. Enzymes are effective wound surface cleaning agents that accelerate eschar degradation and debridement. The removal of debris helps a chronic wound move from the inflammatory stage to the proliferative stage, resulting in enhanced wound healing.[33] Enzymatic agents are an ideal option for patients:

- who are not candidates for surgery
- in combination with surgical debridement of slough to prevent secondary infection
- receiving care in a long-term facility or at home where other

debridement methods may not be available

- requiring maintenance debridement.[34]

Enzymatic debridement is accomplished by applying topical enzymatic agents daily to devitalized tissue. These agents will digest and dissolve necrotic tissue in the wound bed by breaking down collagen, elastin, and other parts of the abnormal devitalized wound matrix.[2,4,12] Before applying the topical enzymatic agent, crosshatching or scoring, if allowed by state licensing laws, can be performed to enhance local penetration of the agent.[35]

If infection has spread beyond the ulcer (as in advancing cellulitis), immediate removal of necrotic tissue is usually recommended. Surgical debridement and then enzymatic debridement should be considered. Enzymes often can be used alone, to break down the eschar before or after sharp debridement, or in conjunction with mechanical debridement.[34] The slough produced from enzymatic debridement often facilitates bacterial growth and makes some patients susceptible to deep and surrounding infection. Some topical antiseptic nonrelease dressings (polyhexamethylene biguanide [PHMB] gauze or foam/methylene blue–crystal violet foam) or topical antibiotics are compatible with enzymatic debriding agents and may be used in conjunction with the treatment.[36] (For more information on dressing types, see Chapter 9.)

Enzymes that act on necrotic tissue are categorized as proteolytics, fibrinolytics, and collagenases, depending on the tissue component they target. Because papain–urea enzymatic debriding agents target eschar, they are often used on necrotic wounds. However, the Food and Drug Administration removed papain–ureas from use in the United States as of 2008.[37] Those practicing outside the United States, where papain–urea is available, are urged to review the literature about this enzymatic debriding agent.[33,35,38–40] Collagenases target nonviable collagen tissue while sparing viable tissue, thereby making them useful in necrotic wounds with slough tissue at the wound base.[36]

Practice Point

Silver and other metals, including zinc oxide, can inactivate enzymes; be careful when selecting cleaning agents and dressings

that contain any ingredients that can interfere with enzyme action.[36]

Enzymes are a prescription drug and require a physician's or prescriber's order and since they are drugs must be signed for by the nurses in the patient's medication record. Collagenase is usually applied once per day. Before reapplying any enzymatic agent, thoroughly clean the wound with normal saline or a wound cleanser to remove any residual enzymatic ointment and loose wound debris.[35] Avoid solutions or dressings with metal ions, such as mercury or silver, when using collagenase, as they may inactivate the enzyme.[36]

If it is within your state's scope of practice and permitted by your facility, crosshatching or scoring the eschar with a scalpel (#11 or #15 blade), without cutting deep enough to cause bleeding, is recommended prior to applying the enzyme to let the debriding agent penetrate into the eschar. After scoring is complete, apply a thin layer (about the thickness of a nickel) of enzymatic ointment onto the necrotic tissue. Cover the wound with an appropriate dressing to keep it moist and let the debriding agent work.[35,36,41]

Collagenase is derived from *Clostridium histolyticum*. It may be more effective than papain–urea at degrading collagen and elastin and is thought to work from the bottom of the wound up.[39] Research supports collagenase as a more selective enzyme that may be more effective in controlling or reducing pain when compared with other debridement methods.[35,42] Collagenase upregulates the migration of keratinocytes over the wound bed and stimulates granulation.[8,43,44] Muller et al. documented debridement with collagenase to be quicker and more cost-effective than autolytic debridement with a hydrocolloid dressing in 24 patients with pressure ulcers.[45] Research by Riley and Herman[46] has shed new light on the role keratinocytes play in wound healing and how wound healing is influenced by different substances, including enzymes. This research assessed that collagenase doubled keratinocyte growth and migration; the increase was further enhanced fivefold when heparin-binding epidermal-like growth factor was added.[46] In an in vitro study, Mekkes et al. detected increased healing rates with collagenase and ineffective debridement with fibrinolysin (desoxyribonuclease).[47] Collagenase has also been shown to reduce scarring in partial-thickness burn wounds that has crucial implications for a patient's quality of life.[48]

The optimal method for enzymatic debriding agents has been the subject of much controversy. Research has also elucidated some differences between papain–urea and collagenase use. While overall healing rates for papain–urea and collagenase were not significantly different in one study, papain–urea did debride eschar at a faster rate.[49] However, another study found that papain–urea decreased keratinocyte migration by 50%.[46]

The search for new enzymes continues. Mekkes et al. have reported on Antarctic krill as an effective debriding agent,[50] while a systematic review by Bradley et al.[51] failed to provide evidence to support the use of enzymatic agents over other methods such as hydrogels or hydrocolloids for autolytic debridement.

Autolytic Debridement

Autolytic debridement uses the body's endogenous enzymes to slowly remove necrotic tissue from the wound bed. In a moist wound, phagocytic cells and proteolytic enzymatic enzymes can soften and liquefy the necrotic tissue that is then digested by macrophages. Autolytic debridement can be facilitated with appropriate dressings in the superficial wound that contains little necrotic tissue or a larger, deeper pressure ulcer.[1,2,4,12] Underlying these concepts is the requirement of adequate circulation and nutrition.[2] Autolytic debridement may take longer than some other methods; however, it represents a less stressful method to the patient and wound than mechanical debridement. Autolytic debridement is contraindicated in deep and surrounding infected wounds.

Autolytic debridement is easy to perform and involves applying a semiocclusive or occlusive moisture-retentive topical dressing including transparent films, hydrocolloids, hydrogels, and calcium alginate dressings.[2] (See chapter 9, Wound treatment options.) Wound fluid accumulates under the dressing, aiding in the lysis of necrotic tissue. This method is relatively pain-free in patients with adequate tissue perfusion.

Studies have compared the efficacy of hydrogel dressings and mechanical debridement with wet-to-dry dressings.[52–54] Several researchers[51–54] have concluded that autolytic debridement with a hydrogel is more time-efficient and cost-effective, resulting in faster healing when compared with wet-to-dry dressings. One case report found that use of a clear acrylic dressing promoted autolytic debridement and had the added advantage of being able to view the wound without removing the

dressing.[55] Schimmelpfenning and Mollenhauer[55] as well as Konig et al.[56] found that autolytic debridement with moist interactive dressings was equal in efficacy to enzymatic methods using collagenase. Smith's *Cochrane* review on debridement in diabetic ulcers concluded that hydrogels were more effective than gauze.[57] A case report by Cuschieri et al. reported the successful autolytic debridement of a large necrotic foot ulcer in a diabetic patient.58

Practice Point

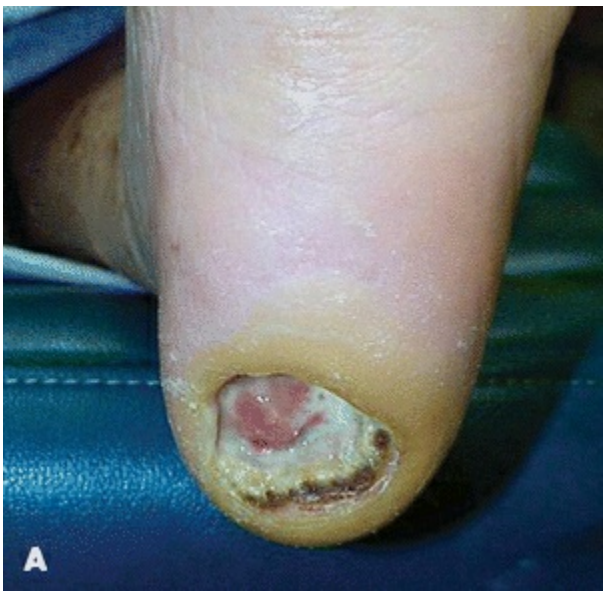
Be sure to tell the patient and family that fluid accumulating under the dressing is a normal part of the debridement process. Discolored wound fluid may not signal a wound infection.

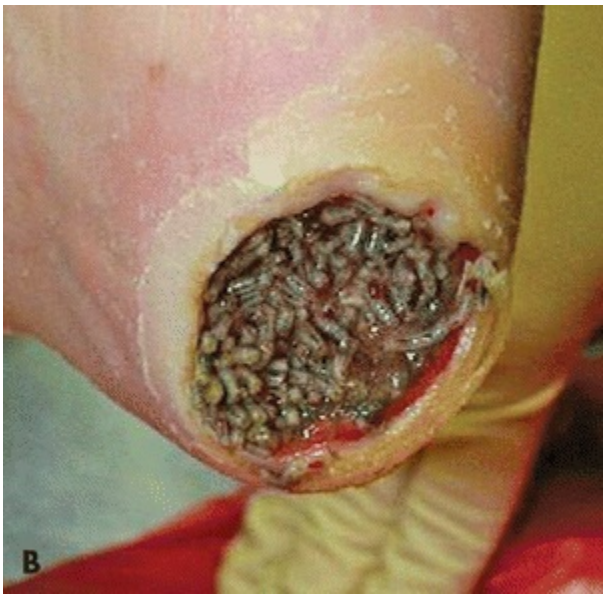
Monitor the wound for signs of infection, such as odor, increasing exudate or wound size, periwound erythema, edema, warmth, or increased pain, and discontinue autolytic debridement if these symptoms occur. Immunocompromised patients should be assessed frequently for any indication of infection. Autolytic debridement isn't the treatment of choice in deep and surrounding wound infections; in fact, it may lead to more severe infection and is therefore contraindicated in these situations. Surgical consult is warranted with appropriate medical management of the infection.

Maggot Therapy (Biological or Larval Therapy)

Maggot therapy was widely used in the early part of the 20th century. It fell out of favor due in part to the "disgust factor" and the use of newer modalities such as antimicrobial agents in wound treatments. With Europe leading the way, there has been a resurgence in the use of larval therapy in the United States.[59,60] In this type of debridement, a single or several applications of sterilized medicinal *Lucilia sericata* (green bottle fly) maggots are placed in the wound bed every 2 to 3 days[61] (Fig. 8-9). The specific application technique for how the maggots are actually put in the wound varies. Some place the maggots directly into the wound so they can roam around (free range), and others place the maggots contained in a

device such as a pouch or tea bag–like sack.[62] Early scientific reports of the evidence support that free-range maggot therapy maximizes debridement benefits compared with maggots that are contained in pouches or sacks and then placed in the wound.[62]





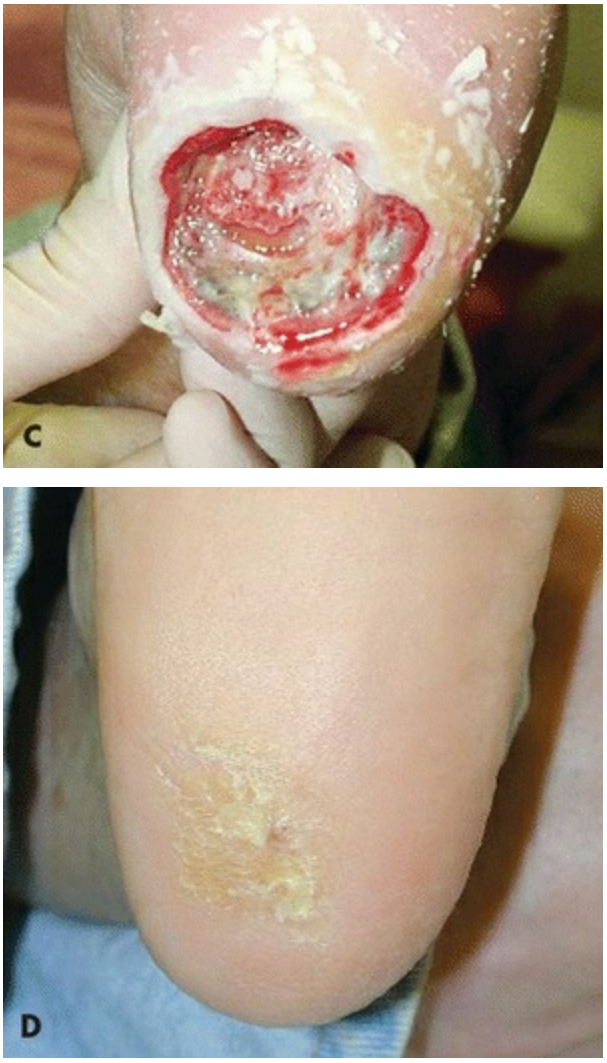


Figure 8-9. Maggots in heel wound. (**A)**This photo shows a heel ulcer with osteomyelitis on a middle-aged woman with diabetes who was on immunosuppressants following a kidney transplant 27 years earlier. **(B)** The same ulcer with sterile maggots placed in the wound for the purpose of debridement. (**C)** The same ulcer after removal of the first application of maggots. (**D)** The same ulcer, healed. (Photos courtesy of Pamela Mitchell, BTER Foundation.)

Just how do these larvae accomplish debridement? The mechanism by which maggot therapy works is believed to be by the enzymes the maggots secrete. These substances are proteinases that degrade the necrotic tissue.[63] The maggots also digest bacteria, making them effective in wounds with resistant bacteria strains.[64,65] A prospective randomized study provided evidence that complete lysis of methicillin-resistant *Staphylococcus aureus* (MRSA) and *Candida albicans* occurred within 24 hours when maggots were applied to 48 culture plates.[66] This effect continued to be observed for 5 days.[66] Maggots also encourage healing by stimulating granulation

tissue.

Prospective trials on the efficacy of maggot therapy have reported debridement and evidence of decrease in wound size.[1,67,68] Some case studies have reported the efficacy and selectivity of maggot debridement therapy.[69,70] Mumcuoglu[71] has reported significant debridement (80% to 85%) with clinical application of maggots as well as the prevention of amputation and bacterial spread.

Maggots can be used in almost all wounds. Richardson[72] and Thomas[73] reviewed current best practices. However, some contraindications for their use include a life- or limb-threatening wound, psychological distress or the "ick factor," bleeding abnormalities, and deep-tracking wounds.[59] The literature is unclear about some aspects of maggot use. Sherman[74] says that maggots should not be used in the presence of osteomyelitis or critical ischemia associated with arterial insufficiency, but Claxton et al. disagree.[59] Maggots are cost-effective when used properly and have the potential to help in situations where other resources are not available.[75] Despite the potential for psychological distress, most patients, as well as healthcare professionals, were satisfied with the treatment in one study.[69]

Something else to consider with maggot therapy is the level of pain it causes. There are conflicting reports about the level of expected pain with maggot debridement therapy. Sometimes, pain is minimal or absent.[76] In one report, however, 25% of patients with superficial painful wounds complained of increased pain during treatment with maggots.[63] These patients need analgesics for pain management. The type of wound might make a difference as to whether the patient has pain during therapy. In a retrospective study, Steenvorde et al.[77] found that diabetic patients, the majority of whom had neuropathy (n = 21), did not experience increased pain, while 40% (n = 8) of the enrolled nondiabetic patients experienced pain when being treated with maggots.

Choosing a Debridement Method

Does it matter which type of debridement you use on a wound? "No one method of debridement has been proven optimal for pressure ulcers," according to the Wound, Ostomy and Continence Nurses Society (WOCN).[8]

Given the various and conflicting evidence about the different debridement options, choosing the right way to debride wounds can be

challenging. Answering the following questions can help guide you in choosing the best debridement method for your patient.[1,2,4,11,12,14,78] Remember, your choice may be limited by the availability of the various debridement methods in your facility or healthcare system.

Determine the Ability of the Wound to Heal

Is there enough blood supply to heal?

Check that the Cause Has Been Corrected?

If you cannot correct the cause or there is inadequate blood supply to heal, conservative debridement of slough, moisture reduction, and bacterial reduction are achieved by conservative surgical removal of the slough and bacterial/moisture reduction through antiseptics such as povidone–iodine or chlorhexidine preparations.

How Much Time Do You Have To Debride?

Infected wounds require immediate attention and may require surgical debridement after systemic antimicrobial therapy has been initiated. The patient's clinical condition and the amount of time that you can devote to a treatment may influence your choice.

What Are the Wound Characteristics?

Consider the size, depth, location, amount of drainage (and whether it is increasing), presence (and extent) or absence of infection, and etiology of the wound.

How Selective a Method Is Needed?

Determine the risk for damage to healthy tissue when necrotic tissue is removed.

What Methods Are Permitted?

Check that the intended debridement method is allowed by your state's practice act and by your facility. For example, using a scalpel to crosshatch eschar requires specialized training and licensure.

What's the Care Setting?

Some resources available in a hospital aren't practical in the home and may not even be available in a long-term care facility.

How Much Debridement Is Enough?

How do you know when you've debrided enough? Assess the tissue in the wound bed: When most of the wound surface is covered with granulation tissue and the necrotic tissue is gone, you've debrided enough. As previously mentioned, Endara and Attinger describe their clinical technique to stain tissues with methylene blue dye prior to wound debridement so that the clinician can readily see the end point for surgical debridement.[26]

There are some methods supported by research that can guide practice for evaluating the effectiveness of debridement. Saap and Falanga[79] developed a method to assess adequacy of wound debridement. Their Debridement Performance Index may make more effective comparisons between different debridement methods and facilitate more predictive prognostic information.[79]

Falanga[80] has proposed that chronic wounds need constant debridement because maintenance debridement is an important part of the WBP in these wounds. In the past, debridement was regarded as a singular event based on the visible assessment of the wound. Now, however, it's thought that frequent, limited, maintenance debridement will keep the biological burden low and stimulate growth factors.[12,81–83] Maintenance debridement may prove challenging in the future due to proposed reimbursement changes in the United States based on the place in which debridement can be done as well as the frequency of the procedure.

Summary

Debridement is an essential part of the WBP management process. Although surgical debridement is the fastest way of removing necrotic tissue from a wound, it may not be appropriate for all patients in all healthcare settings. The selection of the correct method of debridement should be based on the individual patient and the degree of necrosis present. By knowing the options for debridement, you can help prepare the wound bed and assist your patient on the road to healing. Remember to address patient-centered concerns including pain prior to proceeding with the selected debridement method (Table 8-4).

Table 8-4 Overview of Debridement Methods

Method	Considerations	Contraindications
Surgical/Sharp		
Necrotic tissue is removed using a scalpel, scissors, forceps, or curette.	• Urgent need for debridement • Highly selective • Rapid results • Pain unless the patient has neuropathy; analgesia often needed • Risk of hemorrhage/complications • Cost; use of special equipment • Requires patient consent • Requires special training and expert confidence level (including anatomic knowledge) • Must distinguish between necrotic and healthy tissues • Can be done at bedside • May require an operating room and systemic anesthetics for extensive procedures • Anticoagulant therapy may need to be held prior to the procedure.	• Malignant wounds • Patients with clotting/ bleeding abnormalities • Ischemic tissue • Unstable • Underlying dialysis fistula, prosthesis, or arterial bypass graft • Caution with wounds involving hands and face • Caution with immunocompromised patients
Autolyic		
Endogenous enzymes present in wound fluid interact with moist dressing to soften and remove necrotic tissue.	• Need for minor or moderate debridement • Patient has a decreased or minimal risk of wound infection. • Performed in any setting • Can be used with other methods • Selective • Safe, easy to use • Painless and soothing when dressing in place • Slow • Risk of maceration to surrounding skin • Removal of some dressings may be painful. • Odor • Secondary dressing needed for some types of primary dressings • Absorptive dressing can dehydrate the wound bed.	• Some dressings cannot be used with infected wounds. • Exposed tendon/bone • Friable skin • Deep extensive wounds • Severe neutropenia • Immunocompromised patients
Mechanical		
Wet-to-dry: Moist dressing is applied to wound, allowed to dry, and removed with force.	• Larger wounds • Nonsurgical candidates • Nonselective • Painful • Frequent dressing changes required, may need to be done 2–3 times a day; not cost-effective • May macerate surrounding skin • Bleeding • Dressing fibers stick to wound and can cause a foreign body reaction. • May disperse bacteria into the air when removed • Traditional more than a modern accepted practice	• Clean wounds
Hydrotherapy: Moving water dislodges loose debris.	• Increases circulation to wound bed • May macerate periwound skin • Time-consuming • May cause trauma to wound bed and lead to bacterial contamination of wound and environment	• Clean wounds • Presence of diabetic neuropathy

	• Labor-intensive • Theoretical risk of fluid embolism or promotion of infection with irrigation • Healthcare professional needs personal protective equipment due to aerosolization. • Can impede venous blood flow in legs	
Pulsed lavage: irrigation combined with suction	• Bedbound patients • Wound with large amount of necrotic tissue	• Clean wounds
Maggot Larvae (*Lucilia Sericata*—Green Bottle Fly)		
Break down necrotic tissue and digest bacteria	• Psychological distress to patients or clinicians • Allergic reaction • Potential for increased pain especially in ischemic wounds • Time-consuming • Selective debridement • Rapid • Costly • May be painless • Decrease bacterial load • Bedside use • Can be used for various wound types, including infected wounds	• Allergies to adhesives, fly larvae, eggs, soybeans • Patients with bleeding abnormalities • Deep, tunneled wounds
Enzymatic		
Enzymes degrade and remove necrotic tissue.	• Patient on anticoagulants • Can be used on infected wounds • Cost-effective • Bedside use • Selective debridement • Decreased wound trauma • Cost varies • Usually daily application in United States • Sting with application/inflammation around wound with some enzymes • Not used with heavy metal salts (silver and mercury) • May need crosshatching of eschar • Clinicians need to document in patient's medication record because enzymes are prescribed drugs.	• Clean wounds • Allergy to component of the enzyme preparation

Kirshen, C., et al. "Debridement: A Vital Component of Wound Bed Preparation," *Advances in Skin & Wound Care*, 19(9):506-17, quiz 518-19, November-December, 2006.

Sibbald, R.G., Goodman, L., Woo, K.Y., et al. "Special Considerations in Wound Bed Preparation 2011: An Update," *Advances in Skin & Wound Care* 24(9):415-36, 2011.

PATIENT SCENARIO

Clinical Data

Mrs. AA is 55-year-old female patient who presented with a painful left large toe. She has type 2, adult-onset, non–insulin-dependent

diabetes mellitus with neuropathy. There is a palpable dorsalis pedis pulse, and her pain is aggravated by wearing shoes.

The best approach to this patient includes all the components in the WBP paradigm of treating the cause and patient-centered concerns before addressing the components of local wound care (**DIME: D**ebridement, **I**nfection/persistent inflammation, **M**oisture balance, and then the **E**dge effect for advanced therapies if wounds are not healing at the expected rate when all the components of WBP have been corrected).

For patient-centered concerns, the pain that has developed in a neuropathic foot with loss of protective sensation should alert the clinician to the disruption of deeper structures with infection (e.g., osteomyelitis) or a Charcot joint (acute inflamed bony structure due to multiple small pathological fractures).

Case Discussion

In this case, physical examination revealed a keratotic cap on the left second toe that required debridement (Fig. 8-10A). Vascular supply was assessed prior to surgical debridement. The palpable pulse indicated a local pressure of 80 mm Hg or higher, which was deemed adequate for healing. As is often seen in patients with neuropathy, removal of the surface of the callus revealed encasement of the toenail that had rotated under the tip of the toe with the claw deformity. When the nail and callus were removed, there was a small ulcer that probed to bone (Fig. 8-10B). This indicates a high probability of osteomyelitis, which was confirmed with an X-ray showing erosion of the phalangeal bone. Following debridement, a calcium alginate dressing was applied for hemostasis (Fig. 8-10C). With this type of dressing, the calcium donated from the dressing assists in local hemostasis while the sodium exchanged for the calcium creates a sodium alginate hydrogel for moist interactive healing, avoiding a proinflammatory hemorrhagic crust formation.

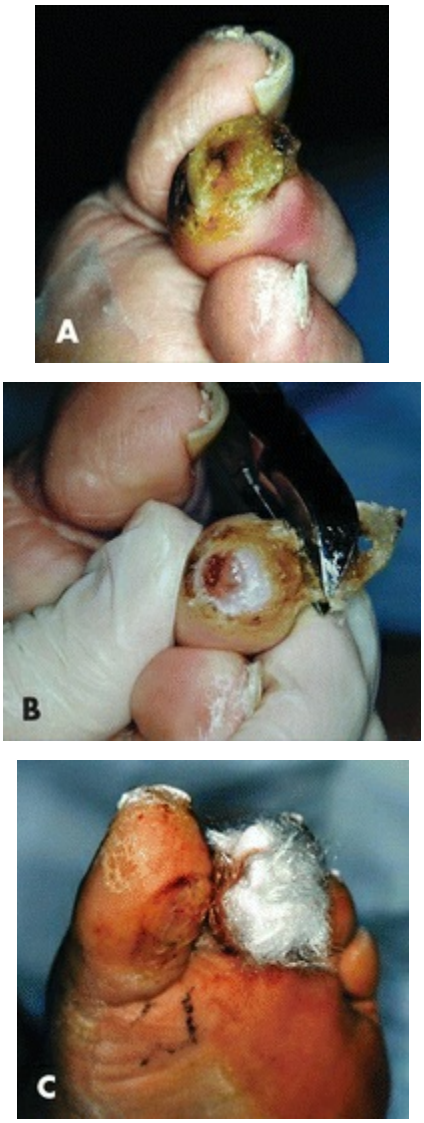


Figure 8-10. (A) Keratotic cap on the left second toe. **(B)**A small ulcer probing to bone was revealed when the nail and callus were removed. **(C)**Calcium alginate dressing applied for hemostasis after debridement.

The treatment principles employed in this case can be remembered with the mnemonic **VIPS**:

- **V**ascular supply with a palpable pulse was adequate for healing (if the pulse is not palpable, an ankle-brachial pressure index, toe pressures, or transcutaneous oxygen determination can be obtained to assess healability).
- **I**nfection: The osteomyelitis was treated with 3 months of oral clindamycin (good bone penetration with gram-positive and anaerobe coverage) combined with ciprofloxacin (gram-negative

coverage including *Pseudomonas*), with resolution of the pain and osteomyelitis.

- **P**ressure: The callus was due to pressure in the toe box of the patient's shoes. This was corrected with the ordering of deep-toed shoes and orthotics to remove local pressure.
- **S**harp surgical debridement was performed, including removal of the nail to reveal the osteomyelitis sinus. Maintenance debridement may also be necessary when callus periodically re-forms.

This case illustrates the need for holistic patient care with treatment of cause (VIPS), pain control (acetaminophen with codeine for nociceptive pain and a second-generation tricyclic nortriptyline at night and gabapentin in the daytime), and then local wound care with debridement and the use of calcium alginate followed by a silver alginate dressing.

Show What You Know

1. Which statement about the purpose of debridement is correct? Debridement:

A. is not essential for wound healing.
B. removes debris so cell movement is enhanced.
C. removes necrotic tissue in order to enhance the wound's biological burden.
D. reduces the need for moist wound healing.

2. Which sign in a stable necrotic heel would signal a need for debridement?

A. Exudate
B. Presence of thick, leathery eschar
C. An impending inspection by a regulator
D. Yellow slough

3. Which method is an example of mechanical debridement?

A. Collagenases
B. Maggots
C. Film dressings

D. Pulsed lavage

4. A resident in a long-term care facility is on Coumadin and needs debridement for a necrotic ulcer on his sacrum. Which of the following methods of debridement would be <u>least</u> indicated?

A. Surgical
B. Enzymatic
C. Mechanical
D. Autolytic

5. Which method of debridement would be best to use initially for a hospitalized client with an infected large sacral pressure ulcer?

A. Surgical
B. Enzymatic
C. Mechanical
D. Autolytic

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Wound Treatment Options

9

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Objectives

After completing this chapter, you'll be able to:

- explain moist wound therapy
- select dressings based on assessment of wound characteristics
- list indications for use of dressings by categories
- state the advantages and disadvantages for each dressing category
- use principles of care in dressing selection
- discuss the use of advanced therapies.

A Challenge for Clinicians

This is an exciting and challenging time for wound care clinicians as a new understanding of the biology of healing wounds has given rise to many new wound care treatments and therapies. Although we are gaining new knowledge as to the biology of wound healing, "we can no longer care only for the wound itself; we must step back and look at the entire human being who happens to have a wound that needs healing."[1] Being able to differentiate among the various treatment options, when and how to apply them, in what combinations, and when to change them has indeed become both an art and a science. "With the emergence of more complex products, we will be increasingly required to use these products appropriately to maximize their impact. As a better understanding of the wound environment

becomes available, our ability to tailor our approach and better treat the patient as a whole increases."[2]

Providing quality care for your wound patients starts with an analysis of the patient's individualized wound assessment and continues with developing a plan of care, selecting the proper product, and re-evaluating the plan of care as appropriate. Wound dressings can present a challenging decision for clinicians. Moist wound healing, moisture-balanced dressings, and certainly the principles of optimal wound interventions are key concepts needed to support the healing process. As clinicians try to heal wounds faster, the marketplace continues to provide many more treatment choices. There are reported to be more than 500 different types of dressings available to manage patients with wounds.[3] Keeping abreast of wound dressing choices and various application techniques, as well as which product to use and when, is an ambitious task for all clinicians.

Moist Wound Healing

Wound healing in the 21st century has certainly changed. There have been more advances in wound care over the past four decades than during the previous 2,000 years. This wound care revolution has been due in part to Dr. Winter's discovery (in the 1960s) of the importance of moist wound healing in experimental animals.[4] Hinman and Maibach[5] paralleled these findings of faster resurfacing in partial-thickness wounds in humans. Their research efforts laid the foundation for understanding the importance of moisture and moisture loss in wound healing. The concept of moist wound healing and moisture-balanced dressings is now commonly accepted by most clinicians throughout the world as best practice.

We now understand that wound healing must take place in a moist environment. Epithelial cells require moisture to migrate from the wound edges to re-epithelialize or resurface the wound. This process is likened to "leapfrogging" of the cells. In a dry wound, these cells have to burrow down underneath the wound bed to find a moist area upon which to "march" or move forward (Fig. 9-1).

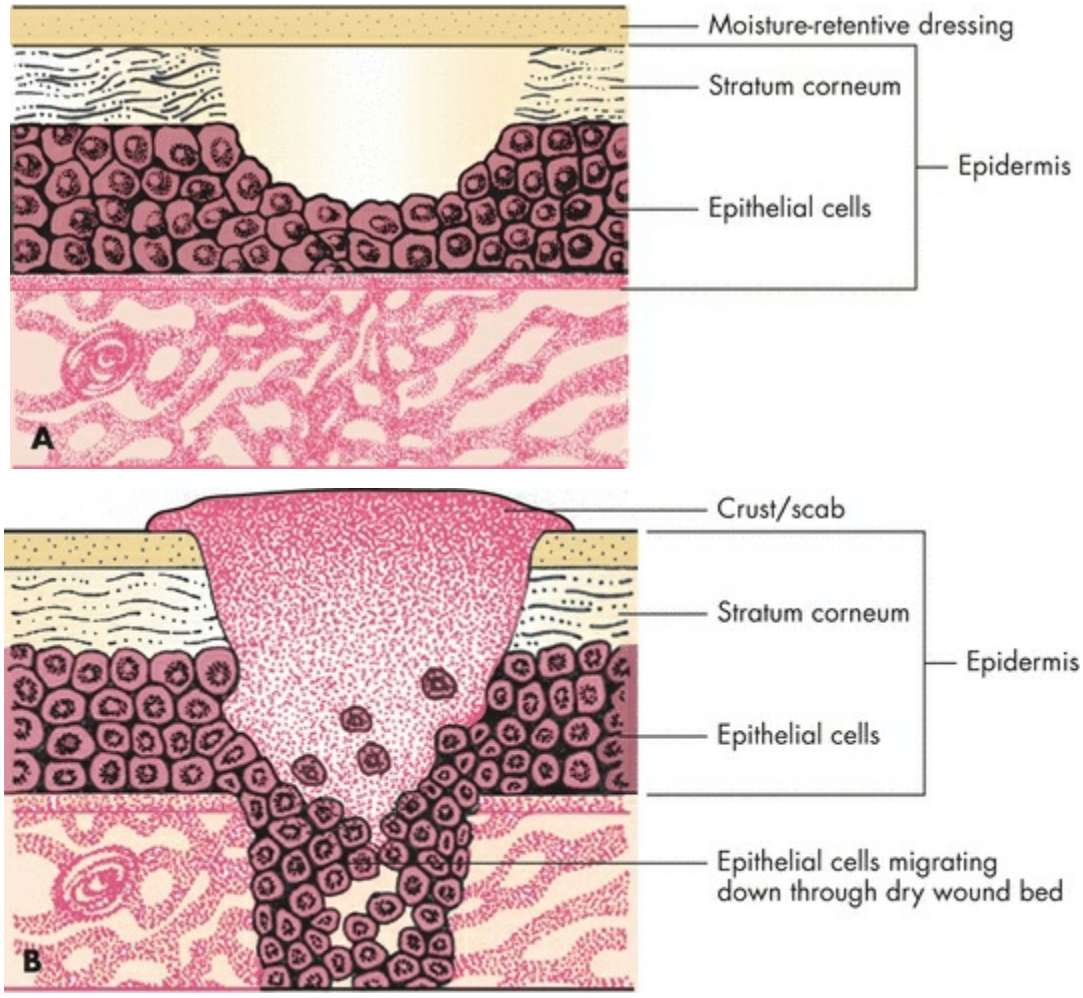


Figure 9-1 Epithelial cell migration in an open wound. **(A)** In a moist environment, epithelial cells can migrate on the wound bed surface to close the wound, as shown. **(B)** In a dry wound bed, epithelial cells burrow underneath the wound bed, as shown.

The concept of moist wound healing based on wound physiology and characteristics required that new dressing materials be developed to replace the passive coverings that had been used in the past, which evolved from such "natural" coverings as feathers, lint, grease, milk, wine, mud, leaves, and other concoctions. Today's wound dressings are actively involved in stimulating cell proliferation and encouraging epithelial cells to migrate. Moisture-balanced or moisture-retentive dressings also act as a barrier against bacteria and absorb excess wound fluid, creating opportune conditions for healing.[5–8] Formerly, wound dressings were used primarily to protect the wound from secondary infection by forming a barrier against bacteria and absorbing wound fluid. The greatest advantage of contemporary dressings is the maintenance of moist wound conditions, which is in contrast to the "classical gauze techniques" that led to the

formation of a dry, firmly adhering scab.[9] Today's dressings promote rapid healing, act as a barrier, decrease or eliminate pain, require fewer changes, provide autolytic debridement, and can be cost-effective if used appropriately.

Despite the benefits of newer dressing products, wet-to-dry gauze is still popular with many physicians, especially surgeons who have not been influenced by the development of modern wound dressings.[10] In a study by Pieper and colleagues[11] of 1,638 wounds treated in the home setting, the most commonly used dressing for all types of wounds (n = 406) was dry gauze. No dressing (i.e., an uncovered wound) was the second most common treatment (n = 252), and saline-moistened gauze (n = 145) was third. Advanced moisture-retentive dressings comprised less than 25% of all dressings used. So despite four decades of information on the benefits of moist wound therapy, some physicians may still revert back to the "old methods."

Research by Kim et al.[12] suggests that a saline gauze dressing acts as an osmotic dressing. As water evaporates from the saline dressing, it becomes hypertonic. Because the body wants to maintain homeostasis by re-establishing isotonicity, wound fluid is drawn into the gauze dressing. In addition to water, wound fluid includes blood and proteins. These substances form an impermeable layer on the dressing that prevents wound fluid from "wetting" the dressing; the net result is that the dressing dries out. The removal of a wet-to-moist dressing that has dried may then cause reinjury of the wound, resulting in pain and delayed wound healing.[10]

Pain at dressing removal is a frequent complaint heard by patients whose gauze dressings have dried into their wound. In "Principles of Best Practice: Minimising Pain at Wound Dressing-Related Procedures. A Consensus Document,"[13] the World Union of Wound Healing Societies recommends that clinicians use dressings that minimize pain and trauma during application and removal.[13]

Gauze dressings don't present a barrier to bacteria. Lawrence[14] demonstrated in an in vitro study that bacteria can pass through up to 64 layers of dry gauze. Once the gauze is moistened, it's even less effective as a barrier against bacteria. Infection rates are higher in wounds in which gauze is used as compared with wounds that are covered with transparent films or hydrocolloids.[15,16]

Successful wound healing depends on maintaining a moist environment. A balanced moist wound environment facilitates cellular growth and

collagen proliferation within a healthy noncellular matrix.[17] The right balance of moisture is critical to wound healing. Too much moisture in the wound bed can impair the healing process, damage surrounding skin, and cause periwound maceration. Excess moisture must be managed with an appropriate wound dressing, thereby preventing further tissue destruction and deterioration of the wound bed.[17]

Film and hydrocolloid were among the first dressing materials that could maintain a moist wound healing environment. New application techniques had to be learned and, more importantly, the clinical significance of wound fluid findings understood. The accumulation of light greenish–yellow fluid seen collecting under film dressings has caused us to relearn what is a normal expectation in a healing wound. Even the different nuance of wound odors has been the cause for new learning. For example, different odors occur as wound fluid interacts with different dressing materials. A wound being treated with alginate dressings, which are made from seaweed, may smell like "low tide."

The new generation of moisture-balanced dressings can maintain the right environment conducive to wound healing. These dressing may have occlusive, semiocclusive, absorptive, hydrating, autolytic, debriding, or hemostatic characteristics. Various wound dressing types will be discussed later in the chapter.

Treatment Decisions

The myriad of products available for wound care have enhanced the overall management of patients, but they have also created confusion about selecting the appropriate product. Optimal wound interventions should be dependent on the basic principles of wound care, attentive wound assessment, and expected outcomes. A complete wound assessment should be the driving element in all treatment decisions. (See chapter 6, Wound assessment.) Wound assessment should be based on the principles of wound care (Table 9-1).

Table 9-1 Principles of Care: The MEASURES Acronym

Minimize trauma to wound bed
Eliminate dead space (tunnels, tracts, undermining)
Assess and manage the amount of exudates
Support the body's tissue defense system
Use nontoxic wound cleansers
Remove infection, debris, and necrotic tissue
Environment maintenance, including thermal insulation and a moist wound bed
Surrounding tissue, protect from injury and bacterial invasion

Wound treatment decisions must be patient centered. What are the patient's goals and preferences? Local wound care starts with a thorough assessment of the wound and a comprehensive collection of data about the patient's overall status. Wound assessment parameters can assist with treatment choices and decisions for appropriate dressing selection[18] (Fig. 9-2).

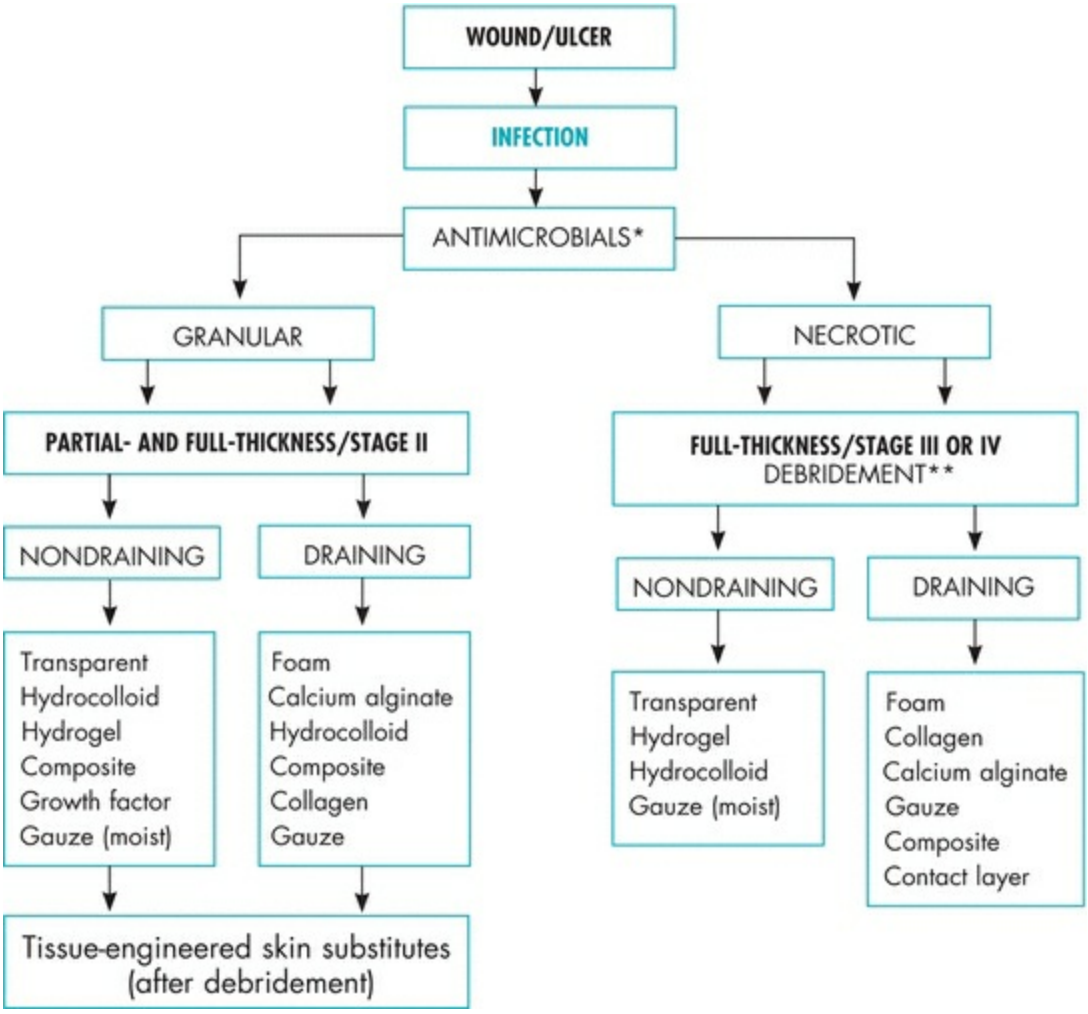


Figure 9-2 Wound care decision algorithm. Algorithm isn't inclusive of all available products. Always read package insert before use; product use may vary. *Antimicrobials may be topical or within a dressing component. **Debridement may be done by surgical, mechanical, autolytic, or enzymatic methods—products vary. (Used from Baranoski, S., McIntosh, A, Montoya, L. Optimizing the use of dressing, Lecture, Clinical Symposium on Advances in Skin & Wound Care. Las Vegas, NV. September 2014, with permission.)

Once a thorough, individualized wound assessment is complete, choosing dressings and treatments becomes a clinical decision that is mutually arrived at with the patient based on data collected during the assessment and the overall expected outcome.

Practice PointPractice Point

Dressing choices = Wound assessment + Principles of wound care

Treatment goals may aim to achieve a clean wound, heal the wound, maintain a clean wound bed, or place the patient in another setting to continue care. Clinicians should match the wound assessment characteristics with the dressing characteristics or function. The goal of care then becomes *using the right product on the right wound at the right time*. For example, a granular, nondraining moist or wet wound needs to maintain a moisture balance that is conducive to healing. The primary dressing choice would be a product that maintains a moist environment but doesn't cause maceration or desiccation of the wound bed. In another example, the goal of dressing selection for a necrotic draining wound is to loosen or soften the eschar for surgical debridement or to assist in autolytic debridement of the wound (see chapter 8, Wound debridement), absorb the excess exudate, and prevent trauma to surrounding tissue.

Practice PointPractice Point

If the wound is dry, add moisture. If the wound has drainage, absorb it. If the wound has necrotic tissue, debride it.

Clinicians need to reassess the wound status when completing dressing changes so that appropriate treatment interventions can be implemented. It's important to also understand that once the characteristics of the wound assessment change, so may the dressing choice. All wound products come with product information and instructions to guide the user in appropriate use of that product. The most appropriate dressing should be selected based on consideration of the patient, the wound, and the site[19] (Tables 9-2, 9-3, and 9-4).

Table 9-2 Characteristics of an Ideal Dressing

Use the following characteristics to determine the ideal dressing for your patient.
The ideal dressing should: • maintain a moist environment • facilitate autolytic debridement • be conformable for the range of use needed (such as to fill tunneling, undermining, or sinus tracts to eliminate dead space) • come in numerous shapes and sizes • be absorbent • provide thermal insulation • act as a bacterial barrier • reduce or eliminate pain at the wound site; pain-free removal.
The following considerations can be used to evaluate the dressing: • Number of days the dressing can remain in place • Reason for change or removal • Appearance of dressing (soiled or intact) • Ease of dressing application • Ease of dressing removal • Ease of dressing maintenance • Ease of teaching about dressing to caregiver

Adapted from Seaman, S. "Dressing Selection in Chronic Wound Management," *Journal of the American Podiatric Medical Association* 92(1):24-33, January 2002. Reprinted with permission from JAPMA.

Table 9-3 NICE© for Dressing Decision Making

There are thousands of dressings available and the clinician needs to decide which dressing to select for a particular wound. Ask yourself the following questions about the wound to help determine the dressing that is NICE© to use:

- Is there any **N**ecrotic tissue that needs to be debrided? (Make sure the wound has the ability to heal; if not, however, moist interactive dressings and active surgical debridement to bleeding tissue are contraindicated.)
- Is the wound **I**nfected or inflamed? (Clinicians often look for more than one sign or symptom before diagnosing infection.)
- Do the specific wound **C**haracteristics, such as location, need to be considered? (If the wound is around the anus, a waterproof adhesive dressing may be preferred.) Is pain an issue?
- Is there any **E**xudate; if so, why, how much, and what are the color and consistency?

Exudate may indicate that the cause of the wound has not been treated (e.g., edema due to venous insufficiency), that congestive heart failure is present (look for bilateral involvement and extension above the knee), that albumin levels are low (malnutrition, kidney, or liver disease), or that infection is present. Periwound skin needs protection from exudate by using absorbent dressings and protecting the periwound skin. Select a dressing by answering the four questions. Remember, it's NICE© to pick the right dressing.

Letter	Key Information to Know	Caution
Necrotic tissue slough, eschar	• Wet-to-dry dressings are a nonselective method of mechanical debridement. • Autolytic debridement of tissue is best accomplished with hydrogels, hydrocolloids, and alginate dressings. • With dressing-stimulated autolytic debridement, watch for secondary infection and remove unwanted slough with dressing change.	• Dressings are a slower method of debridement compared with sharp/surgical methods. • There is limited use of wet-to-dry dressings as a debridement method. • Some dressings cannot be used or caution is urged in necrotic wounds; check with the manufacturer for any contraindications for use. • Removal of nonviable tissue is a critical step in preparing the wound bed for healing.
Infection/inflammation	• Consider using antimicrobial dressings (e.g., silver or iodine). • Infected wounds may require more frequent dressing changes.	• Not all dressings can be used in infected wounds; check with the manufacturer for the specific brand indicated for use.
Characteristics	• Select and reassess a dressing based on location of the wound, such as the use of conformable dressings for hard-to-fit areas. • Waterproof dressings may be used if incontinence is an issue. • Consider the patient's pain and select dressings that may promote comfort and pain reduction.	• Change dressings when they become soiled from feces or urine. • Different dressings can remain in place for different lengths of time; check with the manufacturer for recommended frequency for dressing changes. • Avoid dressings that may increase or contribute to wound pain and consider systemic pain management strategies.
Exudate	• Match the absorbency of the dressing (none, low, moderate, heavy) to the amount of exudate from the wound. • Assess surrounding skin to evaluate for macerations.	• Surrounding skin needs to be protected from wound drainage; refer to the enabler LOWE. Search for the cause of the excessive exudate and the need to correct the cause. Exudate may be an indicator of infection.

Table 9-4 Wound Dressing Categories

Key—Exudate amount: ◊ Dry ◆ Light ◆◆ Moderate ◆◆◆ Heavy

Generic Category	Description/ Composition	Trade Names		Indications	Advantages and Benefits	Disadvantages
Transparent film ◊	Polyurethane or polymer membrane with porous adhesive layer that varies in thickness and allows oxygen to pass through and moisture vapor to escape	3M Tegaderm Bioclusive Blisterfilm ClearSite Comfeel Film Dermatell DermaView Gentell MVP Medipore OPSITE Suresite Transeal		Donor sites Primary and secondary dressings Partial-thickness wounds Pressure ulcers, stages I and II Superficial burns Peripheral IVs Abrasions	Wound inspection Impermeable to external fluids and bacteria Conformable Promote autolytic debridement Prevention/reduces friction Change every 5–7 d or prn if leakage noted Numerous sizes available Waterproof	Nonabsorptive May adhere to fragile skin Not for draining wounds Fluid retention may lead to maceration of periwound area. Third-degree burns
Transparent film with silver ◊	Ionic silver from a controlled-release barrier film	Arglaes		Pos-top incisions Central lines, CVPs, and PICC lines Infected wounds Highly colonized wounds	Antimicrobial Same advantages as transparent films	Sensitivity to silver Will inactivate enzymatic debriding agents Same disadvantages as transparent films
Hydrocolloid ◆ ◆◆	Occlusive or semiocclusive dressing that consists of gelatin, pectin, and carboxymethylcellulose Impermeable to bacteria and other contaminants	3M Tegasorb Comfeel Plus Cutinova Hydro DermaFilm DuoDERM CGF Exuderm FlexiCol Gentell Dermatell	Hydrocol Mediplus MPM Excel Nu-Derm PrimaCol ProCol REPLICARE Restore Ultec	Pressure ulcers, stages II and III Partial- and full-thickness wounds Under compression wraps/stockings Preventive dressing for high-risk friction areas Secondary dressing or under taping procedures First- and second-degree burns	Facilitates autolytic debridement Self-adherent Impermeable to fluids/ bacteria Conformable Reduces wound pain Thermal insulation Long wear time—3–7 d, depending on exudate	Contraindicated with muscle, bone, or tendon Not recommended for heavily draining wounds, sinus tracts, or fragile skin May be difficult to remove Contraindicated for third-degree burns
Hydrogel ◊ ◆	Water or glycerin based, nonadherent; contains 80% to 99% water; numerous sizes and forms (gels, sheets, strips, and gauze available)	3M Tegaderm Hydrogel Amerigel Aqua Flo AquaSite Aquasorb CarraDres Hypergel Intrasite Gel Normlgel NU-GEL Purilon Gel Regenecare Restore Gel SAF-Gel	CURAGEL DermaGauze Dermagran DermaSyn Elasto-Gel Elta Flexigel Gentell Hydrogel Skintegrity SoloSite TenderWet TransiGel Woun'Dres Hydrogel Xcell	Pressure ulcers, stages II–IV Partial- and full-thickness wounds Dermabrasion Painful wounds Dermal ulcers Radiation tissue damage First- and second-degree burns Skin tears Donor sites Necrotic wounds	Nonadherent Trauma-free removal/ soothing to patient Rehydrates the wound bed Reduces wound pain Can be used with topical medications Can be used in cavities or tunnels Softens and loosens necrosis and slough 24–72 h dressing change, depending on the form of gel	Some require secondary dressing to secure. May macerate periwound skin Not recommended for heavily draining wounds Contraindicated for third-degree burns
Hydrogel with silver ◊ ◆	Controlled-release ionic silver	DermaSyn Ag Elta SilverGel Gentell Hydrogel Ag Normlgel SilvaKollagen Gel SilvaSorb Gel SilverMed		Infected wounds Highly colonized wounds	Antimicrobial Can be left on up to 3 d Nonadherent Rehydrates the wound bed	Sensitivity to silver Not recommended for use in conjunction with topical medications Will inactivate enzymatic debriding agents Same disadvantages as hydrogel dressings

Foam ◆◆ ◆◆◆	Hydrophilic polyurethane or gel film–coated foam, nonadherent layer absorptive wound dressing	A	Lyofoam Mediplus Mepilex MPM Foam Optifoam Optiva Polyderm PolyMem Restore Foam Tielle Versiva XuSorb	Partial- and full-thickness wounds Pressure ulcers, stages II–IV Surgical wounds Dermal ulcers Under compression wraps/stocking Tunneling and cavity wounds (varies; check package insert)	Nonadherent Trauma-free removal Conformable, easy to apply and remove Frequency of dressing change depends on amount of drainage 3–5 day dressing change Available with adhesive and nonadhesive border in various shapes and forms	Not recommended for nondraining wounds Not recommended for dry eschar May require secondary dressing May macerate periwound area if not changed appropriately Contraindicated for third-degree burns
Foam with silver ◆◆ ◆◆◆	Controlled-release ionic silver	Acticoat Moisture Control Allevyn Ag Aquacel Ag Foam Biatain Ag Contreet Foam HydraFoam Ag Hydrofera Blue Mepilex Ag PolyMem Silver Optifoam AG Restore Foam with Silver		Infected wounds Highly colonized wounds	Antimicrobial Some can be left on up to 7 d Same advantages as foam dressings	Sensitivity to silver Must have exudate for silver to be released Will inactivate enzymatic debriding agents Same disadvantages as foam

Generic Category	Description/ Composition	Trade Names		Indications	Advantages and Benefits	Disadvantages
Calcium alginate ◆◆ ◆◆◆	Nonwoven composite of fibers from calcium–sodium alginate, a cellulose-like polysaccharide, manufactured from brown seaweed; forms a soft gel when in contact with wound exudate	3M Tegaderm Alginate Aquacel (Hydrofiber) Algicell AlgiSite Biatain Alginate Curasorb DermaGinate Gentell Calcium Alginate Kalginate Kaltostat	Maxorb Extra(CMC Alginate) Melgisorb Nu-Derm Alginate Restore Sorbsan	Partial- and full-thickness wounds Pressure ulcers, stages III and IV Dermal ulcers/dehisced wounds Pos-top wounds for hemostasis Sinus tracts, tunnels, or cavities Donor sites	Trauma-free removal Can be used with tunneling and undermining Hemostatic properties for minor bleeding Change every day to every other day Available in sheets, ropes, and within other composite-type dressings	Contraindicated for dry eschar, third-degree burns, surgical implantation, and heavy bleeding May require secondary dressing Gel may have odor during dressing change.
Calcium alginate with silver ◆◆ ◆◆◆	Controlled-release ionic silver	Algidex Ag Algicell Ag Aquacel Ag (Hydrofiber) DermaGinate Ag Gentell Calcium Alginate Ag Maxorb Ab (CMC Alginate) Opticell Ag Restore Alginate with Silver Silverlon Calcium Alginate Sorbalgon Ag	Silvercel	Infected wounds Highly colonized wounds	Antimicrobial Change every 3 d or prn Same advantages and benefits as calcium alginate dressings	Sensitivity to silver Must have exudate for silver to be released Will inactivate enzymatic debriding agents Same disadvantages as calcium alginate dressings

Composites ◆ ◆◆ ◆◆◆	Combination of two or more physically distinct products manufactured as a single dressing that provides multiple functions; may include a bacterial barrier, absorptive layer, foam, hydrocolloid, or hydrogel; semiadherent or nonadherent	3M Medipore 3M Tegaderm Absorbent Pad Alldress Barrier Island Dressing CombiDERM Comfortell Compdress Island	Covaderm Plus Coverlet Covrsite DermaDress Dudress Film Top Leukomed Mediplus Mepore OPSITE Stratasorb Suresite Telfa Island Viasorb	Primary and secondary dressings for partial- and full-thickness wounds Pressure ulcers, stages I–IV Dermal ulcers Surgical incisions	Conformable Multiple sizes and shapes available Easy to apply and remove Most include adhesive border Frequency of dressing change dependent on wound type (check package insert)	Adhesive borders may limit use on fragile skin. Some contraindicated for stage IV ulcers (check package insert) May not provide moist wound environment
Composites with silver ◆ ◆◆ ◆◆◆	Transparent film with an alginate pad	Arglaes Island		Infected wounds Highly colonized wounds	Antimicrobial Can be left in place up to 5 d Advantages and benefits same as composite dressings	Sensitivity to silver Must have exudate for silver to be released Will inactivate enzymatic debriding agents Same disadvantages as composite dressings
Enzymatic debriders ◊ ◆◆ ◆◆◆	Prescriptive collagenase ointment that digests collagen	Collagenase/ Santyl		To debride necrotic wounds, pressure ulcers, dermal ulcers, pos-top wounds	Collagen in healthy tissue is not attacked. Nonsurgical method of debridement Requires daily dressing changes	Adversely affected by certain detergents, acidic solutions, and heavy metal ions such as mercury and silver
Collagen ◆ ◆◆ ◆◆◆	Major protein of the body. Dressing stimulates cellular migration and contributes to new tissue development derived from bovine, porcine, or avian sources.	Biostep CellerateRx Gel/Powder ColActive DermaCol Endoform Fibracol Promogran Matrix Puracol Stimulen Woun'Dres Collagen Hydrogel		Chronic nonhealing wounds Partial- and full-thickness wounds Pressure ulcers, stage III and some stage IV (check package insert) Dermal ulcers Donor sites Surgical wounds	Absorbent, nonadherent Biodegradable gel Conforms well May be used in combination with topical agents 1–3 d dressing change	Contraindicated for third-degree burns and sensitivities to collagen or bovine products Not recommended for necrotic wounds May require rehydration
Collagen with silver ◆ ◆◆ ◆◆◆	Releases silver ions that are antimicrobial while collagen binds with MMP (matrix metalloproteases) in chronic wound exudate	Biostep Ag ColActive Ag Prisma Matrix Puracol Ag		Infected wounds Highly colonized wounds	Antimicrobial Can be left on up to 7 d Same advantages as collagen dressings	Sensitive to collagen and silver Will inactivate enzymatic debriding agents Same disadvantages as collagen dressings
Antimicrobial Therapies						
Nonadherent antimicrobial dressings that protect against bacteria and/or decrease bacterial load					Decreases bacterial load in wound Reduces risk of infection	Contraindicated in patients sensitive to ingredients described
◆ ◆◆	Cadexomer iodine impregnated: immediate and controlled release of cadexomer iodine	Iodoflex Pad Iodosorb Gel		Infected wounds—any type (pressure ulcers, venous, arterial, diabetic, or surgical wounds)		

◆ ◆◆ ◆ ◆◆	Controlled-release silver powder Polyhexamethylene biguanide impregnated (PHMD)	Arglaes Powder Kerlix AMD		Partial- or full-thickness wounds Colonized, chronic nonhealing wounds		
◆◆ ◆◆◆	Methylene blue and gentian violet	Hydrofera Blue		Inhibits the growth of bacteria and viruses		
Contact layer with silver ◆ ◆◆ ◆◆◆	Immediate and sustained release of ionic silver in a nonadherent primary dressing	3M Tegaderm Ag Mesh Acticoat AFM Ag Restore TRIACT Silver SilverDerm 7 Silverlon		Infected wounds Partial- or full-thickness wounds Colonized, chronic nonhealing wounds Under compression wraps/stockings Over grafts or skin substitutes	Inhibits growth of pathogens, especially antibiotic-resistant strains Antimicrobial action effective up to 7 d	Sensitivity to silver Secondary dressing required Must be removed and wound cleansed prior to MRI Not recommended for use in conjunction with topical medications Some may stain or discolor surrounding tissue due to silver turning black when it oxidizes. Contraindicated with enzymatic debriding agents
Contact Layer ◊ ◆ ◆◆ ◆◆◆	Nonadherent primary dressing that allows exudate to pass through	3M Tegaderm nonadherent contact layer Adaptic Conformant 2 nonadherent contact layer Covrsite Dermanet Drynet Wound Veil Mepilex Transfer Mepitel		Partial- and full-thickness wounds, donor sites, skin grafts	May be used with topical medications Less trauma to the wound bed Prevents the outer dressing from adhering to the wound.	Secondary dressing is required. Impenetrable exudate may macerate.
Antimicrobial Therapies (continued)						
Negative pressure wound therapy ◆◆ ◆◆◆	Noninvasive active therapy using localized negative pressure to promote healing	ActiV.A.C Engenex extriCARE InfoV.A.C. Invia Kalypto PICO Prevena PRO-II Pro-III Quantum Renasys SVED SNaP V.A.C		Moderate to heavy exuding wounds Partial- and full-thickness wounds Venous, arterial, diabetic ulcers, and dehisced wounds Pressure ulcers, stages III and IV Surgical wounds Flaps and grafts Acute traumatic wounds	Decreases edema Decreases bacterial colonization Increases blood supply and granular tissue formation Generally, dressing changed every 48–72 h (specifics for dressing and equipment vary by manufacturer)	Healthcare worker needs training to apply and operate equipment. Not reimbursed in acute and long-term care facilities May adhere to some wounds Not recommended for nondraining wounds or wounds with eschar Contraindicated for wounds with malignancy and untreated osteomyelitis
Advanced Wound Care Therapies						
Cellular- and/or tissue-based products ◆	– Nonviable cells, tissue-based human – Nonviable cells, tissue-based animal – Viable human cells, noncultured: intact tissue – Viable human cells, cultured in vitro: animal substrate – Viable human cells, cultured in vitro: synthetic substrate	AlloSkin DermACELL EpiFix GammaGraft Graftjacket MatriStem Mediskin	Oasis PriMatrix Apligraf Dermagraft Grafix TheraSkin	Partial- and full-thickness wounds Venous and diabetic ulcers Granular wounds Burns Chronic wounds	Growth factors and/or collagen may be incorporated within the product. Decreases wound healing time No donor site Decreases pain for many patients	Wound has to be granular. Check manufacturer's directions for storage and shelf life. Free of infection. Review product description if patient has sensitivity to porcine- or bovine-derived products.

Autologous platelet-rich plasma (PRP) gel	Autologous point-of-care process where small volume of blood is drawn from the patient; the blood is separated in a centrifuge: the resulting PRP gel is extracted and activated. Activation has been shown to cause the platelets to release autologous multiple growth factors; the fibrinogen in the plasma converted to a fibrin matrix scaffold and the liquid forms a gel.	AutoloGel		Chronic nonhealing wounds Exuding wounds Pressure ulcers, stages III and IV Arterial and venous ulcers Diabetic foot ulcers Mechanically or surgically debrided wounds	Moves stalled wounds out of inflammatory stage Growth factors, cytokines, and chemokines from the patient's own blood are applied topically to the wound bed	Special equipment needed Healthcare worker needs training to apply and operate equipment. Multiple weekly applications
Miscellaneous wound dressings	Medical-grade active *Leptospermum or Manuka* honey from New Zealand Available in various forms.	Medihoney Therahoney		Infected wounds Highly colonized wounds Wounds with slough or necrotic tissue Pressure ulcers, stages II–IV Partial- and full-thickness wounds Venous ulcers Diabetic ulcers	Antibacterial barrier Autolytic debridement properties	Allergies to honey Brief stinging sensation
	Sodium chlorite dressing consists of soft nonwoven material (viscose/polyester) impregnated with sodium chloride	Mesalt		Moderate to heavy exuding wounds of any type Infected wounds of any type	Facilitates cleansing of wounds with exudate, slough, or infection	Should not be used on dry wounds or minimally exuding wounds Should not be allowed to come in direct contact with exposed bone or tendon
	Balsam Peru, Castor Oil USP/NF, Trypsin USP	Xenaderm Vasolex Ointment		Pressure ulcers, dehisced wounds Radiation injury	Balsam Peru is a capillary bed stimulant and a mild antiseptic that helps prevent bacteria from growing. Castor oil works as a lubricant to protect the tissue. Helps relieve pain. Trypsin is intended for debridement of eschar and other necrotic tissue. Does not require a secondary dressing	Do not apply to fresh arterial clots. Temporary stinging sensation BID application Prescription is required.
Scar Care	Self-adhesive silicone contained within a gel or flexible sheet Occludes the skin to hydrate the scar area	CICA-CARE Mepiform		Hypertrophic scars, keloids, and closed wounds to prevent hypertrophic and keloid scars	Re-useable, durable Conformable	Contraindicated in open or infected wounds Contraindicated over scabs and stitches Takes 2–4 mo to see some results
Becaplermin gel	Genetically engineered platelet-derived growth factors produced in yeast and then formulated into a gel	Regranex		Neuropathic wounds Diabetic ulcers Granular wounds		Can't be used on infected wounds or wounds with necrotic tissue Must be refrigerated

The products listed are representative of type and are not meant to be all-inclusive; indications, advantages, and disadvantages are some examples; more suggestions may apply.

Antimicrobials may be topical or within a dressing component. Source: Surgical, Mechanical, Autolytic or Enzymatic debridement methods © A. McIntosh and L. (Galvan) Montoya, January 20, 2015.

Practice PointPractice Point

Wound dressings should be changed to meet the characteristics of the wound bed.

Practice PointPractice Point

Read and understand the information in the package insert before using a wound care product. Not all wound dressings will function in the same way.

Dressings that come in contact with the wound bed are considered primary dressings. Primary dressings fall within three categories: those that maintain adequate moisture, those that absorb excess moisture, and those that add moisture.[9] Secondary dressings are those that cover a primary dressing or secure a dressing in place. Clinicians should know which dressings are safe to be put into the wound itself and which are used as securement products. Several dressings on the market act as both primary and secondary dressings. Again, what wound characteristics are you addressing?

Dressing selections should also include an assessment of the patient's outcome of care. High-priced, inappropriate dressings are often used when a more cost-effective product would suffice. Outcome is commonly driven by institutional setting. Acute care patients whose length of stay is 4 to 5 days usually won't achieve healing as their outcome, but they will achieve a moist, clean wound bed that supports the healing environment. Home care and long-term care settings may have a goal of healing or maintaining the current status of the wound based on the overall health status of the patient. Wound outcomes need to be patient-focused and realistic with regard to the length of time a patient is cared for.

The clinician also needs to keep in mind that one of the primary goals of care is the prevention of wound-related infection. Infection is a common complication of all open wounds. Open wounds are colonized with bacteria, which means that low numbers of bacteria are always present on the wound surface.[20] Wounds that are critically colonized can be managed with antimicrobial or antiseptic dressings that provide sustained release of various agents, such as silver or cadexomer iodine.[21] If a wound fails to respond, the clinician should consider switching to a product with a different mechanism of action.[20] Reviewing the NICE model for dressing selection can help streamline your decision process.[17] (See chapter 7, Wound bioburden, for more information regarding wound infection and bioburden.)

The practice of using the same wound dressing during the entire healing period is no longer valid. *All* wounds under the care of clinicians should be assessed a minimum of once a week and more often if notable changes occur. The type of wound, status of the wound, clinical setting, and regulatory compliance may dictate a different interval of assessment, however. Be aware of practice regulations for your facility or institution. (See chapter 6, Wound assessment.)

Wound assessment is the cumulative process of observing the wound itself as well as observing the patient and collecting and evaluating data. For many patients, weekly reassessment will provide the indices of successful treatment and guide decisions that suggest product changes. As the wound characteristics change, so too should the choice of the wound dressing. Indeed, several different types of products may be needed as the wound progresses through the stages of healing.

Dressings should be matched carefully to the wound, the patient, and the setting. For example, a deep wound with a large amount of drainage will require a highly absorbent dressing such as alginate or foam. As the depth and amount of drainage decrease, a dressing such as a hydrogel, hydrocolloid, or film might be used.

Over the course of healing, the treatment plan will change as the wound is filled with granulation tissue and epithelialization occurs. Economic factors should also be considered when selecting dressings. The overall cost and effectiveness of treatment, cost of materials, nursing time, and frequency of dressing changes over time all impact the economic burden of care[22,23] (Table 9-5).

Table 9-5 Economic considerations for nurses

- Clean, rather than sterile, dressings and gloves can usually be used in the home for chronic wounds (refer to your agency's policy).
- Saline solution can be made at home by adding one teaspoon of salt to one quart of boiling water.[21]
- Dressings shouldn't be left open or at the patient's bedside.
- Cost of product selected and resources available for financial assistance should be considered.
- Frequency of dressing changes and cost-effective use of materials should be considered.
- Fistula management may use pouching vs. dressing.
- Nursing time per dressing change should be considered.
- The overall effectiveness of the treatment should be an economic consideration.

Adapted with permission from Baranoski, S., McIntosh, A., Montoya, L. Optimizing the use of dressing. Lecture, Clinical Symposium on Advances in Skin & Wound Care. Las Vegas, NV. September 2014.

The notion that all wounds are alike has also changed. An understanding of the etiology of the wound is essential for appropriate care.[21] Local wound care products as well as supportive care must be individualized for the particular wound. For example, a venous stasis ulcer might require a highly absorptive dressing as well as the necessary compression therapy. A variety of two- to four-layered compression bandages beyond the classic "Unna boot" are now available. Further, checking for ankle–brachial index and/or toe pressures using a Doppler is part of the total care of a patient with a peripheral vascular ulcer (see chapter 14) or history of diabetic neuropathies (see chapter 16).[17]

Using Wound Healing Biology to Select Treatment

An example of increased understanding of the "cellular biology" of wound healing and technology is the use of growth factors in wound care. All growth factors are proteins that are secreted by cells and have the ability to stimulate cell division, a positive action during the wound healing process.[24] Growth factors are now available derived either from a patient's

own platelets or in a drug form dispensed in a tube to apply to certain wounds. Research continues as to what combination, what quantity, and when growth factors will best enhance wound healing.

Yet another way technology is providing new options for wound management is in the use of tissue-engineered skin equivalents or substitutes for healing chronic wounds.

What the future holds for the use of gene therapy in wound healing is yet to be seen. The use of gene delivery or microencapsulation in advanced wound therapy is being investigated.[24]

Moist Wound Therapy and Dressing Options

The essential function of a wound dressing is to provide the right environment to enhance and promote wound healing. Research over the past 50 years has led to the generally accepted phenomenon that moist wound dressings create an optimal environment for wounds to heal faster and with less scar formation. The work of Orland[25] and Winter[4] led to the development of moist wound dressings as a clinical intervention to treat wounds.

George Winter is often cited as the father of moist wound healing. His laboratory work comparing the effect of air drying versus occlusive dressings on epithelialization in the animal model is generally considered a landmark study.[4,26] Fear of increasing infection with occlusive therapy slowed the development of moist wound therapy dressings, however, and 16 years passed between Winter's study and the development of what is considered the first moist wound therapy dressing, OPSITE. Continued research, clinician experiments, and interest in wound healing in general have led many companies to develop an assortment of moist wound treatment dressings. However, this has created a challenge for healthcare providers, who struggle to keep up with the ever-increasing number of new products and new technologies.

The following synopsis reviews the major dressing categories and provides helpful practice points on what, when, and how to use these dressings.

Transparent Film Dressings

Transparent film dressings, so named for their "see-through" properties, are thin polyurethane membranes. They are coated with an adhesive that allows them to adhere to the wound margins without sticking to the actual wound

(Fig. 9-3).

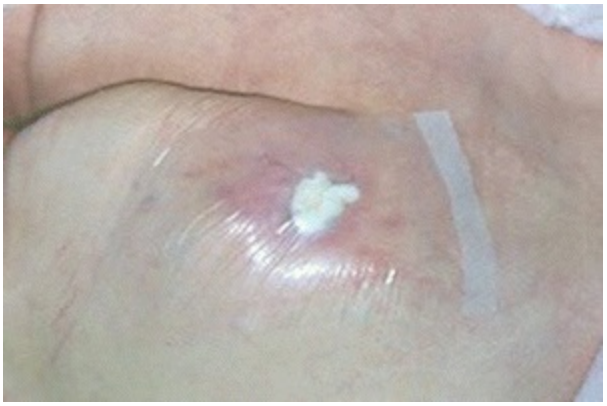


Figure 9-3 Transparent film dressing.

Transparent films have no absorptive capacity but do transmit moisture vapor and are semipermeable to gases. These dressings imitate the outer skin layer to provide a moist environment, similar to a blister. This covering allows epithelial cells to migrate over the surface of the wound. Fluid may accumulate under these dressings. This fluid is sometimes mistaken for pus, a sign of infection. The fluid is a useful adjunct to create an autolytic environment, thereby inducing a cleaner wound surface. When excess fluid accumulates or leaks out from the sides of the dressing, the dressing needs to be changed. Maceration of periwound skin can occur if these dressings are not changed in a timely manner.

Practice PointPractice Point

Don’t use film dressings with higher moisture vapor rates developed for use over IV line sites on open wounds.

Transparent film dressings provide a valuable protective barrier against outside contaminants, fluid, and bacteria. Transparent films also add a layer of protection to the wound bed to minimize further damaging trauma.[17] They provide protection from friction and aid in autolytic debridement and pain control. Film makes an excellent secondary dressing as well. Most films can be left on for up to 7 days. These dressings are indicated for wounds with absent or low levels of exudate.[7,8,17] Transparent films can be used on a variety of wound types, such as stage I and II pressure ulcers, superficial

wounds, minor burns, or lacerations; over sutures, catheter sites, donor sites, and superficial dermal ulcers; and for protection of the skin against friction. Transparent dressings can be used on central lines, peripherally inserted central catheter lines, and infected wounds.

In addition, some of the newer transparent films currently on the market also contain ionic silver.

Practice Essentials

- Apply transparent film dressings to healthy skin; use with caution on aging and fragile skin.
- These dressings *aren't recommended* for use on infants or small children.
- These dressings may be used on dry to minimally moist wounds; not recommended for skin tears.
- Don't use transparent film dressings on exudating wounds.
- Transparent film dressings make excellent secondary dressings.
- Not all film dressings can be used on infected wounds.
- Change the dressing when fluid reaches the edge of the dressing, when the seal is broken, or when the adhesive bond is compromised.
- When removing the dressing, lift the corner and pull the film toward the outside of the wound to break the adhesive barrier.
- Avoid roughness when removing the film; gently stretch the corner of the dressing and support the skin as you remove the dressing.
- Skin protective wipes and sprays can be used on the periwound area before applying the dressing. Skin wipes also provide an additional seal to prevent the dressing edges from rolling.
- Always read the package insert before applying the dressing because product usage may vary.
- Numerous sizes and shapes are available.

Practice PointPractice Point

Transparent film and hydrocolloid dressings are known for their ability to maintain existing moisture levels in the wound.

Hydrocolloid Dressings

Hydrocolloid dressings were introduced in the 1980s and were the mainstay for wound management for many years. Hydrocolloids are impermeable to gases and water vapor[27] and are composed of opaque mixtures of adhesive, absorbent polymers, pectin gelling agents, and sodium carboxymethylcellulose (Fig. 9-4). Hydrophilic particles within the dressing react with the wound fluid to form a soft gel over the wound bed. According to Choucair and Phillips,[27] some hydrocolloid dressings provide an acidic environment and some act as a bacterial or viral barrier. Their translucent appearance allows for viewing of the amount of exudate absorbed and fluid accumulated under the dressing.

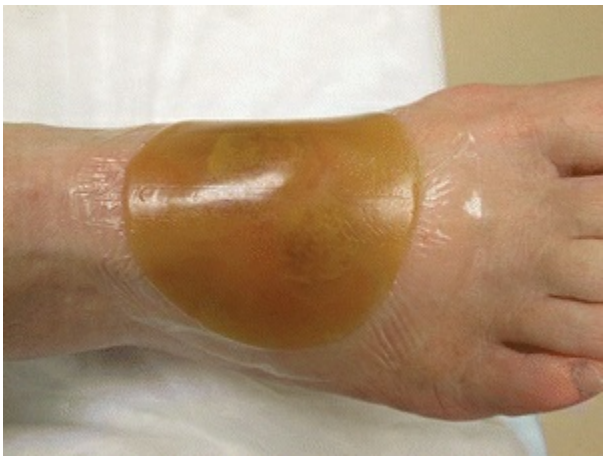


Figure 9-4 Hydrocolloid dressing.

Hydrocolloid dressings may have a noticeable odor during dressing changes. This is normal in the absence of clinical signs of infection. Some hydrocolloids may also leave residue in the wound bed.

Hydrocolloid dressings have evolved into a shape that fits most wounds and locations. They're sold in sheet, paste, and powder forms and are available in many sizes. Adhesive properties and ability to absorb exudate vary by product. Most of these dressings are adhesive so care must be taken when using on fragile skin. Correct application requires the dressing to be bigger than the actual wound size. For optimal dressing adherence, the dressing must extend at least 1″ (2.5 cm) onto the healthy skin surrounding the wound. The dressing should be changed as recommended by the manufacturer. This could be from 3 to 7 days and often depends on the amount of exudate. Some hydrocolloids have other absorptive ingredients added such as alginate and collagen,

Hydrocolloids are indicated for minimally to moderately heavy exudating wounds, abrasions, lacerations, pressure ulcers, dermal wounds,

granular wounds, or necrotic wounds as well as under compression wraps. Hydrocolloids do not absorb fluids rapidly, which is valuable when wound exudate levels are in transition from higher to lower quanitites.[9] Hydrocolloids also provide a moist environment that is conducive to autolytic debridement. Excessive granulation (hypergranulation tissue) and maceration can occur if the dressing isn't changed appropriately. Hydrocolloids are often used as a preventive dressing on high-risk areas (sacrum, heels) and around surgical wounds to protect the skin from frequent tape removal.

Practice Essentials

- Change the dressing every 3 to 7 days or before it reaches its maximal absorption or when fluid reaches within 1″ of the edge.
- Not all hydrocolloid dressings can be used on infected wounds. Check product insert.
- These dressings aren't recommended for undermining, tunnels, or sinus tracts.
- Hydrocolloid dressings may be cut to fit the wound area, such as on an elbow or heel.
- These dressings may be used as primary or secondary dressings or over other wound-filler products.
- Remove the dressing by starting at a corner and gently rolling it off the wound; don't pull to remove.
- Flush out any residue with saline or wound cleanser.
- Skin protective wipes or sprays may be used on the periwound area to enhance adherence.
- Picture-framing with paper tape may prevent the dressing edges from rolling.

Hydrogel Dressings

Hydrogel dressings have provided clinicians with a viable means to hydrate or, stated differently, donate moisture to dry wound beds.

Hydrogel dressings are marketed in multiple formats containing varying amounts of water in various polymer matrices. Hydrogels are available as amorphous gels, three-dimensional sheets, or amorphous gels impregnated into other mesh-type dressings (Fig. 9-5). Their unique cross-linked polymer structure entraps water and reduces the temperature of the wound bed by up to 5°C.[27] This moist environment facilitates autolysis and

removal of devitalized tissue.

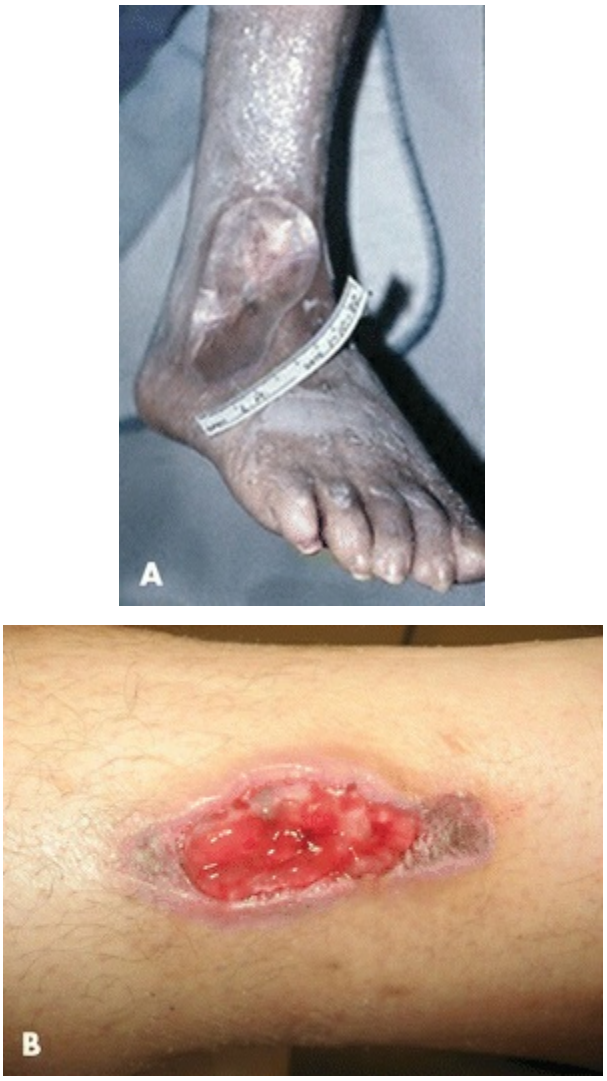


Figure 9-5. (A) Hydrogel dressing. **(B)** Amorphous hydrogel dressing. (Photos courtesy of S. Baranoski, A. McIntosh, and L. Montoya.)

The main application for hydrogels is hydrating dry wound beds and softening and loosening slough and necrotic wound debris. Hydrogels have a limited absorptive capacity due to their high water concentration. Some hydrogels have other ingredients, such as alginates, collagen, or starch, to enhance their absorptive capacity and will absorb low to moderate amounts of exudate. Absorptive capability varies by product and type of gel. They can be used for many types of wounds, including pressure ulcers, partial- and full-thickness wounds, and vascular ulcers. Their soothing properties also make them excellent choices for use in skin tears, dermabrasion, dermal wounds, donor sites, and radiation burns.

Maceration can be a concern for clinicians. Periwound skin areas need

to be protected from excess hydration; therefore, protective barriers are often recommended. One of the benefits of a hydrogel is that it can be used with topical medications or antibacterial agents. Hydrogels are packaged as sheets, tube gels, sprays, and impregnated gauze pads or strips for packing tunneling and undermined areas within the wound bed. Some require a secondary dressing to secure the hydrogel; new versions have adhesive borders. Some newer versions also contain ionic silver and can be left on for several days. Hydrogels with silver should not be used in combination with enzymatic debriding agents because the silver can inactivate the enzyme.

Practice Essentials

- Don't use hydrogels with heavily draining wounds or on intact skin.
- Daily dressing changes may be necessary due to evaporation of the hydrogel. Some sheet hydrogels may last for several days. Check daily to maintain a moist environment.
- Protect the surrounding skin with a skin barrier ointment, wipe, or spray.
- Watch for areas of maceration on surrounding skin.

Honey (Active Leptospermum)

Honey dressings are composed of medical-grade active Leptospermum or Manuka honey from New Zealand. These unique products have multiple mechanisms of action including reducing edema and promoting autolytic debridement. These products are available in various forms. Depending on the exudates level of the wound, different types of dressing may be indicated. It is available in gel, gauze, alginate, and as a hydrocolloid (Fig. 9-6).

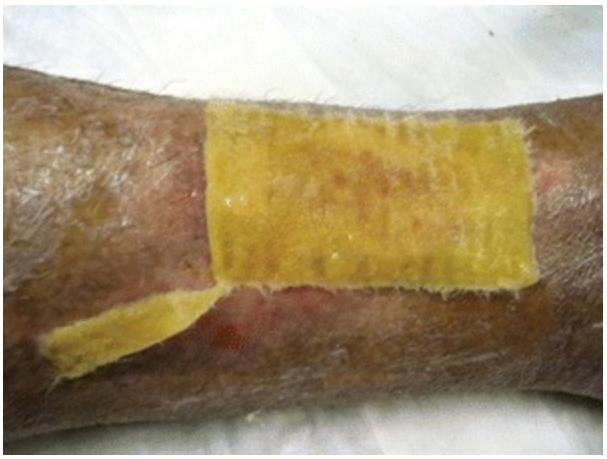


Figure 9-6 Honey dressing. (Photo courtesy of S. Baranoski, A. McIntosh, and L. Montoya.)

Honey dressings are indicated for infected or highly colonized wounds and wounds with slough or necrosis. It is also indicated for pressure ulcers stage II to IV, partial- and full-thickness wounds, and venous and diabetic ulcers. Honey dressings are contraindicated in patients allergic to honey and may have a brief stinging sensation.

Practice Essentials

- Honey dressings can be left on up to 7 days, depending on exudate and product.
- These dressings can be used to promote autolytic debridement.
- They may require a secondary dressing.
- Some patients may experience stinging sensation.

Foam Dressings

Foam dressings are highly absorbent and are usually made from a polyurethane base with a heat- and pressure-modified wound contact layer[28] (Fig. 9-7). Foam dressings are permeable to both gases and water vapor, and their hydrophilic properties allow for absorption of exudate into the layers of the foam.

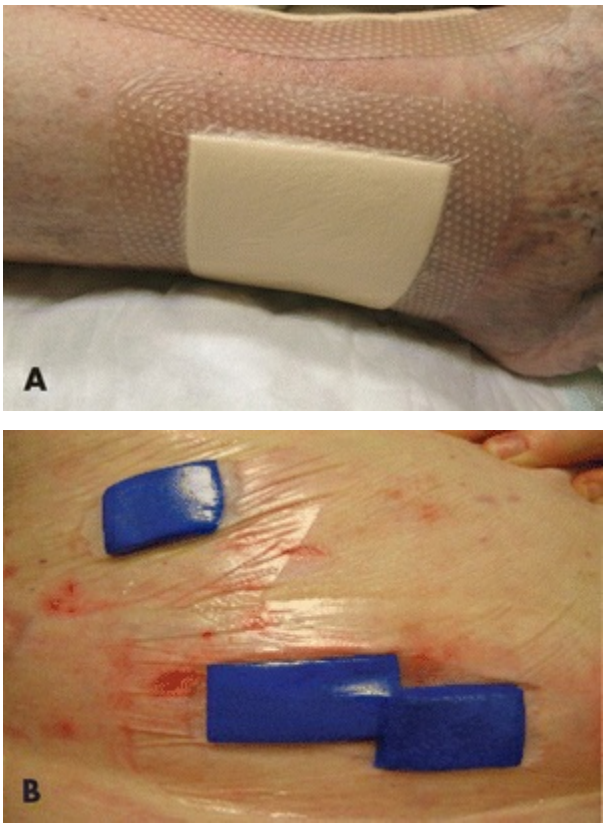


Figure 9-7. (A, B) Foam dressing. (Photos courtesy of A. McIntosh and L. Montoya.)

Foam dressings are some of the most adaptable dressings for wound care. They are indicated for wounds with moderate to heavy exudate, prophylactic protection over bony prominences or friction areas, partial- and full-thickness wounds, granular or necrotic wound beds, skin tears, donor sites, under compression wraps, surgical or dermal wounds, in combination with other primary dressings, and wounds of any etiology. They can also be used on infected wounds and can be left on up to 7 days, depending on product and exudates.[19] Foams are also available with controlled-released ionic silver. These dressings must have exudates for the silver ion to be released.

Foams shouldn't be used on dry eschar wound beds because they could cause further desiccation of the wound site. Foams may be used in combination with topical treatments and/or enzymatic debriders. Foams are available in many sizes and shapes, including cavity dressings. Not all foam dressings have an adhesive border, so they will need to be secured with tape. However, new foam products have emerged with adhesive borders. Caution with fragile skin may be warranted.

Practice Essentials

- Not all foams have FDA approval for use on infected wounds—be sure to check the package insert.
- Foam dressings can be left in place for up to 3 to 7 days, depending on the amount of exudate absorption.
- Removal of these dressings is usually trauma-free.
- Foam dressings can be cut to fit the size of the wound.
- Skin wipes or sprays can be used to protect the periwound area from maceration.
- Nonadhesive border dressings will require taping or wraps to secure.
- Make sure you put the *correct* side of the foam dressing in contact with the wound bed.

Calcium Alginate Dressings

Calcium alginate dressings provide yet another choice for clinicians to use in managing highly exudative wounds (Fig. 9-8). Alginate dressings are absorbent, nonadherent, biodegradable, nonwoven fibers derived from brown seaweed, composed of calcium salts of alginic acid and mannuronic and galuronic acids.[27,28]

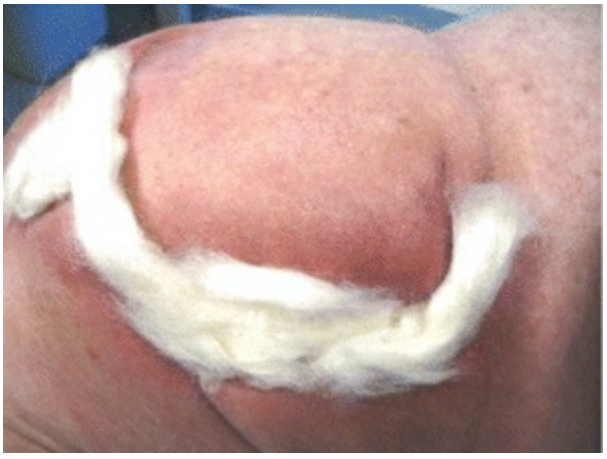


Figure 9-8 Calcium alginate dressing. (Photo courtesy of S. Baranoski, A. McIntosh, and L. Montoya.)

When alginate dressings come in contact with sodium-rich solutions such as wound drainage, the calcium ions undergo an exchange for the sodium ions, forming a soluble sodium alginate gel. This gel maintains a moist wound bed and supports a therapeutic healing environment. Alginates can absorb 20 times their weight; this may vary based on the particular

product. They are extremely beneficial in managing large draining cavity wounds, pressure ulcers, vascular ulcers, surgical incisions, wound dehiscence, tunnels, sinus tracts, skin graft donor sites, exposed tendons, and infected wounds. Additionally, their hemostatic and absorptive properties make them useful on minor bleeding wounds. Alginates are contraindicated for dry wounds, eschar-covered wounds, surgical implantation, or third-degree burns.

Alginates are available in sheet, pad, and rope forms and in numerous sizes. Some calcium alginate dressings also contain controlled-release ionic silver; these calcium alginate–silver dressings should not be used in combination with enzymatic debriding agents. Alginate dressings are usually changed daily or as indicated by the amount of drainage. One drawback is that these dressings can dry out and adhere to the wound bed. Using appropriate secondary dressings that help to maintain a gelling state is recommended.[9] Early wound care interventions may warrant more frequent dressing changes due to high volume of drainage. As fluid management is attained, the frequency of dressing changes can be decreased.

Practice Essentials

- Calcium alginate dressings provide easy application and trauma-free removal.
- These dressings are a good choice for undermined or tunneled draining wounds.
- Calcium alginate dressings require a secondary dressing.
- These dressings may leave fiber residue, which can be removed by flushing with saline.
- Calcium alginate dressings facilitate autolytic debridement.
- These dressings can be used on infected wounds, with or without ionic silver.
- These dressings are cost-effective if used appropriately.

Practice PointPractice Point

In order to release the silver ion, moisture/exudate must be present when controlled-release ionic silver is used in combination with other types of dressings (hydrocolloids, foams, etc.).

Composite Dressings

A combination of materials makes up a single-layered composite dressing. These dressings have multiple functions, such as a bacterial barrier, absorptive layer, foam, hydrocolloid, or hydrogel.[28] Additionally, they must have an adhesive border and semiadherent or nonadherent properties. These dressings are conformable and are available in numerous sizes and shapes.

However, not all composite dressings provide a moist environment; many are used or created by using a secondary dressing. They are also referred to as *island dressings*.

Practice Essentials

- Use composite dressings with caution when treating a patient with fragile skin.
- Composite dressings are easy to apply.
- These dressings may be used on infected wounds and with topical products.
- They may facilitate autolytic or mechanical debridement.
- Frequency of dressing change depends on the wound type and the manufacturers' recommendations.
- These dressings may adhere to wound bed; use caution when removing them.

Collagen Dressings

Collagen is a major protein of the body and is necessary for wound healing and repair. Collagen dressings are derived from bovine or ovine. Collagen dressings either are 100% collagen or may be combined with alginates or other products. They are a highly absorptive, hydrophilic, moist wound dressing (Fig. 9-9).

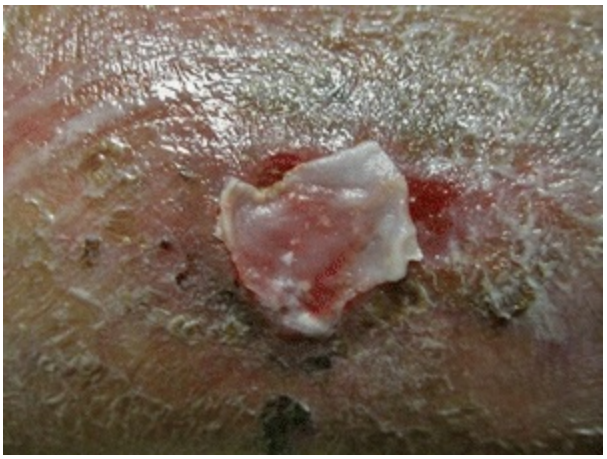


Figure 9-9 Collagen dressing. (Photo courtesy of A. McIntosh and L. Montoya.)

Seaman[19] suggests that collagen powders, particles, and pads are useful in treating highly exudative wounds. If the wound has low to moderate exudate, sheets should be used. If the wound is dry, gels should be used. Collagen dressings can be used on granulating or necrotic wounds and on partial- or full-thickness wounds.[19] They may be used with other topical agents.

A collagen dressing should be changed every 3 to 7 days. Collagen dressings require a secondary dressing for securement.

Practice Essentials

- Use collagen dressings with caution when treating patients with fragile skin if adhesive secondary dressings are also being used.
- These dressings are contraindicated for patients who are sensitive to bovine or ovine products.
- Don't use these dressings on dry wounds or third-degree burns.
- Collagen dressings are easy to remove.
- Their gel properties prevent these dressings from adhering to the wound bed.
- Collagen dressings facilitate a moist wound environment and may be used with other topical products.

Contact Layer Dressings

Contact layer dressings are a single layer of a woven net that acts as a low-adherence material when applied to wound surfaces.[28] A contact layer dressing is applied directly to the wound and acts as a protective interface

between the wound and the secondary dressings. Their main purpose is to allow exudate to pass through the contact layer and into the secondary dressing. They are often used with ointments, creams, or other topical products. Contact layer dressings are not recommended for dry wounds or third-degree burns. Check the package insert for clarification as to which wounds the product can be used on. Various sizes and shapes are available. Frequency of dressing changes is dependent on the etiology of the wound and the amount of exudate.

Practice Essentials

- Contact layer dressings are not recommended for dry wounds or third-degree burns.
- Contact layer dressings are easy to apply and are secured with a secondary dressing.
- They protect the wound bed during dressing changes.

Antimicrobial Dressings

Antimicrobial dressings have added another dimension to the wound dressing arena. Clinicians now have several choices of dressings when dealing with wound infections. These new dressings are different than topical antibiotic therapy. They provide the benefit of an antimicrobial effect against bacteria and a moist environment for healing. The active ingredient may be silver ions, cadexomer iodine, or polyhexamethylene biguanide (Fig. 9-10). Antimicrobial dressings do not replace the need for systemic antibiotic therapy; rather, they serve as an adjunct in treating wound infections. As research continues and new products become available, this classification of wound dressing will expand. Antimicrobial dressings are available in a variety of forms: transparent dressings, gauze, island dressings, foams, and absorptive fillers, to name a few. Some of these dressings can remain in place for 7 days.

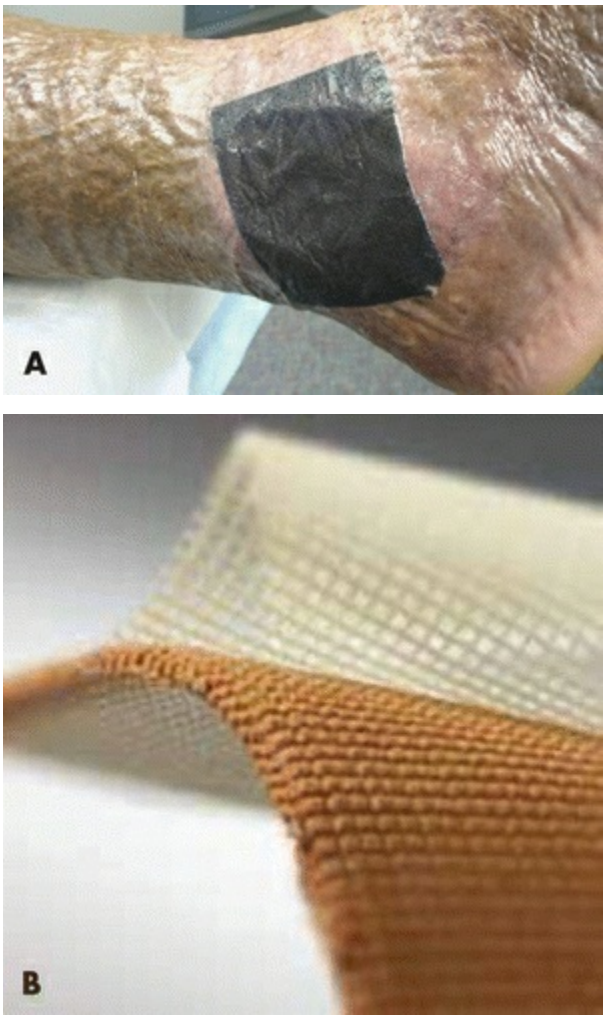


Figure 9-10 Antimicrobial dressings. **(A)** Silver dressing. **(B)** Cadexomer iodine dressing. (Photos courtesy of A. McIntosh and L. Montoya.)

Practice Essentials

- Antimicrobial dressings are an adjunct in treating wound infections.

Advanced Therapies

Advanced therapies (also referred to as advanced or adjunctive interventions or modalities) have been defined as mechanical, biological, or pharmaceutical modalities. These are utilized when standard basic wound care protocols have failed to produce demonstrable improvement in wound healing, as evidenced by poor granulation development and a flattened wound healing trajectory.[29] These advanced interventions are utilized to promote wound healing via multiple mechanisms including stimulating angiogenesis and enhancing tissue perfusion. It is important to understand

that advanced therapies are not typically used as initial or first-line strategies but rather are incorporated into the wound care plan using well-defined guidelines and algorithms.

Based on data published by Dr. Peter Sheehan, wounds that do not demonstrate greater than 50% reduction in size after 4 weeks of basic wound care are unlikely to heal.[30] Patients presenting for wound care consultation should receive aggressive basic wound management based on their initial workup and evaluation. Basic wound care typically includes debridement, control of bioburden and infection, offloading and pressure relief, edema control, and moist wound healing. Patient progress and response are then followed closely. If at 4 weeks the wound fails to show at least a 50% decrease in wound size, advanced therapies should then be considered. This algorithm has been suggested by Expert Consensus Panels and adopted by multiple Professional Wound Care Societies as well as included in National Coverage Determinations.[31]

The list of advanced modalities is vast in number and is constantly changing based on advances in technology and our understanding of the basic tenants of the physiology of healing and wound care. The following sections are not intended to be complete nor exclusive but rather provide a representative overview of examples of several therapies in each category. The listing of an advanced therapy in one category or another is provided only within a suggested organizational framework for educational purposes.

Mechanical

A large number of devices and therapies can be considered in the group of mechanical modalities. Included in this category are negative pressure wound therapy (NPWT), compression therapy, and energy-based therapy (electrical stimulation, light, and ultrasonic). Mechanical therapies impact wound healing via several mechanisms including angiogenesis, cellular stimulation, bioburden reduction, and enhanced tissue perfusion.[32]

Negative Pressure Wound Therapy

NPWT is in its third decade of use in the United States and has become an accepted therapy for many chronic and, in some cases, acute wounds. It is commonly used to assist with wound closure. In general, NPWT is indicated for full-thickness wounds that require contraction and granulation tissue formation. Some NPWT systems are appropriate for tunnels, tracts, and undermining (Table 9-6). Reduction of wound volume is one of the most

widely recognized outcomes from using NPWT. The National Pressure Ulcer Advisory Panel (NPUAP), European Pressure Ulcer Advisory Panel (EPUAP), and Pan Pacific Pressure Injury Alliance (PPPI) recommend the use of this modality for the treatment of deep, category/stage III and IV pressure ulcers.[35]

Table 9-6 Indications, Contraindications, and Precautions for Using NPWT[33,34]

Indications

- Diabetic foot ulcers
- Pressure ulcers (stages III and IV)
- Venous insufficiency ulcers
- Arterial insufficiency ulcers
- Full-thickness burns
- Surgical wounds (especially infected sternal wounds)
- Postoperative and dehisced surgical wounds
- Traumatic wounds
- Explored fistulas
- Skin grafts and flaps

Contraindications

- Exposed vital organs (treatment may continue after the organ has been covered by protective mesh)
- Inadequately debrided wounds (granulation tissue that will not form over necrotic tissue)
- Untreated osteomyelitis or sepsis within the vicinity of the wound
- Presence of untreated coagulopathy
- Necrotic tissue with eschar present
- Malignancy in the wound (negative pressure therapy may lead to cellular proliferation)
- Allergy to any component required for the procedure

SPECIAL CONSIDERATIONS Before Using NPWT

- High risk for bleeding and hemorrhage (i.e., anticoagulation therapy, platelet aggregation inhibitors, bleeding disorders)
- Patients with:
  - friable vessels and infected blood vessels
  - vascular anastomosis
  - infected wounds
  - osteomyelitis
  - exposed organs, vessels, nerves, tendon, and ligaments
  - sharp edges in the wound (i.e., bone fragments)
  - spinal cord injury (stimulation of sympathetic nervous system)
  - enteric fistulas
- Patients requiring:
  - MRI
  - hyperbaric chamber (electrical component may not be taken inside the chamber)
  - defibrillation
- Patient size and weight
- Use near vagus nerve (bradycardia)
- Circumferential dressing application
- Mode of therapy—intermittent vs. continuous negative pressure

NPWT applies subatmospheric pressure, or suction, to the wound bed by way of a device that is attached to a wound contact layer (interface dressing) through a plastic tube (Fig. 9-11). Several types of interfaces are used with these devices, including foam, moistened cotton gauze, and

nonwoven polyester layers joined by a silicone elastomer.[36,37] Most clinicians have migrated to the use of foam interfaces based on adverse outcomes that have been reported with gauze.[38] The interface dressings are covered with a transparent film or thin hydrocolloid, depending on the device used, that seals the wound and dressing to maintain the vacuum effect. Interface dressings containing silver or other antibiotics are available from some manufacturers. Manufacturers recommend initially changing the dressing 48 hours after beginning treatment, then two to three times per week as indicated by the wound's response to the NPWT intervention.[36,37,39–43]

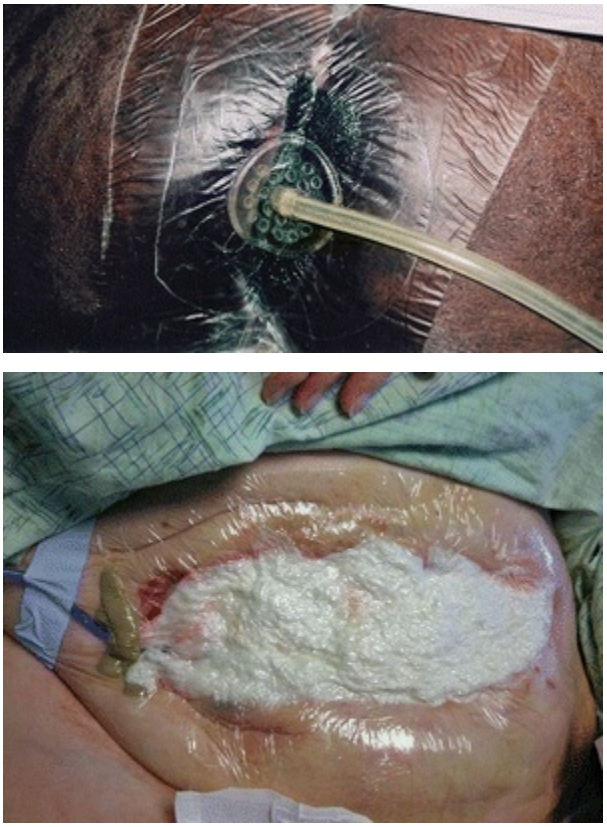


Figure 9-11 Negative pressure wound therapy. (Photos courtesy of S. Baranoski, A. McIntosh, and L. Montoya.)

There are two types of NPWT devices: (1) electrical or battery powered and (2) mechanically powered. The electrical devices were the first type launched and adapted into clinical practice and deliver negative pressure via an electrically or battery-powered pump. Once the wound is cleaned and debrided, the dressing is applied. An evacuation tube runs from the wound through the interface dressing, drawing excess exudate away from the wound and into a canister attached to the other end of the tubing.

The canister is attached to the vacuum pump providing either continuous or intermittent negative pressure, adjusted for the type of wound being treated. Negative pressure created by the pump is in the range of 0 to 200 mm Hg depending on the system used.[39–41] A mechanically powered NPWT device was recently developed using a coiled spring to provide the energy to create the negative pressure. This device has a cartridge providing two functions: (1) contains the spring, which generates continuous negative pressure, and (2) stores fluids removed from the wound (Fig. 9-12).

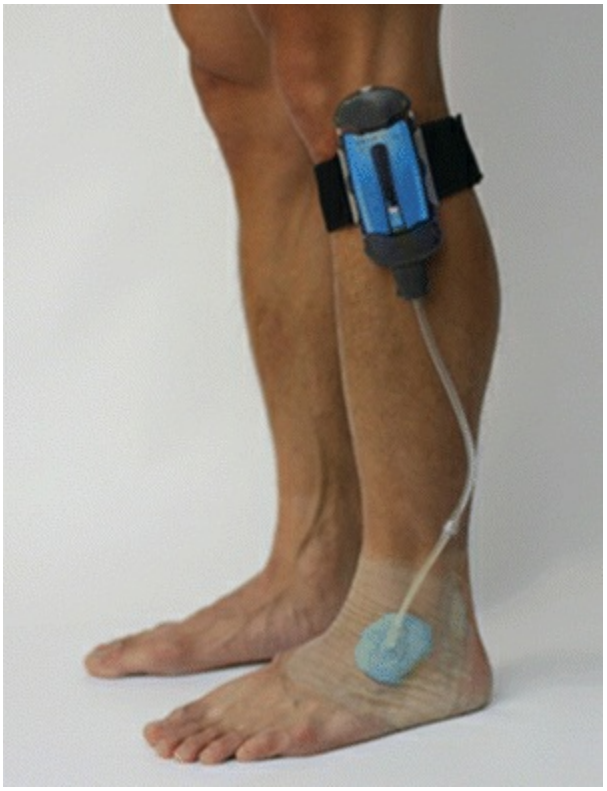


Figure 9-12 SNaP unit on a mobile highly functional patient. (Courtesy Spiracur Inc.)

Comparison studies between the two types of units, electrically powered and mechanical (coiled spring) powered, have been performed in clinical practice. The results of these studies demonstrate no wound closure advantage or disadvantage of one type of power delivery system over the other.[44–46] In general, deciding which unit to use will be determined by the size of the wound, exudate amounts, and patient convenience and comfort. For instance, large wounds with high exudate levels will need the larger canisters found with electrically powered units, whereas smaller wounds with little exudate, especially in very mobile patients leading highly functional lives, may be appropriate for the mechanically coiled spring unit.

The application and type of dressing vary by the specific manufacturer and the goals for use of the device. NPWT reportedly facilitates wound closure and healing through several mechanisms of action, including:[35–37,39–41]

- facilitating wound contraction/retraction
- removing edema, which improves nutrient and oxygen delivery
- removing wound exudate, which may be a medium for bacterial colonization
- decreasing harmful levels of proinflammatory agents, such as matrix metalloproteases (MMPs), found in chronic wounds
- promoting angiogenesis.

One theory as to how NPWT enhances wound closure is that the externally applied stress creates macrostrain and microstrain in the wound bed and in individual cells.[47–50] Macrostrain is the physical response that can be seen immediately as the negative pressure contracts the wound.[47] In addition, this mechanical stress creates changes at the cells' surface (also known as microdeformations), causing growth factors and cytokines to "upregulate" fibroblastic activity, increasing production of the extracellular matrix (ECM) and cell proliferation within the wound and ultimately creating new granulation tissue.[47–50] This increase in the rate of granulation tissue formation has been noted in several studies using NPWT.[47–50]

According to the NPUAP,[35] wound volume was significantly reduced and healing was more rapid in pressure ulcers treated with NPWT compared with other traditional wound treatments.

It has been suggested that NPWT assists in preparing the wound bed for closure once granulation tissue has been well established.[50] When a wound is filled with newly synthesized granulation tissue, resurfacing by epithelial cells can take place more readily.[50]

It is important that the wound be debrided of as much necrotic tissue as possible before using the NPWT device. The exact percentage of necrotic tissue that can be present in the wound bed varies according to manufacturer as well as facility and clinician protocols. In general, it is best to have the wound bed *free* of necrotic tissue prior to application of the NPWT device.

Many clinicians are now combining NPWT with the instillation of infusion of solutions into the wound interface. A variety of solutions have been reported and include normal saline, hypochlorous acid, and other antimicrobials, which serve to enhance wound irrigation and cleansing as

well as decrease bacterial colonization across the surface of the wound. Some manufacturers have provided devices that have been modified to allow this technique, while some clinicians report excellent outcomes with inexpensive simple dressing modifications that they customize themselves. Patients can easily manage this independently in the outpatient setting using the Modified Intermittent Therapeutic Infusion Technique[51,52] (Fig. 9-13). Safety must be taken into consideration when using these devices. In November 2009, the FDA released a Preliminary Public Health Notification Safety Alert[33] describing deaths and serious complications associated with the use of NPWT systems. The FDA had received reports of six deaths and 77 injuries associated with NPWT systems over a 2-year period in relation to bleeding, primarily in conjunction with anticoagulation therapy or infections. Since the publication of this 2009 report, there have been reports of three additional deaths in patients receiving NPWT. The majority of these injuries and deaths occurred in the home or nursing home environment. The excessive bleeding occurred in individuals with blood vessel grafts in the leg and breastbone and groin wounds, patients receiving medication for blood clots, and during wound dressing removal that was attached to the tissues (Fig. 9-14). Worsening wound infection, primarily from retention of dressing pieces, was reported 27 times.[34]

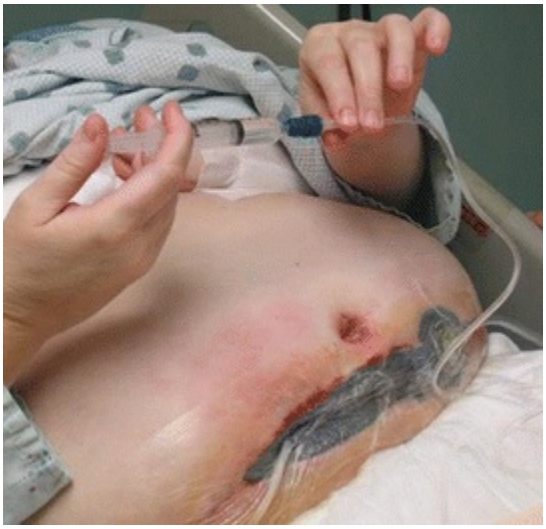


Figure 9-13 Modified Intermittent Therapeutic Infusion (MITI NPWT Technique). (Photo courtesy of J.A. Niezgoda.)

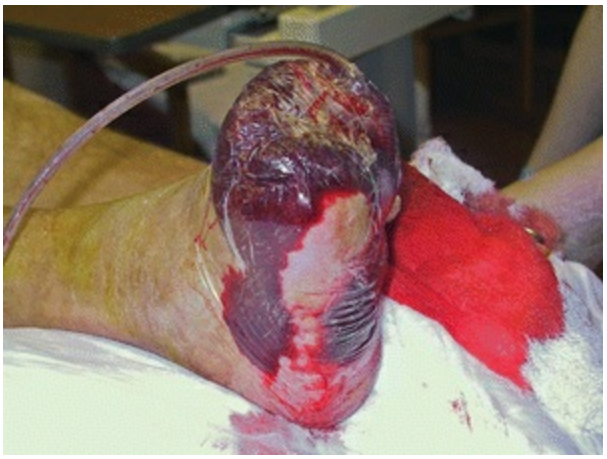


Figure 9-14 NPWT complication from bleeding. (Photo courtesy of J.A. Niezgoda.)

Practice PointPractice Point

NPWT should be used cautiously when there is active bleeding, when the patient is on anticoagulants, when there is difficult wound hemostasis, or when placing the dressing in proximity to blood vessels.

Practice Essentials

- Device operation and utilization should be performed according to the specific manufacturer's directions.
- NPWT is not a substitute for wound debridement, and wounds treated with NPWT should be well debrided prior to initiation of NPWT.
- Application of skin prep or sealant to the periwound area may be helpful in preventing blistering, denuding, and excoriation when removing the adhesive drape.

Compression Therapy

Venous Compression Therapy

Compression therapy is the foundation for successful management in patients with edematous wounds caused by venous insufficiency and/or

lymphedema. Compression therapy wraps are used to manage fluid accumulation and promote sufficient return of venous blood back to the central system and lymph back into the bloodstream. The substances transported by the lymphatic system are called *lymphatic loads* and consist of protein, water, cellular debris, and fat from the digestive system. These loads are filtered by the regional and central lymph nodes prior to re-entry into the venous system.[53]

It is worth noting that the edema associated with venous insufficiency is different in consistency from the edema or fluid accumulation that is seen in lymphedema. Protein-rich lymphedema fluid appears to be more "viscous" or thicker than the edema associated with venous insufficiency and requires different treatment interventions, including higher compression forces when using wraps or garments. (See chapter 14, Venous Disease and Lymphedema Management.)

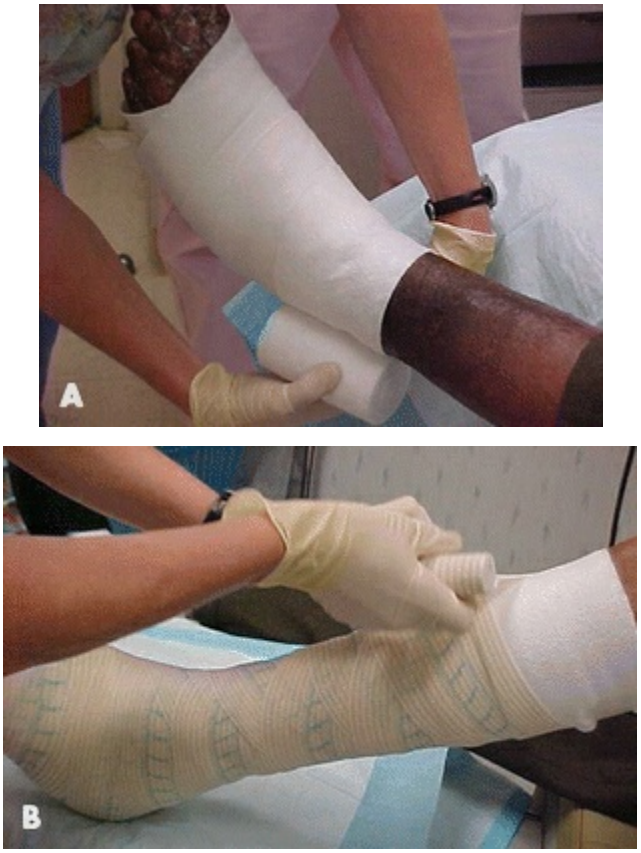
Compression Dressings

Several types of compression wraps are available, and instructions on their application techniques vary. Some have layers that are applied in a spiral fashion, while others are wrapped in a figure-eight configuration, and still others use a combination of the two wrapping techniques. Bandages are also classified by their elasticity. Short-stretch bandages have a smaller degree of stretch than do long-stretch bandages. Short-stretch or rigid compression systems are particularly suited to managing the fluid accumulation of lymphedema and are also indicated for patients with a combination of venous insufficiency and lymphedema in the same leg.[54] An Unna boot is a "short-stretch" system that includes a moist layer impregnated with several substances, including zinc oxide, calamine, and gelatin. This layer molds to the extremity, becoming semirigid when dried and creating high working pressures during ambulation; this improves the calf pump function to facilitate fluid return to the central system and decrease edema in the afflicted extremity.

Practice PointPractice Point

When using an Unna boot, apply a nonadherent dressing over the open wound first so as to avoid damage to the healing tissue when removing the compression dressing.

Long-stretch bandages are so called due to their large amount of extensibility and elastic recoil back to near-original configuration. While short-stretch bandages require the patient to be ambulatory or able to engage the calf muscles effectively (ideally by walking), long-stretch bandages have been shown to be suitable for individuals who are not active or who are nonambulatory.[54] While 78% of nurses who responded to a survey indicated that compression wrapping /bandaging multilayer system is the gold standard for treating venous ulcers, only 68% indicated that they know how to apply these compression wraps.[55] Clinicians need to be trained and skilled to proficiently and safely apply these compression wraps, keeping in mind that they should be applied according to the manufacturer's detailed directions[56] (Fig. 9-15).



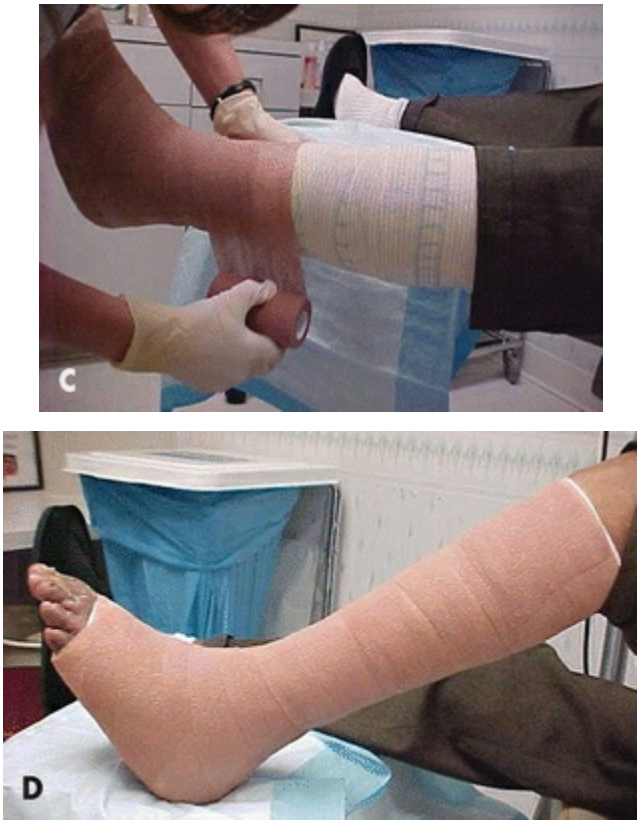


Figure 9-15 Application of a multilayer compression system. **(A)** Absorbent padding applied to leg. **(B)** Application of a light conformable dressing applied in a spiral manner. **(C)** Flexible cohesive bandage applied over the two other layers, also in a spiral manner. **(D)** Completed compression wrap.

Compression Devices

Some compression systems do not fall into the wrap or bandage category. These include garments that are usually short-stretch systems consisting of material that does not give way during ambulation; these systems often include Velcro straps to help secure and conform the garment to the extremity contours (Fig. 9-16). Application techniques vary according to product; it is important that the clinician read the package insert before applying the compression system.

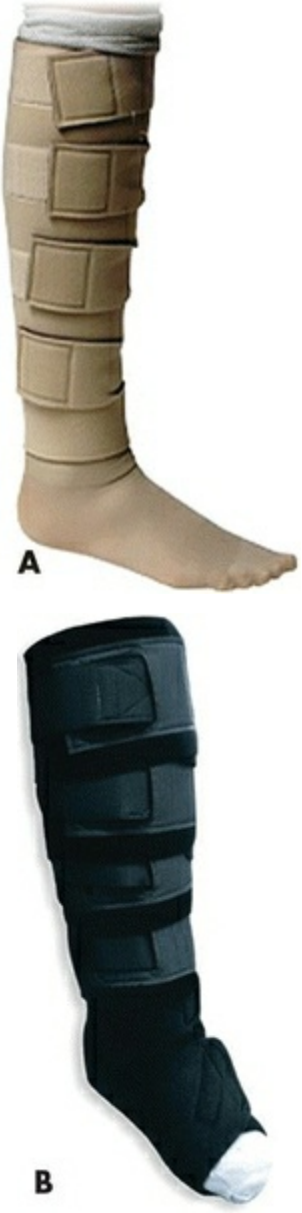


Figure 9-16. Short-stretch bandages, such as the CircAid **(A)** and CompreFit BK **(B)**, do not give way when the calf muscle pump is exercised. The force of the muscle is directed back into the leg and promotes venous return.

Intermittent Pneumatic Compression

Intermittent compression therapy (IPC) or external pneumatic compression therapy contributes to the healing of venous insufficiency ulcers by collapsing the superficial venous system and forcing blood into the deep system, thus increasing subcutaneous pressure. IPC prevents the leakage of blood, fibrin, and protein from the skin capillaries. In addition to the hemodynamic effect of IPC, enhancement of fibrinolysis has been an important outcome of this intervention.[57–59] IPC results in improved

circulation with an increase in oxygenated blood flow and the removal of potentially harmful toxic waste from the wound and periwound area.[59]

Application of IPC to the lower extremity is best accomplished with the patient positioned supine and the extremity elevated above the heart. Treatment time varies but usually lasts from 45 to 60 minutes.

IPC is contraindicated for patients with acute deep vein thrombosis or severe peripheral vascular disease. Although not an absolute contraindication, vigilant observation and communication with the patient who has a history of congestive heart failure must be practiced when using any type of compression system. This is important in order to ensure that the heart is not overloaded with the increased fluid return back to the central system.[59] There is some controversy regarding the use of IPC in patients with lymphedema due to concerns for damaging the superficially located lymphatic anatomical structures.

Practice PointPractice Point

- Compression therapy should be applied cautiously and at lower levels of compression (20 mm Hg) when there is evidence of mild to moderate peripheral arterial disease.
- If a patient presents with mixed etiology of venous and arterial insufficiency, he or she should be monitored diligently to ensure that arterial compromise is not occurring.

Vascular assessment and an ankle–brachial index Doppler study are critically important prior to the use of compression therapy dressings or devices. (See chapter 14, Venous Disease and Lymphedema Management.)

Arterial Compression Therapy

Compression therapy is generally associated with venous insufficiency; however, compressive devices have also been successfully utilized for patients with arterial disease. Pneumatic compression therapy modalities are external limb devices that apply an end-diastolic compressive force to the involved extremity, which enhances distal blood flow and tissue perfusion (Fig. 9-17). These devices also improve venous return and tissue

edema, which in turn enhances tissue perfusion by decreasing the extrinsic compression of the arterial vasculature by edema-associated pressure. Multiple studies have demonstrated symptom improvement and enhanced wound healing via vascular augmentation as evidenced by improved transcutaneous oxygen measurements, ankle-brachial indexes, toe pressure, and pulse volume recordings. Arterial pneumatic compression has gained acceptance for patients with end-stage arterial vascular disease or for those with failed attempts at vascular reconstruction.[60,61] Improved clinical outcomes have also been reported when using arterial compression therapy in combination with hyperbaric oxygen therapy (HBOT).[62]

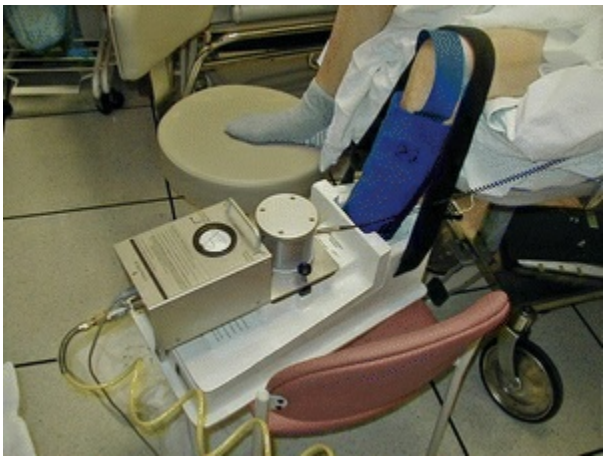


Figure 9-17 Arterial assist compression device. (Photo courtesy of J.A. Niezgoda.)

Energy-Based Therapy

This section discusses various types of energy-based technologies and the application of these devices when used to enhance wound closure and healing. All energy-based therapies should be used in conjunction with appropriate moist wound healing practices according to the advanced therapy algorithm previously discussed. The application of these exogenous energies has been shown to augment cellular, tissue, and vascular responses in both acute and chronic wounds.[35]

Practice PointPractice Point

- NPUAP Statement: Biophysical (energy-based therapy) agents

should be directed by and under the supervision/management of a skilled licensed professional educated and trained in safe and effective selection, application, and monitoring methods for these devices.[35]

Clinicians are advised to refer to Local Coverage Determinations regarding level of evidence and reimbursement issues related to individual energy-based therapies discussed in this section.

Electrical Stimulation

In the 18th and 19th centuries, when Galvani [34,63] and Matteucci[64] made preparations of cut sciatic nerve and muscle tissue from an excised frog leg, they found that small electrical currents were generated at the cut points. The electric potentials associated with these cut points are now referred to as *injury potentials*. In 1860, German physiologist Emil Du Bois Reymond[65] measured a direct current flow out of a cut in his finger and observed the disappearance of these currents when the wounds healed. This theory of this physiological response to injured skin is termed the *"current of injury."* The reason this is important in chronic wound closure and healing is that electrical stimulation (ES) can facilitate *"restarting"* the stalled wound healing sequela of events.

Electrical stimulation has been used for more than three decades to accelerate the rate of chronic wound healing. This energy has been given strength of evidence of A by the NPUAP[35] as an adjunctive therapy for treatment of recalcitrant stage III and IV pressure ulcers. Healthcare professionals who have used ES consider it to be one of the most cost-effective, therapeutically efficacious tissue repair and wound healing accelerators in our wound care tool kit.

Practice PointPractice Point

Electrical stimulation facilitates *"restarting"* chronic wounds that have stalled in the healing processes.

Unfortunately, ES is not widely used due to nonstandardized clinical algorithms combined with the lack of knowledge, education, and training in

the application of this energy.[66] In 2014, the Paralyzed Veterans of America published a Clinical Practice Guideline titled Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury, [67]which stated that ES qualified as a *"stand-alone intervention"* and should no longer be classified as an adjunctive therapy. The reported effects of ES in relation to chronic wound healing and tissue repair include increased blood flow,[68] increased tissue oxygenation,[69,70] increased fibroblast proliferation and collagen deposition,[65] increased angiogenesis,[71] decreased wound pain,[72] increased wound tensile strength,[73,74] and decreased diabetic peripheral neuropathic pain.

ES energy may be considered for treatment of lower limbs with severely compromised arterial blood flow. ES energy delivered as high-voltage pulsed current (HVPC) applied to ischemic lower extremities has been shown to increase transcutaneous pulse oximetry and perfusion, with subsequent healing of these legs and feet in patients who were at high risk for amputation.[65]

The application of ES in wound healing includes therapeutic exogenous (externally applied) electrical currents. These currents are delivered to the wound tissues by using at least two electrodes, one of which may be applied directly into or both around the wound (periwound tissue), or by applying a stocking or glove electrode garment to the affected limb (Fig. 9-18).

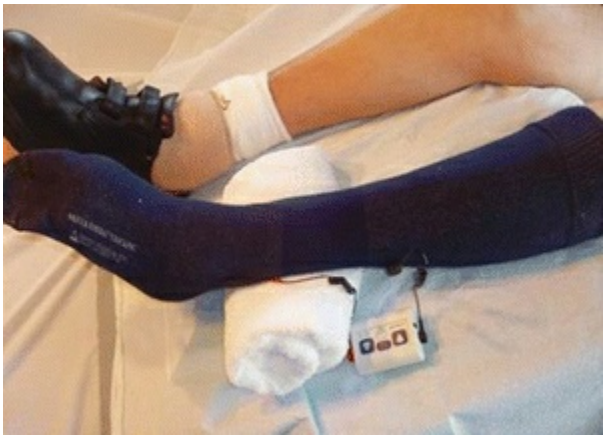


Figure 9-18 Electrical stimulation with use of a stocking electrode garment. (Photo courtesy of P. Scarborough.)

ES uses an electrical current to transfer energy to the tissue. This energy produces a number of cellular processes and physiological responses that are important to wound healing, including:

- stimulation of fibroblasts to enhance collagen and DNA synthesis
- increase in the number of receptor sites for growth factors
- alteration in the direction of fibroblast migration, activation of cells in the wound site, improved tissue perfusion, and decreased edema.[75]

The electrical current may be delivered as low-intensity direct current, HVPC, transcutaneous electrical nerve stimulation, or pulsed electromagnetic energy. However, HVPC has become the current used most often for ES wound treatments in the past decade.

ES is performed by physical therapists or other licensed healthcare professionals who are educated and trained in identifying how and when to apply and change the ES treatment parameters (e.g., ES dosage, polarity, electrode placement) according to the wound characteristics. According to Myer,[76] HVPC has a waveform of paired short-duration pulses with a long interpulse interval. It's a pulsed or interrupted monophasic waveform. The duration of treatment is usually 45 to 60 minutes, delivered five to seven times per week. ES is indicated for all types of wounds regardless of the etiology. It is contraindicated for basal or squamous cell carcinoma in the wound or periwound tissue, for osteomyelitis (if the patient is not responding to systemic treatment with antibiotics), for ion residues of iodine or silver in the wound, and over electronic implants or directly over the carotid arteries or heart.[66]

The decision-making process for how to use ES in a specific wound depends on the phase of wound healing and what the clinician is attempting to accomplish. ES delivers a summation of either a positive charge or a negative charge to the tissues. Therefore, it is important to consider what specific outcome the clinician is attempting to create. For instance, if treating an infection is important, the current selected would be a summation of a positive charge to attract the negatively charged neutrophils, whereas if the goal is to facilitate the proliferative phase for angiogenesis and granulation tissue formation, one would want a summation of the negative current to attract the positively charged fibroblast.

Practice PointPractice Point

- Don't place ES electrodes over the carotid sinus, close to the heart, or near the laryngeal musculature.

- When using silver products, ensure that the residue has been well irrigated from the wound prior to using ES.

Light Therapy

Light is a form of electromagnetic radiation (EMR). Sunlight has been known for many centuries to have beneficial effect on humans. The Egyptians, Greeks, and Chinese all have writings referring to the use of sunlight to treat different conditions and illnesses. Sunlight is still prescribed for certain illnesses today. Most of the time, however, light therapy, or phototherapy as it is known in the healthcare field, involves the application of specific wavelengths to patients using various medical devices.[77]

Light comes in different colors, spreading across the rainbow of hues known as the visible spectrum. Each color in this spectrum corresponds to a different wavelength. The visible light spectrum ranges from short wavelengths of the violet or purple color to long wavelengths, which are red. Photons of light from the violet end of the spectrum have the highest energies and the highest frequencies, while red photons have lower energies and lower frequencies. Beyond the range of our human vision are the longer wavelengths of the infrared and the shorter wavelengths of the ultraviolet regions of the electromagnetic spectrum (EMS).

Low-Level Laser Therapy

When discussing phototherapy in wound care today, one is usually referring to a low-level or cold laser, which is used for tissue regeneration, as opposed to coherent laser beams of high-intensity light, which is used for surgical incisions. While laser therapy for tissue regeneration is still in its infancy in the United States, it has been used in Europe and Russia for more than 30 years as an adjunctive modality for wound healing. “Laser” is an acronym for light amplification by stimulating emissions of radiation. Low-level laser therapy (LLLT) or low-intensity laser therapy (LILT) is also known as *cold laser therapy*, *photobiomodulation*, or *monochromatic infrared light therapy*. Laser light is always one single color (therefore monochromatic) and is in the infrared (nonvisible) area of the light spectrum. Several studies have demonstrated a positive effect of LLLT on the three overlapping phases of wound healing: inflammation, proliferation, and remodeling. The proposed outcome of LLLT is more rapid wound

healing. Dyson and colleagues[78–85] have suggested that this modality can reduce the inflammatory phase, cause earlier initiation of the proliferative phase, and augment the rate of contraction as angiogenesis increases.

More than 2,000 scientific, peer-reviewed studies on the uses of LLLT have been published worldwide. Specific topics have included chronic wound healing, acute soft tissue injuries, shingles, regeneration for both nerve injuries and diabetic peripheral neuropathy, and reducing postoperative pain. There are also a significant number of research articles describing the effects of LLLT on keratinocytes, mast cells, and macrophages and fibroblasts, all of which are critical to the wound healing processes.

Light is applied by a multicluster diode probe (Fig. 9-19), a single diode probe, or a multicluster diode pad. The literature and anecdotal reports indicate that most chronic wounds, including diabetic neuropathic foot ulcers, venous insufficiency, arterial insufficiency, and pressure ulcers, respond well to this treatment intervention.[86–96]

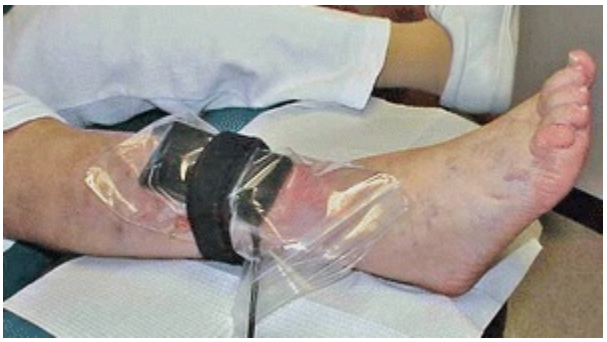


Figure 9-19 Multicluster diode probe for use in low-level laser therapy for wound healing. (Photo courtesy of P. Scarborough.)

Practice PointPractice Point

When treating with LLLT directly over or in an open wound, apply a thin plastic transparent barrier over the wound or place the pad(s) or probe in a plastic bag to keep them free of contaminants.

Ultraviolet Light

Ultraviolet light (UV) has been used for centuries to treat a myriad of health and skin problems in the form of natural sunlight and more recently by

artificial UV-generated sources. Ultraviolet light A (UVA) and ultraviolet light B (UVB) are responsible for the pigmentation and erythema (and sometimes blistering) often seen in light-skinned individuals after significant sun exposure. UVB light assists in wound healing by inducing an inflammatory reaction, stimulating the growth of granulation tissue, and promoting breakdown and elimination of dead tissue from the wound.[97]

Ultraviolet light C (UVC) (wavelength 200 to 290 nm) is the form of UV light most often used in the treatment of chronic wounds specifically for its bactericidal effects. Recent research demonstrated that UVC is capable of killing strains of bacteria in laboratory cultures, in animal tissue, and in patients with chronic ulcers infected with methicillin-resistant *Staphylococcus aureus*.[98,99] In patients with chronic wounds, UVC treatment also reduced the wound bioburden and facilitated wound healing.[100] In addition, in vitro studies have shown that UVC therapy destroyed 100% of antibiotic-resistant bacteria.[101]

Practice PointPractice Point

Contemporary UV light equipment is usually equipped with distance guards, which ensure correct distance and reproducibility of UV treatments.

Ultrasonic Energy

Therapeutic ultrasound delivers energy through mechanical vibrations in the form of sound waves at frequencies above detection by the human ear (>20 kHz). Ultrasound affects tissue through thermal and nonthermal mechanisms, which are determined by the physical properties, which are associated with ultrasound: (1) frequency or the number of oscillations a molecule undergoes in 1 second and (2) intensity or level of power. The thermal effects associated with greater tissue absorption of energy increase with higher frequencies, whereas nonthermal effects are predominant with lower frequencies and when ultrasound is pulsed.[102] Ultrasound is nonionizing radiation and therefore does not impose the hazards attributed to ionizing radiation such as cancer production and chromosome breakage.[103]

High-Frequency Ultrasound

High-frequency ultrasound is currently used in the range of 1 to 10 MHz primarily for diagnostic fetal imaging and vascular duplex scanning. It has also been used in the 1 to 3 MHz range to promote soft tissue injury healing and occasionally reported to facilitate wound healing. High-frequency ultrasound devices create their effect by running electricity through a crystal in the sound head, causing the crystal to "vibrate." The vibrations are then passed through the sound head via an ultrasound coupling medium into the tissues, causing them to vibrate and creating a local thermal effect.

Low-Frequency Ultrasound

Recently, low-frequency ultrasound (LFU) has been added to the arsenal of tools available for wound care and is the most common type of US device used in wound care today. Delivery of LFU to wounds has been shown to effectively debride necrotic tissue, eradicate some strains of bacteria from the wound, and facilitate the wound healing process.[104]

LFU therapy is believed to promote wound healing via two mechanisms: (1) cavitation (production and vibration of micron-sized bubbles within the coupling medium and fluids within the tissues) and (2) acoustic streaming (movement of fluids along acoustic boundaries). The combination of cavitation and acoustic streaming, both of which occur more frequently with kilohertz than megahertz ultrasound, provides a mechanical energy capable of altering cell membrane activity and, therefore, cellular activity[105] Cavitation is the vibrational effect of ultrasound on micron-sized gas bubbles that form in the blood and lymph and tissue fluids and in the ultrasound coupling media solutions. Periods of high and low pressure in treated tissue can cause the bubbles to increase and decrease in size. Cavitation can be both stable and unstable. Stable cavitation acts to enhance the acoustic streaming phenomena. Unstable or transient cavitation occurs when the microbubbles significantly increase in size and violently implode during the low pressure part of the US wave cycle. On implosion, the bubbles release a large amount of energy, which is destructive to bacteria while being essentially harmless to healthy viable tissue and cells. The tiny shock waves produced by bubble implosions cause preferential and rapid liquefaction and fragmentation of adherent necrotic fibrin (fibrinolysis) and the destruction of microorganisms on wound surfaces.[106]

Acoustic streaming has been shown to alter cell membrane permeability and second messenger activity, which in turn may result to increased

production of growth factors as well as macrophage activity and fibroblast proliferation and migration creating a collagen-rich connective tissue matrix.[107,108] The fibroblasts then differentiate into myofibroblasts to facilitate the contraction process of wound repair. The mechanical energy from an ultrasound wave is absorbed by an individual protein molecule, theoretically inducing a conformational change. Signal transduction pathways can also be stimulated from ultrasound-generated mechanical energy. This may result in a broad range of cellular effects that impact wound healing, including leukocyte adhesion, growth factor and collagen production, increased angiogenesis, increased macrophage responsiveness, increased fibrinolysis, and increased nitric oxide levels.

Contact and Noncontact Technology

There are currently two categories of ultrasonic devices available for the delivery of LFU in wound care. Noncontact devices allow for the treatment of wounds with ultrasonic energy without directly contacting the tissue or causing thermal effects. The energy is transferred to the wound via the coupling solution (normal saline), which has the appearance of a mist over the wound surface. These devices stimulate the tissue with both acoustic streaming and cavitational effects but do not physically debride tissues. Contact devices also transfer ultrasonic energy to the wound but accomplish this with the combined effects of the coupling solution and the mechanical action of the probe tip. The probe oscillates at the programmed device frequency. This vibrational energy allows for sharp debridement of the wound in addition to wound stimulation via cavitation and acoustic streaming. Based on the vibrational energy of the probe tip, heat is generated during the procedure. While the heat is typically dissipated via the coupling solution, the clinician must be aware of the potential for focal heating if the probe tip is not moved continuously across the wound base during debridement. The available devices vary in frequency and other features that distinguish them from operationally (Table 9-7).

Table 9-7 Low-Frequency Ultrasound Summary and Comparison of Specifications

	SonicOne	Sonoca-180	Arobella Qoustic Wound Therapy System	MIST Therapy System	Panacea Qoustic Wound Therapy System
Operational Features					
Frequency	22.5 kHz	20–80 kHz	35 kHz	35 kHz	40 kHz
Intensity	Variable: auto gain control	Variable: 40%–100%	Variable: 10%–100%	Preset	Preset
Mode	Continuous or pulsed	Continuous	Continuous or pulsed	Continuous	Continuous
Coupling	Sterile saline	Sterile saline	Sterile saline	Sterile saline vapor	Oxygenated sterile saline vapor
Controls	Foot pedal	Foot pedal		Button on hand piece	Trigger on hand piece
Treatment time	Usually 2–5 min	Usually 2–5 min	Usually 2–5 min	Usually 5–10 min	Usually 5–10 min
Wound contact with applicator	Yes; autoclavable metal probes	Yes; autoclavable metal probes	Yes; autoclavable curette shaped	No Disposable applicator	No Autoclavable applicator
Clinical Features					
Selective excisional debridement	Yes	Yes	Yes	No	No
Antibacterial	Yes	Yes	Yes	Yes	Yes
Associated pain	Yes	Yes	No	No	No
Aerosolization	Yes	Yes	Yes	Yes	Yes

Adapted and used with permission from Luther Kloth, PT, MS, FAPTA, CWS,FCCWS and Jeffrey Niezgoda, MD, FACHM, MAPWCA, CHWS.

Noncontact Nonthermal Devices

There are two noncontact nonthermal devices. These include the MIST Therapy System (Celleration Inc., Eden Prairie, MN) and the Qoustic Panacea Wound Therapy System Model AS1000 Series (Arobella Medical, LLC, Minneapolis, MN). The MIST device operates at a frequency of 40 kHz in the continuous mode and promotes healing by actively stimulating cells, reducing the bioburden, and increasing blood flow to the immediate treatment area[109] (Fig. 9-20). The Panacea device operates at a frequency of 35 kHz in the continuous mode and provides the same wound healing properties provided by MIST, but with the addition of an oxygenated irrigant. Thus, the Panacea provides the benefit of noncontact nonthermal ultrasound with the additional benefit of topical oxygen.

Figure 9-20. (A, B) MIST therapy. (Courtesy Celleration, Inc.)

Contact Thermal Devices

In contrast to the noncontact nonthermal energy transfer devices, the contact thermal devices can accomplish sharp selective and excisional debridement and are often referred to as ultrasound-guided debridement (UGD). Currently, there are three manufactures of UGD devices. The Qoustic Wound Therapy System (Arobella Medical, LLC, Minneapolis, MN) operates at a frequency of 35 kHz in either continuous or pulsed mode. It provides selective, precise, and gentle fragmentation of soft and hard tissues, with preservation of healthy tissue through ultrasonic separation of damaged tissue (Fig. 9-21).[110] This cleanses the wound area with less pressure and pain and also improves granulation formation of treated tissue. The SonicOne (Misonix, Farmingdale, NY) and the Sonoca-180 (Söring, Inc., North Richland Hills, TX) are the other UGD devices on the market (Fig. 9-

21B). The SonicOne operates at a frequency of 22.5 kHz in the continuous or pulsed mode. The Sonoca-180 operates at a frequency of 25 kHz in the continuous mode. All UGD devices allow for deep tissue penetration of the ultrasonic energy and cause bacterial cell destruction within the wound bed through combined vibrational and cavitational effects. Continuous irrigation with the coupling solution provides a medium for cavitation and flushes the wound of necrotic tissue fragments and fibrin deposits mobilized during the debridement while preserving the granulation tissue. The Arobella device is additionally cleared for wound bed preparation.

Figure 9-21. Ultrasound-guided debridement (UGD). (Photos courtesy of J.A. Niezgoda.)

Various clinical studies have evaluated the safety and efficacy of LFU therapy in patients with a variety of wounds, including recalcitrant pressure ulcers, chronic lower extremity leg and foot ulcers, and diabetic foot ulcers.[105] Recent studies have suggested noncontact LFU as beneficial in helping to resolve or mitigate the severity of suspected deep tissue injury (sDTI) when applied early in the sequelae of tissue destruction from these wounds. More research is needed to determine the full effects and outcomes of noncontact LFU on sDTI; however, the questions posed by preliminary

studies are creating interest in determining if this biophysical energy can indeed decrease the severity of tissue destruction caused by DTIs.[111]

Practice PointPractice Point

- *Ultrasound* cannot be used near parts of the body containing electronic implants/prostheses, on areas of malignancy, or over the lower back or abdomen during pregnancy.
- LFU has demonstrated bacterial killing during wound debridement via cavitation. This effect is unique to UGD devices and should be considered when managing contaminated and critically colonized wounds.
- Contact devices are associated with thermal tissue effects and clinicians should be skilled in UGD technique.
- As with all sharp debridement procedures, consideration and management of pain are paramount.

Biological

The group of biological modalities also includes a large number and variety of technologies and is a category that has experienced significant change, growth, research, and development. This progress includes both the actual technologies as well as our way of handling them from a nomenclature, organizational, and reimbursement perspectives. The activities of the Alliance of Wound Care Stakeholders provide a good illustration.[112,113] The alliance is a nonprofit multidisciplinary trade association comprised of professional societies with interest and clinical involvement in wound care (i.e., the American Professional Wound Care Association, the American College of Hyperbaric Medicine, and the Wound, Ostomy and Continence Nurses Society) and patient organizations whose mission is to promote quality care and access to products and services for people with wounds through effective advocacy and educational outreach in the regulatory, legislative, and public arenas. This group closely follows fiscal and regulatory policy, intervening with clinically appropriate commentary and recommendations. Historically, most of the advanced biological therapies were collectively referred to as tissue-engineered skin substitutes. Tissue

engineering is the development of materials that combine novel substances with living cells to yield functional tissue equivalents/skin substitutes.[19] The term skin substitutes has become obsolete not only due to inaccuracy of the descriptor but also as it has caused confusion in discrimination between devices as well as difficulty for coding, billing, and reimbursement. The alliance via consensus recently proposed a new term for this category, Cellular- and Tissue-Based Products (CTPs). This new lexicon has been adopted by many professional societies as well as regulatory agencies.

For educational purposes, this discussion of biological therapies subdivides CTPs in to bioactive materials, biosynthetic matrices and grafts, biopharmaceuticals, and biotechnologies.[29]

Bioactive Materials

Bioactive materials are those derived directly from human tissue sources. Some of the technologies included in the group include Cultured Living Allografts (sometimes still referred to as Bioengineered Skin Substitutes), Dehydrated Human Amnion/Chorion Membrane Allografts, Autologous Platelet Grafts, epidermal grafts, and Autologous Dermal Matrix.

Cultured Living Allografts

Technology has spawned a new generation of biological materials to advance wound healing and provide all the characteristics of natural skin. Skin substitutes were originally designed to replace autografts, which are the harvesting of a patient's own skin to apply to a burn or wound.[9] Current devices may be comprised of epidermal cells, dermal cells, or both supported by a biodegradable matrix.[9] Products containing both epidermal and dermal cells are referred to as bilayered.[9]

Two products containing living cells are approved for use in the United States, Apligraf and Dermagraft. Both contain living cells derived from neonatal foreskin. A single foreskin can produce over 200,000 units of the product.[26] Apligraf is bilayered consisting of a scaffold of type I bovine collagen that contains living human dermal fibroblasts and an overlying cornified epidermal layer of living human keratinocytes. Apligraf doesn't contain Langerhans' cells, melanocytes, or endothelial cells, perhaps explaining why it isn't clinically rejected. Dermagraft is comprised of cryopreserved human fibroblasts on a polyglycan scaffold indicated for use in the treatment of full-thickness diabetic foot ulcers.[114,115] When

Dermagraft is placed on an ulcer, the mesh material is gradually absorbed and the human cells grow into place, replacing the damaged skin.[115] Both products are tested extensively for infectious agents. Cultured Living Allografts are not recommended for use on infected wounds or wounds with sinus tracts and should not be used in patients who are sensitive to bovine products.[115]

Cultured Living Allografts are applied by a clinician, typically in the wound care clinic and several applications may be needed before healing is attained. Prior to application, excellent wound bed preparation is mandatory. Wounds are debrided, moisture balance maintained, and infection monitored. The wound may be cultured prior to the procedure. Appropriate oral antibiotic therapy or antimicrobial wound dressings maybe used to prevent "losing" the graft in the setting of heavy or critical colonization. The graft site must be protected from injury; the secondary dressing is changed without disturbing the graft site.

Cultured Living Allografts have demonstrated improved outcomes over standard of care in several randomized controlled trials in patients with diabetic foot ulcers and venous insufficiency.[116,117] This level of evidence has not been matched by many of the other products discussed in this section, and this has translated to widespread clinical use of these allografts.

Dehydrated Human Amnion/Chorion Membrane Allografts

Dehydrated Human Amnion/Chorion Membrane Allografts (DHACMs) have been used in surgical reconstruction for over 50 years, yet have only recently entered the wound care clinic. DHACMs are harvested from human placentas and processed for use in wounds with the intent to promote healing. DHACMs have been widely used for burns where extensive areas of skin have been damaged. These products contain either both amnion and chorion tissue or single-layer amnion alone. DHACM tissues are nonimmunogenic, reduce inflammation and scar formation, contain relatively high levels of growth factors, and have been shown to stimulate cellular proliferation as well as promotion of mesenchymal stem cell migration.[118,119] Tissues are harvested during planned cesarian section deliveries and undergo extensive maternal and tissue microbiological and viral screening including HIV-1 and HIV-2 antibody, hepatitis B core antibody, HIV type 1 nucleic acid test, hepatitis B surface antigen, HTLV-1

and HTLV-2 antibody, hepatitis C antibody, serological test for syphilis, CMV total antibody test, and hepatitis C virus nucleic acid test (NAT). Several products are marketed and differ based on layers of amnion–chorion tissue used and variations in proprietary tissue processing.

The literature reports excellence healing outcomes can be gained with the use of DHACMs. One study of DFUs compared DHACM with standard of care (SOC) versus SOC alone. The authors reported 92% healed in 6 weeks compared to 8% for control, with an average of 2.5 graft implants used to closure.[120] Early reports and trials suggest that this technology may have a significant positive impact on the management of chronic wounds.

Epidermal Grafts

Epidermal grafting is the application of negative pressure to the skin to create little fluid-filled blisters (suction blistering), during which the lamina lucida of the skin is cleaved from the underlying layers. This separates the epidermis from the dermis. The blisters are then cut and emptied and the loose skin is transferred side by side to the nonhealing wound. Epidermal grafting was first reported in 1964 to treat vitiligo as the epidermal grafting transfers melanocytes as well as keratinocytes.[121] Recently, CelluTome technology was introduced for the management of chronic wounds (CelluTome Epidermal Harvesting System, KCI, San Antonio, TX). The procedure is painless and allows for the efficient transfer of multiple epidermal micrografts to the recipient wound bed. See epidermal grafting, Figure 9-22. The epidermal grafts are uniform in size and contain basement membrane components and keratinocyte-associated growth factors. A prospective trial evaluating the effectiveness of autologous blister grafting for nonhealing leg ulcers reported 89% of the patients treated achieved complete closure in 12 weeks. Additionally the authors reported observations of increased granulation tissue and stimulation of epithelialization from the wound edge in nearly every wound. Epidermal grafting has been suggested to be an excellent option for patients with clean granular wounds to hasten epithelialization and eliminate the need for operative intervention and split-thickness skin grafting. Clinicians skilled in epidermal grafting report painless, rapid healing of donor site especially when compared to standard skin grafting procedures.

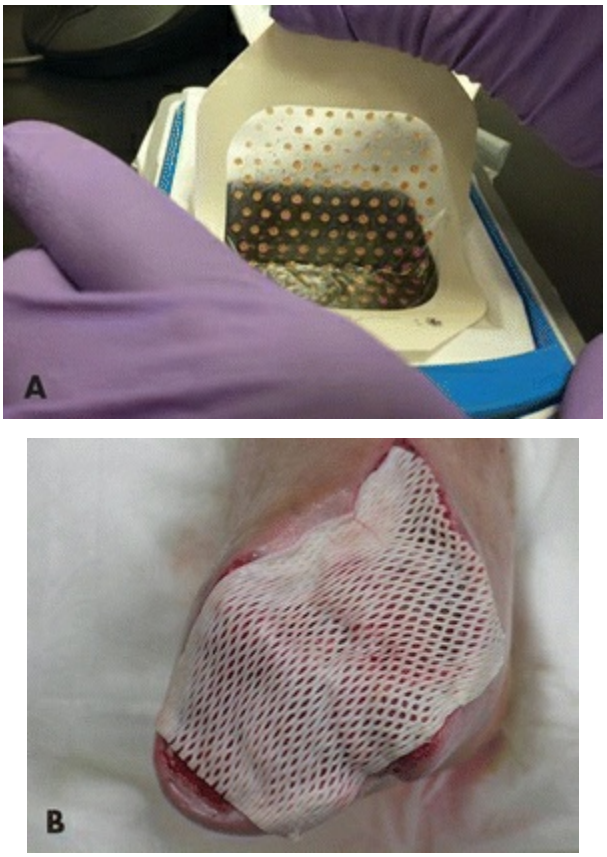


Figure 9-22 Epidermal grafting (CelluTome™ Epidermal Harvesting System, KCI, San Antonio, TX). **(A)** Harvesting the graft. **(B)** Graft on the wound. (Photos courtesy of J.A. Niezgoda.)

Autologous Dermal Matrix

Autologous Dermal Matrices (ADMs) are allograft scaffolds, which are produced by harvesting human skin. The dermis is then minimally processed to remove the epidermis and all cellular components but maintain ECM structure using septic processing without terminal sterilization. The finished product is lyophilized and packaged for storage and shipping. In this freeze-dried state, the product must be rehydrated prior to clinical application. This technology has been available for clinical use for several years from a variety of vendors.

Graftjacket Regenerative Tissue Matrix

Graftjacket (KCI, San Antonio, TX) is available in two forms, a flat graft or Graftjacket Regenerative Tissue Matrix (RTM) and Graftjacket Xpress, a flowable soft tissue scaffold (FSTS) (Fig. 9-23). The suggested mechanism for action of ADMs is to implant an ECM scaffold to support the ingrowth

of host tissue, provide an inductive and conductive stimulus for cell and vessel migration, and allow for normal tissue remodeling via dynamic reciprocity.[122] Clinical studies have reported efficacy in the use of Graftjacket with both wound and wounds complicated by sinus tracts. In a randomized controlled trial of 86 patients with diabetic foot ulcers, Graftjacket RTM improved healing (2.7 times greater probability of healing) at the 12-week interval compared to moist wound therapy with a statistically significant increase in percentage of wounds healed (p = 0.0289).[123]

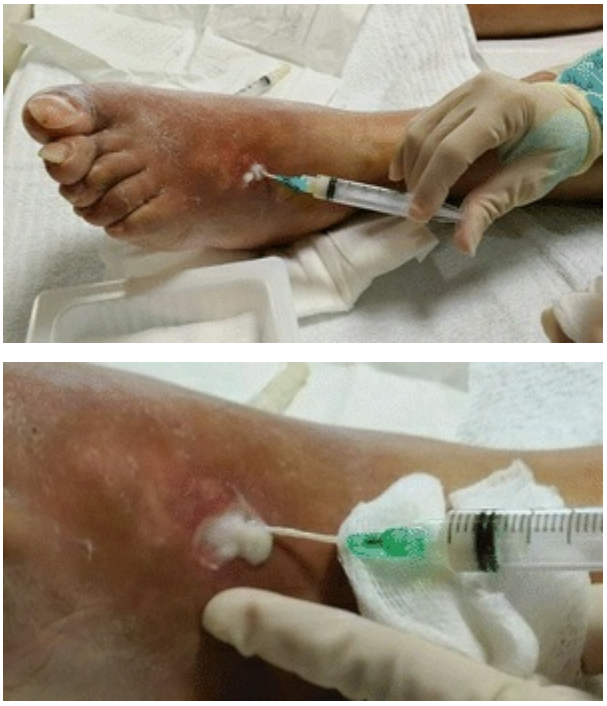


Figure 9-23 Graftjacket Regenerative Tissue Matrix. (Photos courtesy of S. Baranoski.)

Practice Essentials

- Meticulous wound bed preparation is paramount to good outcomes when using any biological material including Cultured Living Allografts, epidermal grafting, or Autologous Dermal Matrices.
- Patients with plantar surface wounds mandate aggressive offloading after grafting.
- Watch for signs and symptoms of infection.
- During follow-up after implantation, it is not necessary to debride the yellow caramelized crust at the edges or in the wound; this is likely

graft incorporation and must be distinguished from nonviable tissue.

Practice PointPractice Point

To date, no biological material is able to completely replicate normal, uninjured skin.[9]

Biosynthetic Matrices/Xenografts

Biosynthetic matrices and xenograft materials comprise a group of technologies where the product is either produced synthetically or created using nonhuman animal tissues (typically bovine or porcine). These products are decellularized and sterilized, maintaining a rich collagen matrix, which is applied to the wound base to serve as a scaffold and orchestrate healing via dynamic reciprocity. The finished product is lyophilized and must be rehydrated. Collagen is typically cross-linked.

OASIS Wound Matrix (Cook Biotech, West Lafayette, IN/Smith & Nephew, London, England) is an intact xenograft matrix naturally derived from porcine small intestinal submucosa (SIS) that provides a three-dimensional, bioresorbable ECM (Fig. 9-24). It is incorporated and absorbed into the wound and is indicated for the management of partial- and full-thickness wounds, pressure ulcers, venous ulcers, chronic vascular ulcers, diabetic ulcers, trauma wounds (abrasions, lacerations, second-degree burns, skin tears), draining wounds, and surgical wounds (donor sites/grafts, post-Mohs' surgery, postlaser surgery, and wound dehiscence). Clinical data and randomized studies demonstrate improved healing over standard of care in a variety of chronic wounds including diabetic foot ulcers, venous ulcers, and surgical wounds. A prospective, randomized, multicenter trial evaluated the effectiveness of SIS plus standard of care (SOC with compression therapy) versus SOC alone in 120 patients with chronic venous insufficiency and lower leg ulcers of greater than 1-month duration. At 12 weeks, the OASIS group demonstrated 55% healing compared to 34% of the SOC group ($p = 0.0196$).[124]

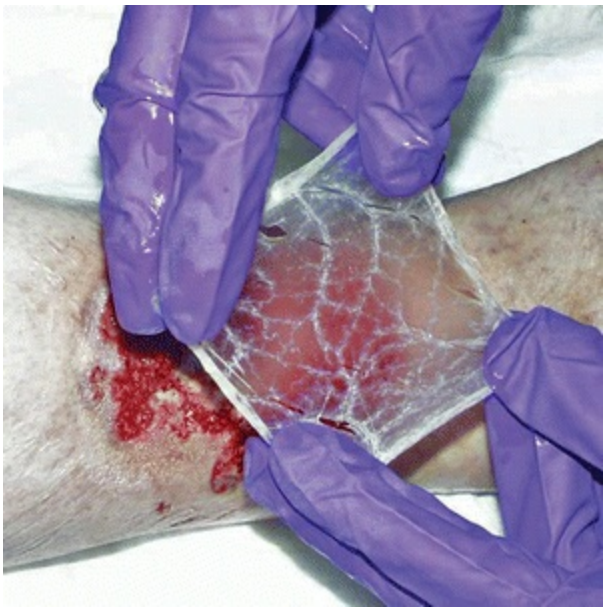


Figure 9-24 Xenograft matrix (OASIS Wound Matrix, Cook Biotech, West Lafayette, IN/Smith & Nephew, London, England). (Photos courtesy of J.A. Niezgoda.)

Biopharmaceuticals

Biopharmaceuticals are distinct from chemically synthesized pharmaceutical products. A pharmaceutical drug (medicine or medication and officially medicinal product) is any chemical substance formulated or compounded as single active ingredient or in combination of other pharmacologically active substance. Pharmaceuticals are typically engineered macromolecular products, and although they can include protein-based and nucleic acid–based drugs, which are sometimes called medicinal products or therapeutic biological products, they are chemically synthesized and not of biological origin. Synthetically derived compounds can therefore be distinguished from biopharmaceuticals, which are usually directly extracted from a biological source like blood or blood components.[125] Examples of biopharmaceuticals include vaccines, blood or blood components, allergenics, recombinant therapeutic proteins, and growth factors.

Growth Factors

Growth factors, also referred to as cytokines, are proteins (polypeptides) that occur naturally in the body. They are found primarily in platelets and macrophages. Currently, there is only one growth factor available in the

United States, platelet-derived growth factor (PDGF). Several other types of growth factors are being studied in clinical trials, and many are already available for clinical use outside the United States. These include epidermal growth factors, transforming growth factors, fibroblast growth factors, and vascular endothelial growth factors. From a clinical and research perspective, growth factors can be categorized into two major groups: single growth factors manufactured through recombinant DNA technology (PDGF) and multiple growth factors secured from human platelet releasate.[24]

Regranex (becaplermin) Gel 0.01% (Smith & Nephew, London, England) is a recombinant PDGF that is available by prescription and is indicated for the treatment of lower extremity diabetic neuropathic ulcers that extend into the subcutaneous tissue or beyond and have an adequate blood supply. Regranex has been available for many years and has extensive clinical experience. It has been found to be efficacious in the management of diabetic ulcers and in granulating wounds but has recent concern raised secondary to a postmarketing retrospective cohort study, which reported an increased rate of mortality secondary to malignancy that was observed in patients treated with three or more tubes. The manufacturer has subsequently issued the recommendation that Regranex should only be used when the benefits can be expected to outweigh the risks and should be used with caution in patients with known malignancy.[126]

Practice PointPractice Point

Becaplermin gel (Regranex) must be kept refrigerated and used with caution in patients with known malignancy.

Platelet-Rich Plasma

Platelet-rich plasma (PRP) is the exogenous application of autologous platelet-derived concentrate. Platelet-rich plasma contains multiple growth factors. Unlike becaplermin, which is produced by the manufacturer and comes ready to use, platelet-rich plasma is prepared at the point of care for the patient. Blood taken from the patient is mixed with a small amount of anticoagulant citrate dextrose and the solution is then centrifuged to separate the platelets and serum.[127,128] The concentrated platelets (Fig. 9-25) are

then placed into the specially provided syringe that mixes the concentrate as it is applied to the prepared wound bed as a gel.[127,128]

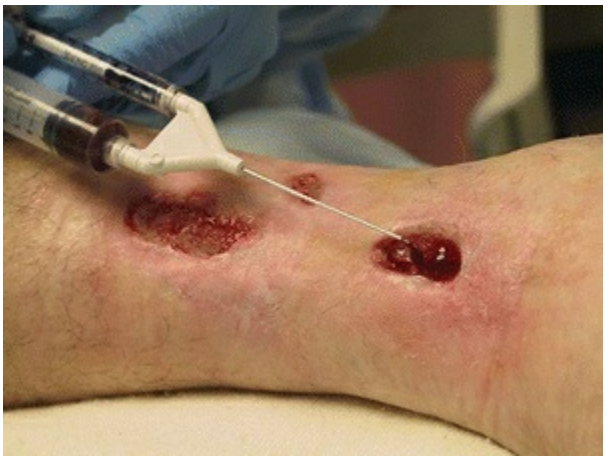


Figure 9-25 Platelet-rich plasma. (Photo courtesy of J.A. Niezgoda.)

Biotechnologies

This final category of Biological Advanced Therapies includes genetic transfer, stem cell therapies, and other progressive and novel technologies. Most of these therapies continue to be developed under research protocols with limited clinical utilization. While early data is intriguing and even compelling in some case, much more work is required to fully integrate these modalities in clinical algorithms with corresponding reimbursement.

Stem cell therapy is the process of delivering autologous stem or progenitor cells directly to wounded tissues to differentiate and replace deficient cells such as fibroblasts, endothelial cells, and myocytes or to produce proangiogenic proteins, which stimulate angiogenesis tissues for the purpose of enhancing wound healing. Stem cells are biological cells found in all multicellular organisms defined by two characteristics: (1) division, allowing mitosis or mitotic division and self-renewal to produce more stem cells, and (2) differentiation, creation and formation of diverse specialized cell types. While embryonic cell are called pluripotent with the ability to differentiate into multiple cell types, adult or somatic stem cells (the type used in wound care applications) are called progenitor cells and have limitations on their ability to differentiate and replicate. Clinically, stem cells are harvested from bone marrow, adipose tissue, or blood and processed via cell separation using high-speed centrifuge techniques. The isolated stem cells are then applied clinically using tissue injection or grafting. Literature data in wound care are early and sparse but studies have

reported improved healing as well as synergy with HBOT.[129,130]

Pharmaceutical

Oxygen Therapy

As defined above, a pharmaceutical drug is any chemical substance formulated or compounded as single active ingredient or in combination of other pharmacologically active substance. Although oxygen is a critical element to living organisms as it drives nearly all biological processes, it does meet the criteria of a pharmaceutical as a chemical substance when administered for therapeutic purposes. Oxygen when utilized for the purpose of wound healing is provided as a prescriptive device, a pharmaceutical, and therefore is discussed separately from other biological therapies. There are various types of oxygen therapy available for the management of acute and chronic wound conditions. This section will discuss hyperbaric oxygen in detail based on the widespread use within the wound care community and then briefly discuss several types of topical or transdermal oxygen devices.

Hyperbaric Oxygen Therapy

Definition

HBOT is defined as treatment during which a patient inhales 100% oxygen while enclosed in a pressurized chamber exceeding 1.4 atmospheres absolute (ATA). HBOT has been used worldwide for more than 50 years in the treatment of many chronic nonhealing wounds and ulcers.[131] Treatments are administered either in a multiplace chamber (Fig. 9-26A) occupied by several patients or in smaller monoplace chamber (Fig. 9-26B), containing only one patient at any one time. In multiplace chambers, patients are treated simultaneously in an environment of pressured air, while breathing pure oxygen delivered via mask or hood tent. In a monoplace chamber, a single patient is placed in a pressurized environment of pure oxygen. For routine conditions, HBOT is typically administered between 2.0 and 2.5 ATA, with 90 minutes of oxygen breathing, interrupted briefly by an “air break” (5 to 10 minutes of air breathing). The safety record of HBOT in this country is excellent and the technology has become a vital part of the growth of comprehensive wound care programs across the nation.[62,132]

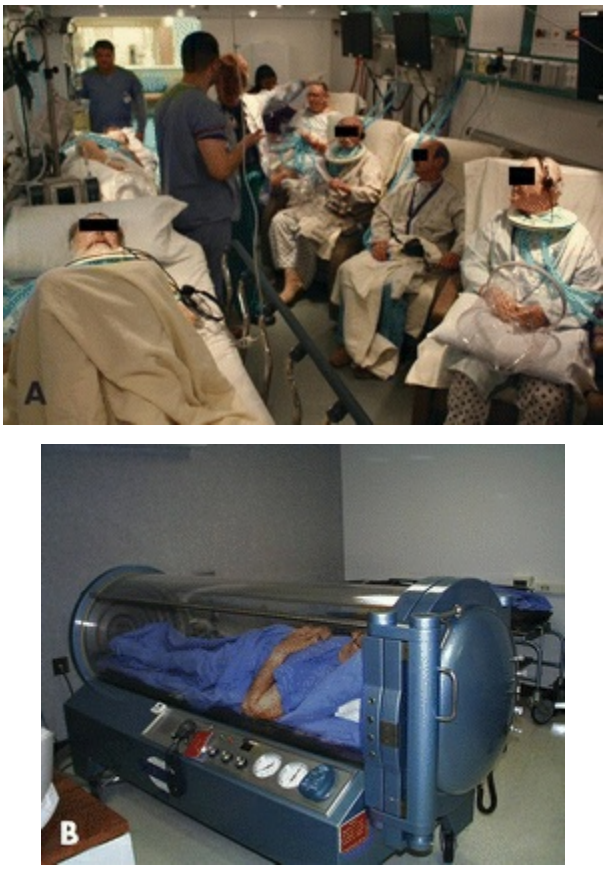


Figure 9-26 Hyperbaric chambers. **(A)** Hyperbaric mulitplace chamber. **(B)** Hyperbaric monoplace chamber. (Photo courtesy of J.A. Niezgoda.)

Mechanism of Action of HBOT

An uncompromised patient, breathing air at sea level, will typically have an arterial pO_2 of 100 mm Hg. The majority of this oxygen is carried bound to hemoglobin with only a small percent of oxygen physically dissolved in the plasma. The normal diffusion radius for an oxygen molecule is 64 microns at the end arteriole and about 36 microns from the venous system where the pO_2 might be as low as 34 mm Hg. However, this same patient breathing 100% oxygen at 3 ATA will have an enormous increase in the amount of oxygen physically dissolved in the plasma (6.9 volumes percent) compared to the volume of oxygen dissolved at 1 ATA (0.3 volume percent) (Fig. 9-27). Plasma pO_2 values well in excess of 1,500 to 1,800 mm Hg are routinely achieved in hyperbaric patients. In this setting, the hemoglobin never desaturates, and thus, hemoglobin plays a minimal role in the transport of oxygen. Tissue oxygen partial pressures can reach supraphysiologic oxygen levels of 250 to 300 mm Hg, far above the normal 30 to 40 mm Hg when breathing 1 ATA air. This 18- to 20-fold increase in

dissolved oxygen establishes a tremendous gradient between the plasma and the peripheral tissues and provides tissue hyperoxygenation and stimulation of neoangiogenesis and drives the other physiological benefits of HBOT.[62,133]

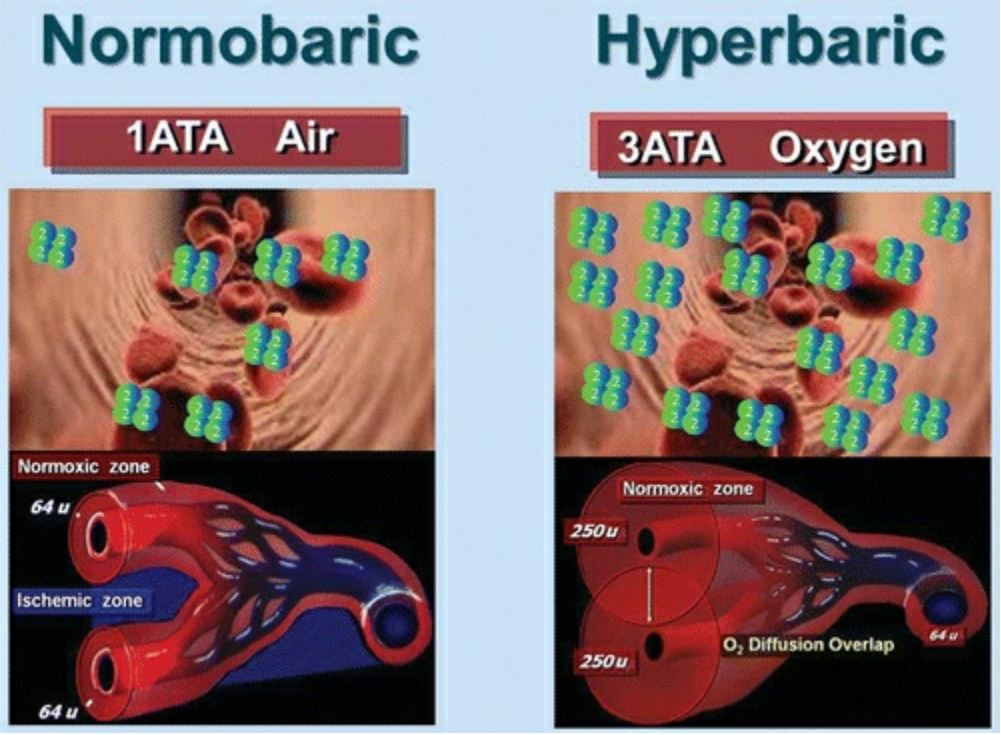


Figure 9-27 Hyperbaric physiology. (Courtesy of J.A. Niezgoda.)

Physiological Effects of HBOT

Hyperbaric oxygen has been demonstrated to deliver several physiological effects that can have a positive impact on wound healing and well as other non–wound-related indications, such as carbon monoxide poisoning and decompression illness.

- Hyperoxygenation of ischemic tissue: Chronic wounds are frequently hypoxic. HBOT increases tissue partial pressure of oxygen (pO_2), correcting wound hypoxia intermittently.[94] It then allows for acceleration of the wound healing process through a series of actions that continue long after the HBOT session has ended and tissue oxygen levels have returned to pretreatment values.[134–136]
- Angiogenesis: HBOT can induce angiogenesis and neovascularization in hypoxic tissues via induction and increased expression of vascular endothelial growth factor (VEGF). Studies demonstrate that VEGF

levels in hypoxic wounds rose by approximately 40% within five days of starting HBOT but then dropped to control levels within three days of stopping the treatment. In addition to an increase in angiogenesis, there was a 20% increase in wound bed perfusion following ten days of HBOT as demonstrated via laser Doppler imaging.[137,138]

- Vasoconstrictive effects of oxygen: Vasoconstriction takes place in both arterial and venous vessels, which reduces edema and congestion while the amount of oxygen supplied by the plasma is increased.[139]
- Improved wound metabolism: The elevation in pO_2 promotes wound healing by directly enhancing fibroblast replication, collagen synthesis, and the processes of neovascularization and epithelialization.[134,140]
- Upregulation of growth factors: HBOT causes the up-regulation of cytokines, including PDGF and VEGF, thereby stimulating cellular repair and regeneration as well as enhancing angiogenesis.[141,142]
- Antibacterial effects: The increase in available oxygen enhances the leukocyte bactericidal effect, including the killing of aerobic gram-positive (*S. aureus*) and gram-negative organisms in addition to being cytotoxic to anaerobes. Neutrophils or polymorphonuclear cells (PMNs) require oxygen for phagocytosis and killing of bacteria via oxygen-free radical formation and other oxidative mechanisms. Should the oxygen tension fall below 30 mm Hg, the efficiency of bactericidal action of PMNs decreases dramatically, leaving the patient at higher risk for infection.[142]
- Antioxidant effect: HBOT has been demonstrated to have a positive impact on limiting the deleterious effects of ischemia–reperfusion injury. It blocks guanylate cyclase, the trigger molecule for beta-2 integrin that forms the leukocyte adhesion molecule. HBOT also blocks the action of the intracellular ICAM adhesion molecules that cause any leukocytes that become adherent to the capillary wall to bore through into the tissues surrounding the vessel, releasing the superoxide anion, proteinases, and elastase that liquefy tissue following crush injury and other forms of ischemia. Thus, following ischemia or sepsis, when the patient is treated early with HBOT leukocytes, do not adhere to the capillary walls to block circulation or cause lipid peroxidation.[143,144]

Clinical Indications for HBOT

The clinical indications for HBOT are provided based on review of current available evidence by expert consensus of professional societies. The

American College of Hyperbaric Medicine and the Undersea and Hyperbaric Medicine Society list the following indications:[145,146]

- Air or gas embolism
- Carbon monoxide poisoning
- Carbon monoxide poisoning complicated by cyanide poisoning
- Clostridial myositis and myonecrosis (gas gangrene)
- Crush injury, compartment syndrome, and other acute traumatic ischemias
- Decompression sickness
- Arterial insufficiencies (enhancement of healing in selected problem wounds)
- Central retinal artery occlusion
- Severe anemia
- Intracranial abscess
- Necrotizing soft tissue infection
- Osteomyelitis (refractory)
- Delayed radiation injury (soft tissue and bony necrosis)
- Compromised grafts and flaps
- Acute thermal burn injury
- Idiopathic sudden sensorineural hearing loss

Despite the opinion of these professional societies, many payors including CMS have different approved indications for reimbursement purposes. The hyperbaric clinician must be aware of these discrepancies in interpretation of the literature and evidence.

Special Considerations

The physiological effects of systemic oxygen on the human body are more similar to a drug than a physical modality and, like any drug, there is the risk of overdose (oxygen toxicity) and side effects (primarily barotrauma).[7,134,147,148] To ensure appropriate utilization and patient safety and to minimize the potential of adverse outcomes, HBOT must be supervised by a hyperbaric-certified physician with expertise and training in this highly specialized treatment modality. The American College of Hyperbaric Medicine has published credentialing guidelines and training recommendations and endorses the hyperbaric certification examinations offered by the American Board of Wound Healing[149] (ABWH).

Summary

Hyperbaric oxygen therapy is an advanced wound care technology that can have a significant positive and powerful effect in the management of patients with compromised wound healing, especially when the primary deficit is tissue hypoxia. When patients are carefully selected and appropriately managed with HBOT as part of a comprehensive wound care strategy, wound healing enhancement can be realized and limb loss avoided.

Practice Essentials

- HBOT is an adjunctive therapy accompanying good wound care practices.
- Patients should undergo aggressive arterial evaluation and revascularization when possible prior to the initiation of HBOT.
- Clinicians must be trained and educated in the physiology, mechanisms, and indications for HBOT, understand the importance of patient selection and safety, and seek certification to ensure appropriate utilization and optimal outcomes.
- Treatment cost and insurance approval should be considered before initiating HBOT.

Practice PointPractice Point

Carefully monitor the blood glucose of patients with diabetes before HBOT. Blood glucose levels have been known to drop precipitously when patients are in the HBOT chamber, putting them at risk for seizures or other complications from hyperglycemia.

Transdermal Oxygen/Topical Oxygen Therapy

Transdermal or topical oxygen therapy is the application of oxygen to the surface of a wound. Historically, this has been accomplished by placing the affected limb in an enclosed acrylic or plastic box and filling it with oxygen. Topical oxygen is frequently (and inappropriately) called “topical hyperbaric oxygen.” Use of this term simply adds confusion and is a

misapplication of the word hyperbaric. Hyperbaric oxygen is defined with the inhalation of 100% oxygen while the entire patient is enclosed within a chamber at pressures of at least 1.4 atmospheres absolute or greater neither of which is achieved by extremity devices. The stratum corneum in intact skin represents a significant barrier to oxygen diffusion from the atmosphere. However, in a patient with a wound, the absence of the dermis more easily allows for direct oxygenation via diffusion. Multiple studies report that topical oxygen that dissolves in tissue fluids is bacteriostatic and stimulates angiogenesis and wound healing.[150] A novel device that provides direct transdermal tissue oxygenation has been produced and studied. OxyBand Wound Dressing (OxyBand Technologies, St Louis, MO) is a transdermal therapy that delivers oxygen to a wound for up to 5 days following application. The oxygen is contained in a reservoir and diffuses into the wound base continuously at a rate controlled by a propriety semipermeable membrane (Fig. 9-28). A recent prospective, randomized, controlled study of burn patients undergoing harvesting of two donor sites reported significantly faster wound healing for OxyBand (9.3 ± 1.7 days; control, 12.4 ± 2.7 days [$p < 0.001$]) as well as lower pain scores in the OxyBand group [$p < 0.01$]).[151]

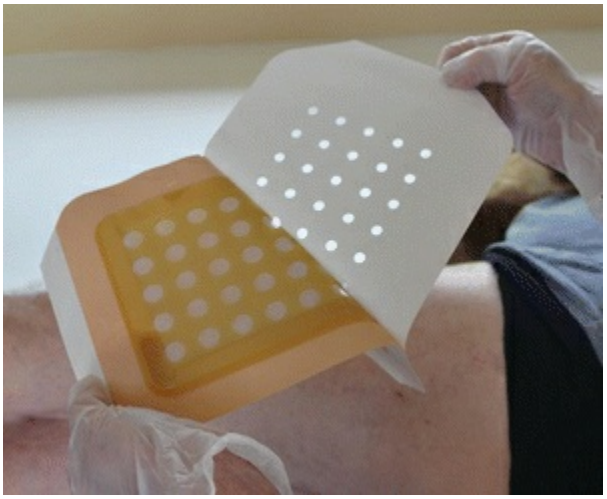


Figure 9-28 Transdermal oxygen therapy (OxyBand Wound Dressing TM, OxyBand Technologies, St Louis, MO).

Oxygen therapy plays a significant role in wound healing. The wide use of hyperbaric oxygen has been recently challenged on the basis of overutilization and cost as well as on clinical efficacy. In our ever-changing world of wounds, these issues must and will be addressed. Alternative oxygen delivery systems should be considered and warrant additional

clinical investigation.

- Frequency of dressing change varies among antimicrobials.
- Antimicrobial dressings are used in wounds with high bacterial bioburden or in wounds that are critically colonized to prevent wound infection.
- Antimicrobial dressings may be used under compression wraps to prevent infection in wounds with a high bacterial bioburden.

Scar Management

Patients, clinicians, and researchers are all concerned about scar appearance. Progress has been made in our understanding of the mechanisms involved in producing an exaggerated scar. The scientific principles for scar management and minimization—support, controlled inflammation, adequate hydration, and remodeling/maturation of collagen[152,153]—form the basis of product selection for scar control strategies (Table 9-8).

Table 9-8 SCAR Acronym for the Practical Application of Principles for Scar Management

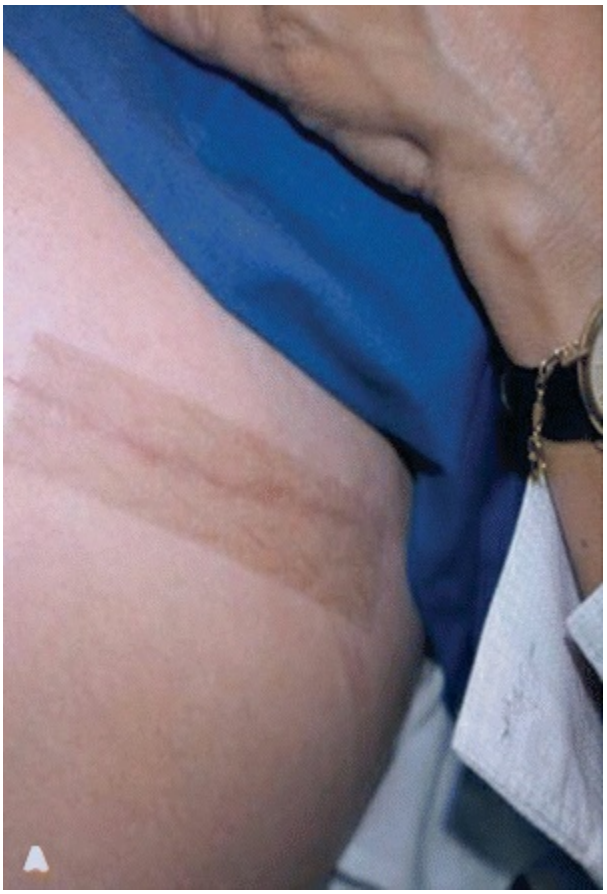
Principle	Pathophysiology	Proven Agents (Examples)
Support	• Vector forces increase the production of collagen. • All scars, especially long scars, need support in areas where vector forces continually pull on the scar.[152–155] For example, in a presternal chest area wound, neck, shoulder, and arm movements as well as weight from the breasts generate vector forces on the scar. (See Fig. 9-29B.)	• Microporous tape is the best form of scar support.[153,156,157] • To ensure that there is consistent support for the scar, the tape must be applied longitudinally along the scar path and not at right angles. (See Fig. 9-29A.) • Leave the tape in place for several days until it spontaneously separates from the skin. • Premature removal of the tape results in skin stripping, which sets up inflammation with negative consequences on the scar. • Small scars in some areas (face) may not need support.

Controlled inflammation	Because excessive inflammation results in exaggerated scars, controlled inflammation is a sought-after principle in scar management.	• Topical application of olive oil compounds (phenol compounds such as oleuropein in newly pressed olive oils) has anti-inflammatory activity and antimicrobial effects.[158,159] • Oleuropein stimulates proteasome function and fibroblast formation of new collagen.[160] Proteasomes prevent collagen from clumping as they up fragmented protein particles, including fragmented collagen, a process critical to the prevention of clumped collagen. Thus, stimulation of proteasomes and inhibition of inflammation are extremely advantageous to the process of scar maturation.
Adequate hydration	• Hydration of the scar surface is the basis of action of 90% of scar management systems on the market. • Most oils (tissue oils), lotions, and creams have beneficial effects on scars purely on the basis of their hydrative capacities.[161–163] Although this is obviously beneficial, it is limited in terms of the outcome it can produce and affects only one area of scar control. • Normal skin has a mature stratum corneum characterized by minimal transepidermal water loss (TEWL). • Dehydration of the stratum corneum initiates signaling to keratinocytes. These keratinocytes are stimulated to produce cytokines, which activate dermal fibroblasts to synthesize and release collagen. Excessive collagen production leads to abnormal scarring.[164]	• The most effective barrier to TEWL and stratum corneum breach is silicone, in the form of either sheeting or gels (dimethicone).[164–166] • In addition, gel derived from the plant *Bulbine frutescens* has been found to be effective as a hydrating agent; the glycoproteins of this plant extract are large and remain on the surface of the skin long enough to produce effective hydration of the skin.[153,154]
Remodeling/ maturation of collagen	• The quicker the scar matures, the less chance there is of hypertrophy. • Collagen maturation goes through phases, with collagen type III being present in greater levels in the early scarring phase. • As the scar matures, the ratio of type III to type I collagen returns to normal levels.[167] Thus, any agent that encourages a return to stable ratios is advantageous to scar outcome. • Transforming growth factor (TGF)-β is the prototype of a protein superfamily that has been recognized as the major fibroproliferative and collagen-stimulating agent involved in excess scarring (particularly TGF-β_1). Many isoforms of the protein exist, most of which share the same fibroproliferative properties. One isoform (TGF-β_3), however, appears to have a protective effect against excess collagen formation counteracting the TGF-β_1 effects.[168]	• Extracts of the *Centella asiatica* plant increase levels of mature collagen and encourage normalization of collagen ratios.[167–170] • Purified extracts (triterpenic fractions, including asiaticoside) isolated from *C. asiatica* have been shown to induce type I collagen synthesis in human dermal fibroblast cells.[167–169] • Asiaticoside downregulates TGF-β_1 expression and upregulates TGF-β_3 expression and is also capable of decomposing the products of type I collagen, contributing to the reduction of hypertrophic scar formation.[168] • Laboratory evidence of asiaticoside efficacy was demonstrated in the rabbit ear model, one of the only consistent animal models producing hypertrophic scarring.[170,171]

Adapted with permission from Widegerow, A.D. "Scar Management: The Principles and Their Practical Application," *World Council of Enterostomal Therapists Journal* 31(1):18-21, 2011.

Scar control does not rely on a single modality but rather on a number of proven factors whose combination results in a good outcome. Controlling

scar formation is an important part of wound management practice. Widgerow and colleagues[152,153,172] have described a patented process of applying a cream/gel that contains antiscar active agents (*Centella asiatica*, oleuropein, dimethicone, *Bulbine frutescens*) to the surface of microporous tape. This process has been used successfully for scar management. Within 2 minutes, the active agents in the gel are absorbed through the tape and onto the scar tissue. The saturated tape continues to work as an occlusive scar dressing. The tape remains in place during patient bathing and is only replaced when it separates from the skin (usually in 3 to 5 days). Gel is reapplied to the tape surface twice a day until scar maturation (white color) begins to occur. Once the scar is maturing well (usually 6 weeks), use of the tape can be stopped and the gel applied directly to the scar.[172] In a comprehensive trial,[153] 170 scars were assessed based on the SCAR acronym (Fig. 9-29). Hypertrophy was prevented in more than 80% of cases. Hypertrophy and scar exaggeration are seen in 60% to 80% of cases when there is no scar management.[173]



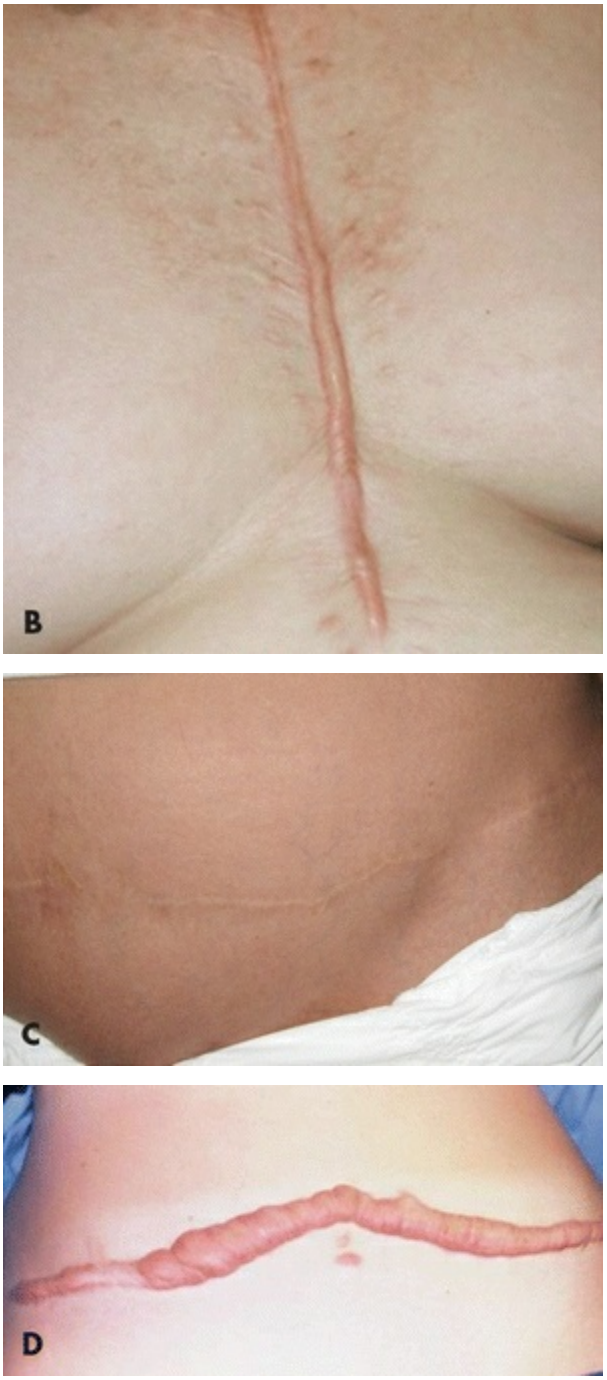


Figure 9-29. (A) Tape applied longitudinally along the direction of the scar. White or flesh-tone tape may be used, with the same effect. **(B)** Untreated hypertrophic scar in the presternal area; multiple vector forces are working against this scar. **(C)** The mature treated abdominal scar is flat, white, and nonreactive (note that it is normal for most scars to become hypopigmented to some extent). Compare this scar with the untreated scar. **(D)** Keloid scarring, which grows like a tumor and flows over the scar boundary, occurs wherever the skin is breached. Its pathogenesis and treatment are different from those of hypertrophic scars.

Although often confused as the same, keloid scars are different from

hypertrophic scars. Keloid scarring usually has a genetic component, where collagen type 1 is produced in a tumorlike fashion with uncontrolled growth of scar tissue. The typical wound history includes a well-managed noninfected wound that progressively increases in size and extends over the wound boundary. These scars may be painful, sensitive, and extremely uncomfortable. Treatment (often radiotherapy) is unpredictable and unsatisfactory. The SCAR principles are not applicable to keloid scarring. The different mechanisms between hypertrophic and keloid scars require different product approaches.

Summary

The selection of appropriate wound treatment options must be patient centered and driven by the specific goals of care. A succinct overview of the important characteristics and use of wound dressing products is provided (see Table 9-4).

Selecting from the abundant number of wound dressings that are available today can pose a challenge. Tools such as wound product charts and enablers such as MEASURES© or NICE© give clinicians a model by which to enhance their clinical decision making.

The concepts of moist wound healing and the significance of clinical treatment decisions regarding wound care dressing options described herein are essential elements of your wound care arsenal. Other therapies, such as ES, HBOT, ultrasound, and growth factors, to name a few, are also important wound healing options available for consideration. By using helpful practice points, tables, figures, and product algorithms, the clinician is guided through the milieu of product alternatives. Improving technology and evolving research into wound care dressings and modalities will continue to create new and challenging opportunities for all of us.

Show What You Know

1. **Wound dressings have evolved into a new concept of:**
   A. dry gauze.
   B. moist wound therapy.
   C. open to air.
   D. wet to dry.

2. Which of the following is not a category of moist wound care dressings?

A. Hydrogel dressing
B. Calcium alginate dressing
C. Roller gauze dressing
D. Foam dressing

3. Wound dressing selection should be based on the characteristics of the wound. All of the following should be considered when selecting dressings *except*:

A. size of dressing.
B. nurse preference.
C. moist or dry wound bed.
D. drainage.

4. A disadvantage of transparent film is that it:

A. is nonabsorptive.
B. is conformable.
C. allows wound inspection.
D. is impermeable to bacteria.

5. The acronym "MEASURES" is a useful tool for remembering the principles of wound care.

A. True
B. False

6. Which one of the following is a dermal skin substitute?

A. OpSite
B. Tegaderm
C. Allevyn
D. Apligraf

7. Which one of the following treatment options would not be an appropriate treatment option for a heavily draining wound?

A. Negative pressure therapy
B. Foam dressing
C. Calcium alginate dressing
D. Hydrogel amorphous gel

8. Prior to the application of tissue-engineered skin substitute, the

clinician needs to evaluate:

A. whether the wound bed is free of necrotic tissue.
B. the patient's immune system status.
C. that antirejection drugs have been administered.
D. the patient's weight.

9. All of the following are therapeutic effects of electrical stimulation <u>except</u>:

A. increased blood flow.
B. decreased tissue oxygenation.
C. increased angiogenesis.
D. decreased wound pain.

10. Which one of the following is <u>not</u> a method by which hyperbaric oxygen therapy affects wound healing?

A. Increased fibroblast replication
B. Upregulation of growth factors
C. Vasodilation effects of oxygen on the blood vessels
D. Enhanced leukocyte bactericidal effects

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Nutrition and Wound Care

10

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Objectives

After completing this chapter, you'll be able to:

- describe the process of nutritional screening using a validated form to identify risk for malnutrition and pressure ulcers
- identify the parameters involved in completing a nutritional assessment
- define the importance of nutrients for wound healing
- implement validated nutrition guidelines for the prevention and healing of pressure ulcers.

Nutrition plays a key role in both the prevention and treatment of wounds. The goal in preventing pressure ulcers is to screen, identify, and provide appropriate interventions for individuals at risk for malnutrition and pressure ulcer development. A nutritional assessment should be completed both for individuals who are at high risk for developing pressure ulcers and for those who currently have wounds. The data derived from the assessment should be used to develop the nutritional section of the wound treatment and/or care plan. The nutritional interventions selected to manage the current condition should be individualized, based on standard guidelines, and reviewed as new research data become available.

Impact of Undernutrition/Malnutrition

The National Pressure Ulcer Long-Term Care cohort study of 1,524 residents in 95 nursing homes noted a higher-pressure ulcer incidence among frail residents and those who had more severe illness, low BMI, significant weight loss, and difficulty eating independently.[1] A study demonstrated that weight loss and/or preexisting malnutrition was a positive predictive variable in all eight major surgery–associated "never events" defined by the Centers for Medicare and Medicaid Services (CMS) including pressure ulcers where the odds increased 3.8 times.[2] Studies diagnosing malnutrition have used various criteria, such as anthropometric benchmarks, biochemical tests, or physical assessment parameters, to define malnutrition often failing to differentiate between undernutrition and severe malnutrition.[3–5] Malnutrition was defined by the Agency for Healthcare Research and Quality (AHRQ) as one of the common syndromes associated with increased risk for mortality and institutionalization that may be impacted by primary and secondary preventions.[6] Malnutrition is defined as any nutritional imbalance[7]; therefore, even overweight or obese individuals who experience a severe acute illness or major traumatic event are at risk for malnutrition. The consensus statement of the Academy of Nutrition and Dietetics (Academy) and the American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) recommends a set of diagnostic characteristics be used to identify and document adult malnutrition.[5]

Chronic starvation occurs solely as a result of inadequate nutrient intake as metabolism adapts by lowering energy expenditure, thus protecting lean body mass (LBM). Inflammation is the body's method of protecting itself when metabolic stress arises from the impact of injury, illness, or infection. Inflammation is necessary to remove damaged cells or pathogens and begin the healing process. The pathophysiology of malnutrition and the inflammatory process are associated with declining nutritional status and health. Diseases such as diabetes mellitus (DM), cardiovascular diseases, arthritis, and cancers cause chronic inflammation that is sustained, low grade, and persistent. Elevated energy expenditure and catabolism of LBM is associated with chronic inflammation. Individuals with severe acute illness or trauma experience severe inflammation and increased metabolic stress, which lead to increased energy expenditure and nitrogen excretion. This process triggered by the acute-phase inflammatory response leads to malnutrition and can limit the effectiveness of nutritional interventions.[8] The anorexia accompanying inflammation promotes continued loss of lean tissue, especially if nutrient intake is inadequate. Jensen describes the

severity and length of inflammation leading to the loss of LBM and physical function as disease-related malnutrition.[8] Individuals with chronic or acute disease/injury and inflammation have elevated inflammatory markers (tumor necrosis factor, interleukin-6, and alpha interleukin-18), reduced cell mass, and loss of lean body protein.[9] LBM is vital for wound healing, muscle strength, plus immune and organ function. An international guideline committee composed of members from A.S.P.E.N and the European Society for Enteral and Parenteral Nutrition (ESPEN) recommended an etiology-based approach for diagnosing adult malnutrition, which was adapted by the Academy and A.S.P.E.N.[8] Figure 10-1 etiology-based malnutrition syndromes, is based on Jensen's proposal that malnutrition is driven by various etiologies and degrees of inflammation.

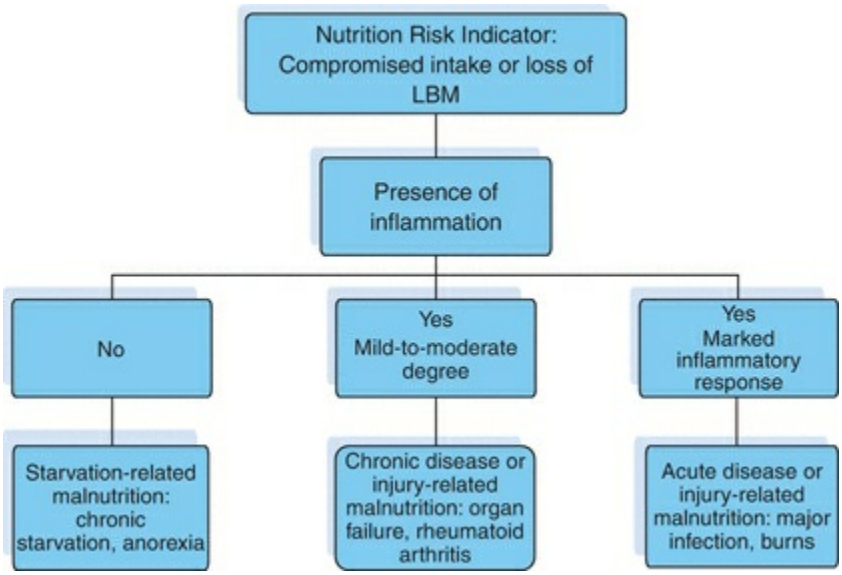

Figure 10-1. Etiology-Based Malnutrition. Adapted with permission from White, J., Guenter, P., Jensen, G., et al. "Consensus Statement of the Academy of Nutrition and Dietetics/American Society for Parenteral and Enteral Nutrition: Characteristics Recommended for the Identification and Documentation of Adult Malnutrition (Undernutrition)," *Journal of the Academy of Nutrition and Dietetics* 112:730, 2012. Copyright © 2012 American Society for Parenteral and Enteral Nutrition and the Academy of Nutrition and Dietetics. Published by Elsevier Inc. All rights reserved.

The Academy and A.S.P.E.N consensus statement recommends identifying two or more of the following six characteristics when diagnosing malnutrition[5]:

- Insufficient energy intake based on individual's estimated needs

- Weight loss
- Loss of muscle mass
- Loss of subcutaneous fat
- Localized or generalized fluid accumulation that may sometimes mask weight loss
- Diminished functional status as measured by handgrip strength

Implementing this standardized method to diagnose the nutritional status of adults should become part of the interdisciplinary team's (IDT) protocol for prevention and/or treatment of individuals with wounds. Nutritional screening supports the concept of a standardized method to diagnose malnutrition.

Nutritional Screening

Nutritional screening is the process of identifying characteristics that are known to be associated with nutrition problems. Its purpose is to pinpoint individuals who are malnourished or at nutritional risk and then to determine appropriate interventions based on the findings. Screening tools should be cost-effective, validated for the population served, easy for the practitioner to use, and acceptable to the individual. Any qualified member of the healthcare team, including the registered dietitian, nutritionist (RDN) dietetic technician, registered (DTR), nurse, physician, or other qualified health professional, can complete a nutritional screening tool. The result should be documented in the medical record and communicated to the RDN and IDT.

A number of validated nutritional screening tools are available, including the Mini Nutritional Assessment (MNA), the Malnutrition Universal Screening Tool (MUST), the Malnutrition Screening Tool (MST), and the Subjective Global Assessment (SGA).[1] The MNA screens six areas, recent food intake, weight loss mobility, psychological stress, neuropsychological problems, and body mass index (BMI), and assigns each a numerical score. The total score defines the risk of malnutrition. It has been validated in the elderly population in community and long-term care settings.[10,11] Research comparing the nutritional status of individuals with and without pressure ulcers concluded that the MNA is easy to use for assessing older adults with pressure ulcers and multiple comorbidities.[12] Tsai et al. in a comparative study of six screening tools used to sample elderly people established the MNA had a sensitivity of 98.1% and

specificity of 75% for identifying nutritional risk.[13]

The Malnutrition Universal Screening Tool (MUST) has been validated in acute care, long-term care, and community settings. Poulia et al. concluded that the MUST was the most appropriate tool for evaluating the risk of malnutrition in older adults admitted to hospitals, with a high sensitivity (87.3%) and a high negative predictive value (75%) for identifying nutritional risk.[14]

The Nutrition Risk Screening (NRS) 2002 is also a validated screening tool for adults in hospitals.[15] The Short Nutritional Assessment Questionnaire (SNAQ) has been validated in both the hospital and residential care. Weight change, appetite, supplements, and tube feeding are the parameters of the SNAQ.[16]

When the screening tool triggers risk for or actual malnutrition, undernutrition, or unintended weight loss, a timely referral to the RDN is critical for nutritional assessment and intervention. Conditions that require immediate nutritional assessment and intervention include:

- Unintended weight loss: 5% in 30 days, or 10% in 180 days
- Disease states and conditions: diabetes, malabsorption, dementia, chronic obstructive pulmonary disease (COPD), cancer, or renal disease
- Immobility and inactivity: hip fracture, spinal cord injury, and stroke
- Chewing and swallowing difficulties (dysphagia) resulting from stroke, Parkinson's disease, cerebral palsy, or other conditions
- Appetite decline, anorexia, and poor food and fluid intake
- Adverse effects of medications

Altered mental status often limits an individual's ability to eat independently or to comprehend the importance of consuming a balanced diet. Advanced dementia can result in weight loss, dysphagia, and malnutrition. When individuals become incapable of responding to caregivers' assistance to nourish them, this can lead to unintended weight loss, which may in turn increase their risk for pressure ulcer development.

Immobility affects an individual's ability to purchase food, prepare meals, or travel to a restaurant or congregate setting for meals and sometimes affects ability to enjoy food or metabolize food properly. This may result in the consumption of a diet lacking in the proper nutrients. For example, hip fracture and spinal cord injury restrict mobility, and hip fracture often results in increased pain that makes it difficult for the

individual to concentrate on consuming healthy meals. Functional limitations, such as difficulty chewing or swallowing, affect the individual's ability to ingest adequate calories and fluids. Sensory problems, such as declining hearing and vision, compromise a person's communication skills, often resulting in reduced caloric intake.

Nutritional Assessment

Nutritional assessment is a systematic process of obtaining, verifying, and interpreting data in order to make decisions about the nature and cause of nutrition-related problems. It is an ongoing process that involves initial data collection followed by continued reassessment and analysis of the individual's status compared with specific criteria.[11] The Academy of Nutrition and Dietetics Nutrition Care Process (NCP), which is designed to improve the quality and consistency of individualized nutrition care for individuals, should be followed by RDNs and DTRs in all healthcare settings.[17] The NCP consists of four steps: nutritional assessment, nutritional diagnosis, nutritional intervention, and nutritional evaluation and monitoring. RDNs and DTRs are the only healthcare professionals who utilize the NCP. Table 10-1 describes the Nutrition Care Process.

Table 10-1 Nutritional Care Process

Steps	Definition
Nutritional assessment:	A systematic method for obtaining, verifying, and interpreting data needed to identify nutrition-related problems, their causes, and significance. Nutritional assessment is an ongoing process, involving initial data and continual assessment as conditions change.
Nutritional diagnosis:	A nutritional diagnosis is *not* a medical diagnosis. Nutritional diagnosis involves diagnosing and labeling a nutritional problem that the nutrition and dietetics professional is responsible for treating independently. An example of a diagnosis is: Unintentional weight loss related to oral intake not meeting energy needs as evidenced by weight loss of 5% in 3 wk.
Nutritional intervention:	Planned action intended to change/solve a nutritional diagnosis/problem or aspect of health status for an individual (and his or her family/caregiver), a target group or the community at large. The RDN and/or DTR collaborates with the individual/family and interdisciplinary team prior to recommending an individualized nutritional intervention. Example here: Provide six small meals per day plus high-calorie supplement at bedtime. Weigh weekly. Monitor intake and weights.
Nutritional evaluation/ monitoring:	Method used to quantify progress made and whether goals/expected outcomes are being met relevant to the nutrition diagnosis. Monitoring and evaluation is an ongoing process.

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Nutrition Terminology Reference Manual: Standardized Language for the Nutrition Care Process, 4th ed. Chicago, IL: Academy of Nutrition and Dietetics, 2013. Copyright © 2013.

The NCP identifies five domains for nutritional assessment: Food–Nutrition Related History, Biochemical Data, Anthropometric Measurements, Nutrition-Focused Physical Findings, and Client History. Nutritional assessment also includes interpretation of data from the screening process as well as a review of entries from other disciplines (such as speech, occupational, or physical therapy) that may affect the evaluation process. Nutritional assessment leads to identification of a nutritional diagnosis or problem that can be resolved or improved through a treatment (nutritional intervention).

Food–Nutrition Related History

The RDN should evaluate the adequacy of the individual's current and previous food intake and meal pattern including any supplements, food intolerances, allergies, and cultural food preferences.

As part of the nutritional assessment, observation during mealtime gives the healthcare professional the opportunity to determine whether the individual has self-feeding, chewing, or swallowing problems that may require either speech or occupational therapy screening and/or treatment. Inability to feed oneself has been identified as a risk for unintended weight loss.[18] Adequate consumption of food and fluid is also a concern for those with swallowing problems, which places them at risk for malnutrition and pressure ulcers. The speech language pathologist (SLP) screens, assesses, and defines the food texture and fluid consistency as well as the need for individualized feeding techniques such as positioning for ease of swallowing. These interventions are implemented by the dining services department and nursing staff and monitored and evaluated by the RDN. For example, individuals may require thickened liquids to prevent aspiration. The occupational therapist determines the appropriate self-help feeding devices to promote eating independence. Similarly, the physical therapy sessions often result in the need for both increased calories and fluid, which the RDN will calculate and arrange to provide in the form of food and/or supplements at appropriate times.

Medications can influence an individual's nutritional status and have been identified as a cause of weight loss.[19] Drugs may either inhibit or induce metabolism of a nutrient or increase the excretion of a nutrient.

Medicine designed to calm and reduce agitation may in turn reduce mobility and activity levels and place individuals at risk for pressure ulcer development. Medications may increase or decrease appetite, alter sense of taste or smell, or cause gastric disturbances. Radiation therapy, chemotherapy, and renal dialysis can result in increased nausea and vomiting as well as decreased activity, placing the individual at nutritional risk. Drug therapy may trigger adverse effects; for example, antibiotics often cause nausea and gastric disturbances that curtail an individual's food and fluid intake. The list of medications that may influence nutrient intake or interfere with nutrition is very long and complicated.[20] Consultation with a pharmacist may be helpful.

Biochemical Data

Biochemical tests are evaluated as part of the nutritional assessment process. However, there is no one test specific to nutritional status (Table 10-2).

Table 10-2 Useful Lab Values to Screen for Hydration Status

Test	Normal Values	Dehydration	Overhydration
Osmolality	280–303 mOsm/kg	>303 mOsm/kg >320 mOsm/kg (critical)	<280 mOsm/kg
Serum sodium	135–145 mEq/L	>145 mEq/L	<130 mEq/L
Albumin	3.4–5.4 g/dL	Higher than normal	Lower than normal
Blood urine nitrogen (BUN)	7–20 mg/dL	>35 mg/dL	<7 mg/dL
BUN/creatine ratio	10:1	>25:1	<10:1
Urine specific gravity	1.002–1.028 g/mL	>1.028 g/mL	<1.002 g/mL

Albumin is a serum protein with a large body pool size distributed between the vascular and interstitial spaces, with 50% located in extravascular spaces. It functions as a carrier protein and assists in maintaining oncotic pressure. Only 5% of albumin is synthesized every day by the liver; therefore, protein intake does not affect the albumin pool on a daily basis. The majority of the changes in albumin are caused by the redistribution between the intravascular and extravascular spaces. For example, edema, ascites, and overhydration depress albumin levels, and dehydration falsely elevates albumin.

Prealbumin (transthyretin) is a transport protein for thyroxine and a carrier for retinal binding protein. Prealbumin levels are elevated in acute renal failure as the kidneys degrade it. It is also affected by some of the

same factors as albumin.

Transferrin has been touted as a marker of nutritional status, but since it is involved in iron transport, its levels are affected by iron status.

Albumin, prealbumin, and transferrin are negative acute-phase reactants that are stimulated by the inflammatory process including infection, trauma, surgery, autoimmune processes, burns, etc. Acute-phase proteins change by approximately 25% during inflammation and are expected to return to normal once the inflammatory response resolves. The response in acute and chronic inflammation is a release of cytokines such as interleukin-6 that is responsible for the production of most acute-phase proteins.[21] Cytokines are responsible for fever, inflammation of chronic disease, and cachexia. However, serum albumin levels may drop in as few as 8 hours in severe stress or inflammatory conditions even when protein intake is adequate. The Academy's Evidence Analysis Library analyzed changes in albumin and prealbumin for several conditions and indicated that these acute-phase proteins do not consistently or predictably change with weight loss, calorie restriction, or nitrogen balance. They reflect severity of the inflammatory response rather than nutritional status. [22] For this reason, a decrease in serum albumin is increasingly seen as a poor reflection of nutritional status.[5,23–27] Low serum protein levels may indicate that the individual is ill and therefore at risk for malnutrition. Frequent monitoring of weight status and oral intake would be important, especially if the individual is at risk for impaired skin integrity.

Other biochemical data that should be evaluated include glucose levels (fasting blood sugar and HgbA1c) for persons with diabetes along with renal function to determine if individuals can tolerate the increased protein that is recommended for wound healing.

Anemia, defined as a blood disorder in which there are a reduced number of red blood cells, low hemoglobin, and low hematocrit, can have a negative effect on wound healing. When anemia is present, the blood has reduced oxygen-carrying capacity leading to various side effects and negative consequences such as lower endurance, poor temperature regulation, decreased immune function, increased rates of infection, impaired memory/cognitive function, and possibly increased mortality in the elderly.[28] Since impaired wound healing may be a symptom of anemia, establishing the type of deficiency quickly and implementing a nutritional intervention is essential.

The nutritional assessment should focus on identifying risk factors that may contribute to the development of dehydration (Table 10-3). Dehydration

can have serious consequences especially for the frail older adult: decreased functional ability, predisposition to falls, infections, and fluid and electrolyte imbalances. The dehydrated individual exhibits unintended weight loss (2%, mild; 5%, moderate; and 8%, severe), dry skin and mucous membranes, rapid pulse, decreased venous pressure, subnormal body temperature, low blood pressure, and altered sensation.

Table 10-3 Signs of Dehydration

Sufficient hydration is essential for all individuals and even more so for the individual with a wound. Use the following guidelines to prevent dehydration—and to recognize and treat it should it occur.

- If the patient can drink independently, keep water or other beverages at bedside so that they are easily accessible and in a container the individual can handle easily.
- If the individual doesn't consume fluids on his or her own, offer water at least every 2 h.

Monitor dehydration by looking for:

- dry skin
- cracked lips
- thirst (often diminished in elderly individuals)
- poor skin turgor (Note: the pinch test for skin turgor may be an unreliable indicator of dehydration in elderly individuals. If you use this test, use only the skin on the forehead or sternum, and pinch gently. If well hydrated, the skin goes back into place in 2 s.)
- fever
- appetite loss
- nausea
- dizziness
- increased confusion
- laboratory values (serum creatinine, hematocrit, blood urea nitrogen, potassium, chloride, and osmolarity are increased. Sodium can be increased, normal, or low, depending on the underlying cause of dehydration.)
- decreased blood pressure
- increased pulse rate
- constipation (Note: recent diarrhea may explain the dehydrated state, and constipation is common when dehydration exists.)
- concentrated urine.

Individuals who are at risk of dehydration require careful monitoring, such as daily weights (Table 10-4). A weight loss of 2 kg in 48 hours indicates a corresponding loss of 2 L of fluid. Elderly patients, whose sense of thirst often declines, should be offered fluids more frequently. The American Medical Directors Association recently published a guideline, which may be helpful to those working with older adults for the management of dehydration.[29]

Table 10-4 Nutrition Screening

1. Screen nutritional status for each individual at risk of or with a pressure ulcer:
   - ☐ _at admission to a healthcare setting
   - ☐ _with each significant change of clinical condition
   - ☐ _when progress toward pressure ulcer closure is not observed. (Strength of Evidence = C)
2. Use a valid and reliable nutritional screening tool to determine nutritional risk. (Strength of Evidence = C)
3. Refer individuals screened to be at risk of malnutrition and individuals with an existing pressure ulcer to a registered dietitian or an interprofessional nutritional team for a comprehensive nutritional assessment. (Strength of Evidence = C)

Nutritional Assessment

1. Assess the weight status of each individual to determine weight history and identify significant weight loss (≥5% in 30 d or ≥10% in 180 d). (Strength of Evidence = C)
2. Assess the individual's ability to eat independently. (Strength of Evidence = C)
3. Assess the adequacy of total nutrient intake (food, fluid, oral supplements, and enteral/parenteral feeds). (Strength of Evidence = C)

Care Planning

1. Develop an individualized nutritional care plan for individuals with or at risk of a pressure ulcer. (Strength of Evidence = C)
2. Follow relevant and evidence-based guidelines on nutrition and hydration for individuals who exhibit nutritional risk and who are at risk of pressure ulcers or have an existing pressure ulcer. (Strength of Evidence = C)

Adapted with permission Haesler, E., ed. National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel and Pan Pacific Pressure Injury Alliance. *Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline*. Osborne Park, Western Australia: Cambridge Media, 2014.

Anthropometric Factors

Anthropometry, the measurement of body size, weight, and proportions, is used to evaluate an individual's nutritional status. A change in anthropometric values can signal problems such as wasting or edema, reflecting nutritional excess or deficit. Accurate heights and weights are critical as they are the basis for determining caloric and nutrient requirements. Adjustment or notations should be made for casts and other appliances that alter true weight.

Practice Point

Weigh your patient each time on the same scale, at the same time of day, and with minimal clothing. Be careful to subtract wheel chair weights, if weighing on a wheel chair scale. Make sure to note any casts or braces, if you are unable to remove them before taking the weight.

Body mass index (BMI) is a weight-to-height ratio derived from body weight in kilograms divided by the square of the height in meters:

$$\frac{\text{Weight (kg)}}{\text{Height (m}^2\text{)}}$$

or

$$\text{BMI} = \frac{\text{Weight (pounds)}}{\text{Height (inch}^2\text{)}} \times 705$$

A normally hydrated person with a BMI greater than 30 is considered obese (see Chapter 21, Bariatric Population). A BMI less than 19 is considered underweight for adults and may place the individual at nutritional risk.[30]

One measurement of determining the severity of undernutrition/malnutrition is any deviation from usual body weight.

$$\frac{\text{Usual weight} - \text{current weight}}{\text{Usual weight}} \times 100 = \%\ \text{Weight Loss}$$

For example, a usual body weight of 145 pounds that decreased to 137 pounds in 30-day period is a 5.5% loss.

When evaluating the severity of weight variances, it is important to determine whether the weight loss was intended or unintended. If the weight loss was unintended, it is essential to determine possible causes, such as recent surgery, diuretic therapy, or other new treatments, that may affect weight status. Unintended weight loss can signify malnutrition and increase the risk of mortality. Weight loss, especially in older adults, results in undernutrition and increases the risk of mortality. A 5% loss in 30 days or a 10% decline in 6 months increases the risk of death.[31,32]

Nutrition-Focused Physical Findings

The RDN and IDT should assess the individual's skin condition, checking for loss of subcutaneous fat as evidenced by loose skin in the extremities. Observe for listlessness, muscle wasting, and the presence of peripheral edema in the absence of cardiac disease or circulatory disorder. Dull, dry, sparse hair can signify a possible protein–energy deficiency.

The older adult is particularly prone to pressure ulcers as a result of decreased mobility, multiple comorbid conditions, poor nutrition, and loss of muscle mass. In addition to malnutrition, undernutrition, and unintended weight loss, nutritional factors thought to contribute to skin breakdown include protein deficiency, which creates a negative nitrogen balance; anemia, which inhibits the formation of red blood cells; and dehydration, which causes dry, fragile skin.

Immune function declines with age, thus increasing the risk of infection. With advancing age also comes decreased skin response to temperature, pain, and pressure. This affects the skin's elasticity and the healing process. (For further signs,[33] see Table 10-5.)

Table 10-5 Physical Signs of Malnutrition[33]

Signs	Possible Causes
HAIR	
Dull, dry, lack of natural shine, easily plucked	Protein–energy deficiency
	Essential fatty acid (EFA) deficiency
Thin, sparse, alopecia	Zinc, biotin, protein deficiency
Color changes, depigmentation, lack luster	Other nutrient deficiencies: manganese, copper
Easily plucked with no pain	Protein deficiency
Corkscrew hair; unemerged, coiled hairs	Vitamin C deficiency
EYES	
Small, yellowish nodules around eyes	Hyperlipidemia
White rings around both eyes	
Angular inflammation of eyelids, "grittiness" under eyelids, ulcerations of cornea	Riboflavin deficiency
Pale eye and mucous membranes	Vitamin B_{12}, folacin, and/or iron deficiency
Night blindness, chronic dry eye, dull or soft cornea	Vitamin A, zinc deficiency
Redness and fissures of eyelid corners, red and inflamed conjunctiva, swollen and sticky eyelids	Riboflavin/pyridoxine deficiency
Ring of fine blood vessels around the cornea	General poor nutrition
Bitot's spots (white spots in the eyes)	Vitamin A deficiency
LIPS	
Redness and swelling of the mouth	Niacin, riboflavin, iron, and/or pyridoxine deficiency
Angular fissures, scars at corner of the mouth	Niacin, riboflavin, iron, and/or pyridoxine deficiency
Soreness, burning lips, pallor	Pyridoxine deficiency
GUMS	
Spongy, swollen, bleed easily, redness	Vitamin C deficiency
Gingivitis	Vitamin C, niacin deficiency

MOUTH	
Cheilosis, angular scars	Riboflavin, iron, niacin, pyridoxine deficiency
Soreness, burning	Riboflavin deficiency
TONGUE	
Sores, swollen, scarlet, raw, "beef tongue"	Folacin, niacin deficiency
Soreness, burning tongue, purplish color	Riboflavin deficiency
Smooth with papillae (small projections)	Riboflavin, vitamin B_{12}, pyridoxine, niacin, folate, protein, iron deficiency
Glossitis	Iron, zinc, riboflavin, pyridoxine deficiency
TASTE	
Sense of taste diminished	Zinc deficiency
TEETH	
Gray-brown spots, mottling	Increased fluoride intake
Missing or erupting abnormally	Generally poor nutrition
FACE	
Skin color loss, dark cheeks and eyes, enlarged parotid glands, scaling of skin around nostrils	Protein–energy deficiency, specifically niacin, riboflavin, and pyridoxine deficiencies
Pallor	Iron, folacin, vitamin B_{12}, and vitamin C deficiencies
Hyperpigmentation	Niacin deficiency
NECK	
Thyroid enlargement	Iodine deficiency
Symptoms of hypothyroidism	Iodine deficiency
NAILS	
Brittle, banding	Protein deficiency
Spoon shaped	Iron deficiency, protein deficiency
Central line ridges	Folate, iron deficiencies, malnutrition
SKIN	
Slow wound healing	Zinc, vitamin C, protein deficiency; malnutrition
Psoriasis	Biotin deficiency
Eczema, lesions	Riboflavin, zinc deficiency
Scaling of the scalp, dandruff, oiliness of the scalp, lips, and nose	Biotin deficiency, pyridoxine, zinc, riboflavin, EFAs deficiency; vitamin A excess or deficiency
Petechiae (purple or red pinpoint hemorrhages in the skin)	Vitamin C
Dryness, mosaic, sandpaper feel, flakiness	Increased or decreased vitamin A
Follicular hyperkeratosis (gooseflesh)	Vitamin A deficiency
Dark, dry, scaly skin	Niacin deficiency
Lack of fat under skin, cellophane appearance	Protein–energy deficiency, vitamin C deficiency
Bilateral edema	Protein–energy deficiency, vitamin C deficiency
Yellow colored	Beta carotene excess, B_{12} deficiency
Cutaneous flushing	Niacin
Body edema; round swollen face	Protein, thiamin deficiency
Pallor, fatigue, depression, apathy	Iron, folate deficiency
GASTROINTESTINAL	
Anorexia, flatulence, diarrhea	Vitamin B_{12}, folate deficiency

MUSCULAR SYSTEM	
Weakness	Phosphorus or potassium deficiency, vitamin C, vitamin D deficiency
Wasted appearance	Protein–energy deficiency
Calf tenderness, absent knee jerks, foot and wrist drops	Thiamin deficiency
Peripheral neuropathy, tingling, "pins and needles"	Folacin, pyridoxine, pantothenic acid, phosphate, thiamine, B_{12} deficiencies
Muscle twitching, convulsions, tetany	Magnesium or pyridoxine excess or deficiency, calcium, vitamin D deficiencies
Muscle cramps	Chloride decreased, sodium deficiency; calcium, vitamin D, magnesium, potassium deficiencies
Muscle pain	Biotin, vitamin D deficiency
SKELETAL SYSTEM	
Demineralization of bone	Calcium, phosphorus, vitamin D deficiencies
Epiphyseal enlargement of leg and knee, bowed legs	Vitamin D deficiency
Bone tenderness	Vitamin D deficiency
NERVOUS SYSTEM	
Listlessness	Protein-energy deficiency
Loss of position and vibratory sense, decrease and loss of ankle and knee reflexes, depression, inability to concentrate, defective memory, delirium	Thiamin, vitamin B_{12} deficiencies
Seizures, memory impairment, and behavioral disturbances	Magnesium, zinc deficiencies
Peripheral neuropathy, dementia	Pyridoxine deficiency
Dementia	Niacin, vitamin B_{12} deficiencies

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The nutritional assessment information collected is documented in the medical record, and an individualized interdisciplinary care plan is established.

Table 10-4 outlines nutritional screening, assessing, and care planning recommendations from the 2014 of the National Pressure Ulcer Advisory Panel (NPUAP), European Pressure Ulcer Advisory Panel (EPUAP) and Pan Pacific Pressure Injury Alliance, Prevention and Treatment of Pressure Ulcers Clinical Practice Guidelines.[34]

Role of Nutrients in Healing

There are six major classes of nutrients: carbohydrates, proteins, fats, vitamins, minerals, and water. Through the process of metabolism, organic nutrients are broken down to yield energy, rearranged to build body structures, or used in chemical reactions for body processes.

Calories

Individuals at risk of malnutrition and pressure ulcers or who currently have wounds require additional calories to compensate for the increased metabolic stress due to hypermetabolism. Stress as a result of injury, surgery, burn, fracture, or wounds results in depletion of the nutrient stores required for healing.

Carbohydrates provide energy and prevent gluconeogenesis, which occurs when the body is forced to convert protein stores for energy. Approximately 50% to 60% of an individual's total caloric intake should come from carbohydrates. Glucose is the main source of fuel for collagen synthesis required for wound healing. The current recommendation for calories for individuals with pressure ulcers is 30 to 35 calories/kg/body weight[34] (Table 10-6).

Table 10-6 Function and Sources of Nutrients

Nutrient	Function	Source
Calories	Supply adequate energy, prevent weight loss, preserve lean body mass	Carbohydrate, protein, and fat are all sources of calories. Carbohydrate and protein provide 4 calories per gram, and fat provides 9 calories per gram. Alcohol is also a source of calories at 7 calories per gram; however, it does not provide any nutrients.
Carbohydrates	Deliver energy, spare protein	Whole grains, fruits, and vegetables, with complex carbohydrates the preferred source due to their nutrient density.
Proteins	Builds muscle, ligament, skin cells, blood, draws fluid into the capillary bed. Edema develops without adequate protein. Protein transports lipids, vitamins, minerals, and oxygen. Protein fibers form clots to slow bleeding.	Meats, fish, poultry, eggs, legumes, nuts, seeds, soy, and dairy products
Fat	Most concentrated energy source carrying the fat-soluble vitamins Provides insulation under the skin and padding for bony prominences	Meats, eggs, nonskim dairy products, butter, margarine, mayonnaise, and vegetable oils
Fluids	Solvent for minerals and vitamins, amino acids, and glucose Help maintain body temperature and transport materials to cells and waste products from cells	Water, juices, milk, broth, and other beverages; anything that is liquid at room temperature (such as gelatin, ice cream sherbet, ices); fruits and vegetables contain ~95% water.
Vitamin C Vitamin A	Water-soluble, noncaloric organic nutrient essential for collagen formation and iron absorption Fat-soluble, essential for protein synthesis, immune function, maintenance of epithelium	Citrus fruits and juices, tomatoes, strawberries, potatoes, tomatoes, broccoli, peppers, and fortified juices Deep green and yellow vegetables and fruits such as sweet potatoes, kale, spinach, carrots, apricots, beef liver, and fortified milk
Minerals: Zinc Copper Iron	Inorganic, noncaloric nutrients Cofactor for collagen formation, metabolizes protein, and assists in immune function, liberates vitamin from the liver Assists in the formation of red blood cells and is responsible for collagen cross-linking and erythropoiesis Oxygen, hemoglobin transport, collagen formation	Meats, liver, eggs, dairy products, and seafood Nuts, dried fruit, organ meats, dried beans, whole grain cereal Liver, meats, fish, and poultry

Protein and Amino Acids

Protein accounts for 17% of an individual's body weight, mainly in the form of lean tissue, muscle, and connective tissue. Half of the body's protein consists of collagen, actin, and myosin, which provide structure to the body, and hemoglobin, which transports oxygen to the cells. Protein is responsible for repair and synthesis of enzymes involved in wound healing, cell multiplication, and collagen and connective tissue synthesis. It is essential for promoting positive nitrogen balance, and increased protein levels have been linked to improved healing rates.[35,36] The recommended dietary

allowance (RDA) for all adults, including older adults, is 0.8 g/kg/body weight. Current evidence supports the concept that LBM can be better maintained when an older adult consumes higher levels of protein in the range of 1.0 to 1.2 g/kg/d.[37–39] A report by the PROT-AGE Study Group suggested that individuals with severe injury or illness might need 2.0 g/kg/body of protein daily.[40] The 2014 NPUAP/EPUAP/PPPIA guideline recommendation is 1.25 to 1.5 g protein/kg body weight for an individual assessed to be at risk for a pressure ulcer and malnutrition and for individuals with pressure ulcers, when it is compatible with goals of care.[34] Clinical judgment should be used to determine individual protein requirements, considering the number and severity of wounds, renal function, and comorbidities, as well as tolerance to interventions. For example, high protein levels may not be appropriate for individuals with chronic kidney disease.

When the body is under stress, glutamine, cysteine, and arginine become conditionally indispensable amino acids. L-arginine is composed of 32% nitrogen, and its function is to stimulate the insulinlike growth factor that is involved in wound healing. Arginine enhances immune function and wound collagen deposition in healthy elderly people.[41] A 12-week RCT study by Cereda et al. in four nursing homes concluded that the rate of pressure ulcer healing accelerated when a nutritional formula enriched with protein, arginine, zinc, and vitamin C was consumed daily for at least 8 weeks.[42] Another RCT by van Anholt et al. investigated the same formula used in the Cereda study to improve healing in well-nourished older adults with stage III and IV pressure ulcers. Participants in the trial were from eight healthcare centers, hospitals, or nursing homes in four European countries. The study concluded that a high-protein supplement with arginine and micronutrients might be associated with improved healing rate in older adults who were not malnourished.[43]

Fats and Fatty Acids

Fat is the most concentrated source of energy and provides a reserve source of energy in the form of stored triglycerides in adipose tissue. Fat calories should constitute 20% to 25% of total caloric intake. Lean meats, poultry, fish, low-fat dairy products, and vegetable oils are appropriate sources of fat.

Fat-Soluble Vitamins

Fat-soluble vitamins A, D, E, and K remain in the liver and fat tissue of the body until used. Because the body does not excrete excess fat-soluble vitamins, the risk of toxicity from overdose exists.

Practice Point

Older adults may have a difficult time consuming adequate amounts of calcium, vitamin D, and Vitamin B_{12} without supplementation. A multivitamin with minerals supplement daily is appropriate for older adults. However, if a vitamin or mineral deficiency is confirmed or the diet intake is extremely inadequate, additional supplementation may be warranted.

Vitamin A is responsible for epithelium maintenance. It also stimulates cellular differentiation in fibroblasts and collagen formation. Vitamin A deficiency, which is uncommon, may result in delayed wound healing and increased susceptibility to infection. The wound healing process may be compromised for individuals taking steroids; however, there is limited research to support mega doses of vitamin A to accelerate healing.

Vitamin E is an antioxidant and is responsible for normal fat metabolism and collagen synthesis. Vitamin E deficiency does not appear to play an active role in wound healing, [44] and it impedes the absorption of vitamin A by reducing the rate of hepatic retinyl ester hydrolysis.[45]

Water-Soluble Vitamins

Water-soluble vitamins C and B play a role in wound healing. Vitamin C is essential for collagen synthesis. Collagen and fibroblasts compose the basis for the structure of a new wound bed. A deficiency of vitamin C prolongs healing time and contributes to reduced resistance to infection.[46] However, there is no clinical evidence that wound healing is improved by providing doses of vitamin C above the dietary reference intake (DRI) of 70 to 90 mg/d. A multicenter, blinded trial of 88 patients with pressure ulcers who were randomized to either 10 or 500 mg of vitamin C twice daily failed to demonstrate any improved healing or closure rate between groups.[47]

Coenzymes (B vitamins) are necessary for the production of energy from glucose, amino acids, and fat. Pyridoxine (vitamin B_6) is important for maintaining cellular immunity and forming red blood cells. Thiamine and riboflavin are needed for adequate cross-linking and collagenation, but their effect has not been demonstrated in pressure ulcers.

Minerals

Minerals also contribute to a patient's well-being. Zinc, a cofactor for collagen formation, also metabolizes protein, liberates vitamin A from storage in the liver, interacts with platelets in blood clotting, and assists in immune function. Deficiency may occur rapidly through wound drainage or excessive gastrointestinal (GI) fluid loss or from long-term poor dietary intake. Albumin transports zinc through the body, so zinc absorption declines as plasma albumin declines (e.g., with infection, sepsis, or trauma). No clinical evidence exists to support supplementation (such as with zinc sulfate 200 to 300 mg daily, which contains more than 50 mg of elemental zinc). In a small study of individuals with pressure ulcers, no effect on ulcer healing was seen at 12 weeks in zinc-supplemented versus non–zinc-supplemented individuals.[48] The DRI for zinc is 8 to 11 mg, and the maximum daily intake or tolerable upper intake level for elemental zinc is 40 mg.[49] High serum zinc levels may inhibit healing, impair phagocytosis, and interfere with copper metabolism.[50]

Copper is essential for preserving the strength of the skin, blood vessels, and epithelial and connective tissue throughout the body. The ratio of copper to zinc is clinically more important than the concentration of either of these trace minerals.

Iron is needed for hemoglobin, collagen formation, and oxygen transport. Iron is essential for new cell generation, amino acids, and hormones. Individuals who consume a diet low in nutrient rich foods, those who are food insecure, or individuals with poor nutrient absorption or metabolism may not be consuming an adequate diet to meet established nutritional reference standards.[51]

A multivitamin with 100% of the DRI for minerals is the general recommendation if the diet is inadequate or if deficiencies are suspected or confirmed. Many of the oral supplements, enteral formulas, and fortified foods recommended for individuals with wounds contain additional micronutrients that should be considered before recommending additional supplementation.[34]

Water

Water constitutes about 60% of the adult body weight. It is distributed in the body in three fluid compartments (intracellular, interstitial, and intravascular). Water serves many vital functions in the body, including:

- aiding in hydration of wound sites and in oxygen perfusion
- acting as a solvent for minerals, vitamins, amino acids, glucose, and other small molecules and enabling them to diffuse into and out of cells
- transporting vital materials to cells and removing waste from cells.

Individuals with draining wounds, emesis, diarrhea, elevated temperature, or increased perspiration need additional fluids to replace lost fluid. Individuals on air-fluidized beds may require additional fluids daily. Total fluid needs are met from the water content of food plus liquids. Food accounts for 19% to 27% of the total fluid intake of healthy adults.[52] Refer to Estimating How Much Fluid You Need Daily (Table 10-7).

Table 10-7 Estimating How Much Fluid You Need Daily

The following chart will guide you in deciding how much fluid you need every day to stay hydrated. Remember that every person is different and you may need more or less fluids based on your individual health condition. Your doctor and/or dietetics professional can assist you in determining your specific fluid needs.	
Weight	**Drink a Minimum of Water**
≤100 pounds	6¼ cups
100–125 pounds	7 cups
125–150 pounds	8½ cups
150–175 pounds	10 cups
175–200 pounds	11½ cups
More than 200 pounds For every 10 pounds over, add a ½ cup fluid.	_____ cups

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Nursing Home Survey and Regulations: Pressure Ulcers

The CMS, which regulates long-term care facilities, has targeted pressure ulcers, inadequate nutrition, and unintended weight loss as key survey issues. The development of a pressure ulcer in a person who was at low risk is automatically considered a sentinel event. Federal Tag 314 provides guidance to surveyors as part of the CMS *State Operations Provider Certification Manual*. The guidance contains a section on Under-Nutrition and Hydration Deficits, noting that "continuing weight loss and failure of a pressure ulcer to heal, despite reasonable efforts to improve caloric and nutrient intake, may indicate that the resident is in multisystem failure or in an end-stage or end-of-life condition warranting an additional assessment of the resident's overall condition."[53] The IDT should assist in the development of nutrition goals considering the individual's prognosis and projected clinical course (Table 10-8).

Table 10-8 Definition of High Risk for Pressure Ulcer Development

The Centers for Medicare and Medicaid Services has identified the following risk factors for the development of pressure ulcers:

- Impaired/decreased mobility and decreased functional ability
- Comorbid conditions, such as end-stage renal disease, thyroid disease, or diabetes mellitus
- Drugs such as steroids that may affect wound healing
- Impaired diffuse or localized flood flow, e.g., generalized atherosclerosis or lower extremity arterial insufficiency
- Resident's refusal of some aspect of care or treatment
- Cognitive impairment
- Exposure of skin to urinary and fecal incontinence
- Undernutrition, malnutrition, and hydration deficits
- A previously healed ulcer

Reprinted from F Tag 314. "Procedures: 483.25(c): Pressure Sores." *Federal Register* 56(187), November 2004.

Diabetes

Individuals with diabetes frequently have a higher rate of complications, including infections, which can both cause and affect poor wound healing.[54] Hyperglycemia may impair leukocyte function, thus lengthening the inflammatory process and the resolution of infection. For individuals with diabetes, a glycosylated hemoglobin (HbA1c) test is the best indicator of glucose status, as it indicates blood glucose control over the previous 3 months. Marston noted that failure to manage uncontrolled HbA1c levels impedes healing of diabetic foot ulcers.[55] Effective glycemic control requires an interdisciplinary approach that addresses key area including dietary management with educations provided by the RDN and/or certified diabetes educator.

The primary nutrition goal for individuals with diabetes is to improve metabolic control of glucose and lipids and provide the appropriate calories. The American Diabetes Association (ADA) does not recommend or endorse any single diet. The Consistent Carbohydrate Diabetes meal plan is one plan recommended by the ADA and incorporates carbohydrates daily at each meal and at snack time. This plan generally includes 50% of calories from carbohydrates, 20% from protein, and 30% from fat, with an emphasis on monounsaturated and polyunsaturated fats. The consistent carbohydrate approach has been successful in the management of type 2 diabetes in nursing homes.[56] The recommended nutrition guidelines for individuals with pressure ulcers and diabetes would be appropriate.

Chronic Kidney Disease

Individuals with chronic kidney disease (CKD) often have multiple medical conditions, such as diabetes or heart disease, which complicates the nutrient parameters of their diet. For example, a CKD diet that is limited in calories, protein, potassium, phosphorus, sodium, and fluid often results in poor dietary intake that may not meet the nutrient requirement for an individual with wounds. The Academy's position paper on individualized nutritional approaches for older adults in healthcare communities' advocates for RDNs to assess and evaluate the need for nutritional intervention tailored to each person's medical condition, needs, desires, and rights.[7]

Obesity levels have risen dramatically in the last few decades.[58] The World Health Organization (WHO) defines overweight and obesity as abnormal or excessive fat accumulation that may impair health and notes that obesity is associated with greater morbidity than being underweight.[59]

Nutritional screening and assessment are important to determine if the

obese individual is at risk for malnutrition or is already malnourished. Currently, there is no validated method for calculating caloric or protein needs for obese individuals.[50] The RDN should exercise clinical judgment when estimating nutritional needs. The nutrition plan for an obese individual with pressure ulcers is to offer adequate protein and calories to promote healing rather than restricting the diet to achieve weight loss. Once the wound has healed, the IDT can focus on a weight loss program.

Nutritional Interventions

Daily Caloric Requirements

Estimating an individual's daily caloric requirement is part of the assessment process and assists in determining the appropriate plan for the prevention and/or treatment of wounds. Indirect calorimetry is the gold standard for measuring energy expenditure estimation and quantifies stress due to illness, injury, and other medical conditions.[60] However, indirect calorimetry is not widely used outside the acute care setting. The Harris-Benedict equation measures resting metabolic rate rather than basal energy expenditure, but controversy exists over its accuracy in obese or severely undernourished individuals. Cereda and Klersy et al.'s (2011) study noted that the estimation of energy needs for individuals with pressure ulcers using Harris-Benedict formula clinicians should consider a correction factor based on the underestimation of 10% of energy needs.[61] The Mifflin-St. Jeor equation[62] is appropriate for healthy, noninjured adults as well as overweight and obese individuals.[63,64]

The management of undernutrition is extremely important for individuals with wounds such as pressure ulcers. Nutritional status, undernutrition, nutritional support, and nutritional intake are all critical to this process. The 2014 NPUAP/EPUAP/PPPIA pressure ulcer prevention and treatment guidelines provide nutritional recommendations that are a resource for practitioners when developing nutritional interventions for individual patients[34] (Table 10-9). Clinical judgment should be used when applying these guidelines, as they may not be appropriate in all circumstances.

Table 10-9 Nutrition for Pressure Ulcer Prevention and Healing

Energy Intake

1. Provide individualized energy intake based on underlying medical condition and level of activity. (Strength of Evidence = B)
2. Provide 30–35 kcalories/kg body weight for adults at risk of a pressure ulcer who are assessed as being at risk of malnutrition. (Strength of Evidence = C)
3. Provide 30–35 kcalories/kg body weight for adults with a pressure ulcer who are assessed as being at risk of malnutrition. (Strength of Evidence = B)
4. Adjust energy intake based on weight change or level of obesity. Adults who are underweight or who have had significant unintended weight loss may need additional energy intake. (Strength of Evidence = C)
5. Revise and modify/liberalize dietary restrictions when limitations result in decreased food and fluid intake. These adjustments should be made in consultation with a medical professional and managed by a registered dietitian whenever possible. (Strength of Evidence = C)
6. Offer fortified foods and/or high-calorie, high-protein oral nutritional supplements between meals if nutritional requirements cannot be achieved by dietary intake. (Strength of Evidence = B)
7. Consider enteral or parenteral nutritional support when oral intake is inadequate. This must be consistent with the individual's goals. (Strength of Evidence = C)

Protein Intake

1. Provide adequate protein for positive nitrogen balance for adults assessed to be at risk of a pressure ulcer. (Strength of Evidence = C)
2. Offer 1.25–1.5 g protein/kg body weight daily for adults at risk of a pressure ulcer who are assessed to be at risk of malnutrition when compatible with goals of care, and reassess as condition changes. (Strength of Evidence = C)
3. Provide adequate protein for positive nitrogen balance for adults with a pressure ulcer. (Strength of Evidence = B)
4. Offer 1.25–1.5 g protein/kg body weight daily for adults with an existing pressure ulcer who are assessed to be at risk of malnutrition when compatible with goals of care, and reassess as condition changes. (Strength of Evidence = B)
5. Offer high-calorie, high-protein nutritional supplements in addition to the usual diet to adults with nutritional risk and pressure ulcer risk, if nutritional requirements cannot be achieved by dietary intake. (Strength of Evidence = A)
6. Assess renal function to ensure that high levels of protein are appropriate for the individual. (Strength of Evidence = C)
7. Supplement with high protein, arginine, and micronutrients for adults with a pressure ulcer Category/stage III or IV or multiple pressure ulcers when nutritional requirements cannot be met with traditional high-calorie and protein supplements. (Strength of Evidence = B)

Hydration

1. Provide and encourage adequate daily fluid intake for hydration for an individual assessed to be at risk of or with a pressure ulcer. This must be consistent with the individual's comorbid conditions and goals. (Strength of Evidence = C)
2. Monitor individuals for signs and symptoms of dehydration including change in weight, skin turgor, urine output, elevated serum sodium, and/or calculated serum osmolality. (Strength of Evidence = C)
3. Provide additional fluid for individuals with dehydration, elevated temperature, vomiting, profuse sweating, diarrhea, or heavily exuding wounds. (Strength of Evidence = C)

Vitamins and Minerals

1. Provide/encourage individuals assessed to be at risk of pressure ulcers to consume a balanced diet that includes good sources of vitamins and minerals. (Strength of Evidence = C)
2. Provide/encourage an individual assessed to be at risk of a pressure ulcer to take vitamin and mineral supplements when dietary intake is poor or deficiencies are confirmed or suspected. (Strength of Evidence = C)
3. Provide/encourage an individual with a pressure ulcer to consume a balanced diet that includes good sources of vitamins and minerals. (Strength of Evidence = B)
4. Provide/encourage an individual with a pressure ulcer to take vitamin and mineral supplements when dietary intake is poor or deficiencies are confirmed or suspected. (Strength of Evidence = B)

Adapted from Emily Haesler, ed. National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel and Pan Pacific Pressure Injury Alliance. *Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline*. Osborne Park, Western Australia: Cambridge Media, 2014, with permission.

Strategies Enhance Oral Intake

When an individual cannot meet his or her nutritional needs through consumption of traditional meals, then additional strategies should be considered. Options include providing oral supplements between meals, adding fortified foods or between-meal snacks to meal plan. Oral nutritional supplements (ONS) include commercial products that supply nutrients such as protein, carbohydrates, fat, vitamins, minerals, and/or amino acids.

Stratton et al. (2005) pooled the results of four RCTs comparing ONS with routine care, following a normal diet and implementing routine pressure ulcer care, and one RCT comparing enteral tube feeding to routine care.[64] The results of the meta-analysis showed that oral nutritional supplementation (primarily high protein, 400 to 500 kcal, duration of 4 to 72 weeks) was associated with a significant reduction in pressure ulcer development compared to routine care. A retrospective cohort study of 1,524 nursing home residents reported that the consumption of an oral supplement was a predictor of pressure ulcer healing.[1] Additional research supports the value of providing ONS as a therapeutic intervention to promote healing.[36,42,43] Wilson et al. studied the timing of supplements and concluded that consuming ONS between meals resulted in better absorption of nutrients and less interference with meal intake.[65]

Rather than relying on commercial supplements to increase calories and nutrients, consider adding powdered milk to foods the patient is already eating, such as hot cereal or mashed potatoes. Offer small, frequent meals and snacks, such as high-calorie bars, sandwiches, Greek yogurt, and other nutrient-rich items (Table 10-10). Enhance overall caloric and nutrient intake by tailoring meals and snacks based on the person's cultural and religious preference.[66]

Table 10-10 Sample Nutritional Supplement Schedule

In order to provide variety and avoid flavor fatigue, it is important to offer variety and to rotate types and flavors of high-calorie/high-protein supplements. Here is an example of how supplements and snacks may be varied to achieve these goals.

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
2 PM ½ Egg salad sandwich 4-oz milk	**2 PM** 4-oz vanilla pudding	**2 PM** ½ chicken salad sandwich 4-oz milk	**2 PM** 4-oz chocolate cream pie	**2 PM** ½ tuna salad sandwich 4-oz milk	**2 PM** 4-oz butterscotch pudding	**2 PM** ½ peanut butter and jelly sandwich 4-oz milk
Evening 8-oz cherry vanilla shake	**Evening** 8-oz peach shake	**Evening** 8-oz orange cream shake	**Evening** 8-oz strawberry shake	**Evening** 8-oz chocolate shake	**Evening** 8-oz strawberry banana shake	**Evening** 8-oz vanilla shake

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Exploring why a patient isn't eating is the first step in helping to meet his or her nutritional needs. For example, is there an emotional or physical reason why eating is a problem? Find out if something is bothering the individual that is preventing him or her from eating. Provide an environment that reduces noxious smells, which may decrease appetite, while increasing pleasing aromas of food being prepared or other pleasant smells such as baking bread. A quiet, unhurried eating environment with frequent cueing is particularly helpful for cognitively impaired persons. Similarly, in evaluating the individual's physical ability to eat, consider the following questions:

Beverage Consumption

- How much time does it take the individual to eat? Fatigue or fear of choking may cause the patient to eat slowly.
- Does the individual have the physical ability to bring the food to the mouth? Can he or she handle eating with utensils appropriately? Neuromuscular impairments, fatigue, or decreased endurance can interfere with the patient's ability to eat independently. Consider use of assistive devices and consultation with an occupational therapist. Some may prefer "finger" foods. Even with appropriate utensils, consider whether the patient has the coordination to bring the food to the mouth.
- Can the patient see the food on the tray? Changes in the visual field as a result of stroke, cataracts, glaucoma, or diabetes may alter the ability

to see food on all or part of the tray. Arrange food so the patient can see and reach it.

- Can the individual chew? Check on the condition of the oral cavity. Provide appropriate mouth care to cleanse the mouth and stimulate the appetite. If the individual has dentures, is he or she using them and do they fit?
- Can the person swallow? Cranial nerve and other neurologic conditions can cause swallowing difficulties. Evaluate the individual for any signs of abnormal swallowing. Teach the person to direct food to the unaffected side of the mouth. Consultation from an SLP for management of swallowing difficulties and recommendations about food textures may be helpful.
- Is the individual's diet unappealing and unappetizing due to the restrictions of the diet order? Diet restrictions may result in reduced food and fluid intake. Ask the RDN to assess the value of the diet restriction, and recommend individualization to a less restrictive diet, as appropriate.

Many elderly people are chronically dehydrated, and ensuring adequate oral fluid intake can be challenging. Care providers who deliver trays should open and uncover all liquids and encourage individuals to drink beverages. A frail older adult with arthritis, for example, may not be able to open sealed containers. Fluids include broth, gelatin, ice cream sorbets, sherbet, soft drinks, juice, ice cream, or any liquid that is fluid at room temperature. Offer additional fluids at medication pass or during therapy sessions. Use creative ways to encourage daily fluid intake. For example, give ice pops in warm climates or hot soup in cooler climates.

Suggestions for meeting nutrient requirements are detailed in Table 10-11.

Table 10-11 Sample Interventions for Pressure Ulcers

All interventions must be individualized based on assessment of needs.		
	High Risk (Prevention)	**Stages 1–4, Unshakeable (Based on Individualized Assessment of Needs)**
Sample interventions for increased caloric needs	• Offer favorite foods. • Offer fortified foods (high calorie and protein as needed) • Honor food preferences • Additional interventions as needed to meet individual needs	• Offer favorite foods • Offer fortified foods (high calorie and protein as needed) • Extra margarine, gravy, and other calorie concentrated additions as tolerated with meals/snacks • 8 oz half and half or whole milk (or whole milk and half and half mixed together), milk shakes, and/or ice cream with meals • Snacks of choice between meals • House supplement between meals (preferably at least 60 min before meals) • Additional interventions as needed to meet individual needs
Sample interventions for increased protein needs	• Extra ounce of protein at each meal • 8 oz of whole milk or high-protein milk shake each meal • High protein between meal snacks (sandwiches, cottage cheese, etc.)	• Extra ounce of protein at each meal • 8 oz of whole milk or high-protein milk shake each meal • High protein between meal snacks (sandwiches, cottage cheese, etc.) • House supplement between meals (preferably at least 60 min before meals)
Sample interventions for increased fluid needs	• 12- to 16-oz favorite beverages at every meal • Offer fluid with every contact • Consider hydration cart/pass between meals. • Water or fluid of choice at the bedside	• 12- to 16-oz favorite beverages at every meal • Offer fluid with every contact • Consider hydration cart/pass between meals • Water or fluid of choice at the bedside
• **Multivitamin and mineral (up to 100% USRDI)[a]** • **Elemental Zinc ≤40 mg daily[a]**	• Daily • NA	• Daily • Reevaluate as pressure ulcer heals

[a]Sample interventions for vitamins/minerals *if* deficiencies are confirmed or suspected. These are just suggestions. All interventions must be based on individual assessment of nutritional needs.

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Offer Ad Providing Enteral/Parenteral Support

Nutritional support, enteral or parenteral feeding, is used to place the individual into positive nitrogen balance (in which the body maintains the same amount of protein in its tissues from day to day) according to the goals of care and whether it's compatible with the individual's and family's /surrogate's wishes. A tube feeding may be initiated when the ability to

chew, swallow, and absorb nutrients through normal GI route is compromised. This occurs in conditions such as stroke, Parkinson's disease, cancer, and dysphagia or when patients can't meet their nutritional needs orally. Most enteral tube feeding formulas are nutritionally complete and designed for a specific purpose. Parenteral nutrition is the delivery of nutrient solutions directly into a vein, bypassing the intestine. This form of feeding is necessary in patients when enteral tube feeding is contraindicated, is insufficient to maintain nutritional status, or has led to serious complications.

Does providing enteral feedings prevent pressure ulcers? The answer may lie in the limited number of published studies involving the use of tube feedings in patients with pressure ulcers. There was no difference in the number or healing of pressure ulcers in 49 long-term care residents with pressure ulcers who received enteral feedings for 3 months.[67] Although pressure ulcers occur frequently in patients with hip fractures, randomized clinical trials of enteral nutrition in this population haven't demonstrated success in preventing pressure ulcer development.[68] It is possible that poor tolerance of the feedings may have contributed to this result. In another study of 135 long-term care residents with severe cognitive impairment, provision of tube feedings didn't increase survival or have an apparent effect on the prevalence of pressure ulcers.[69] A meta-analysis of high-quality trials found that it was not possible to draw any firm conclusions on the effect of enteral and parenteral nutrition on the prevention and treatment of pressure ulcers.[70]

Palliative/Hospice Care

The IDT has the ethical obligation to protect life and to relieve suffering; however, the individual has the right to refuse treatment, including feeding and hydration. It is the position of the Academy that "individuals have the right to request or refuse nutrition and hydration as medical treatment."[71] Many disease conditions, such as end-stage congestive heart failure and end-stage renal failure, are considered terminal illnesses; therefore, individuals with these conditions who also have wounds are candidates for palliative and/or hospice care. Advanced dementia is now considered a terminal illness.[72] The IDT is responsible for discussing the risk and benefits of initiating or withdrawing EN with the individual and/or their families or surrogate. The RDN can explain that loss of appetite is common in terminally ill individuals and does not reduce quality of life. Minimizing

or withholding hydration has the positive effect of reducing oral and bronchial secretions, thus relieving pulmonary congestion. Several studies of withholding nutrition indicate that the physiological adaptation allows individuals not to suffer from the absence of life.[73,74] Treatment goals should be client centered, respecting each person's unique values and personal decisions.[71]

Documentation in the Medical Record

Medical nutrition therapy documentation in the medical record should include:

- amount of food consumed in both quantity (% of meal served) and quality (type of food) related to amount needed
- average fluid consumed with meals
- amount in ounces of liquid supplements or percent of snacks consumed
- ability to eat—assisted, supervised, or independent
- acceptance or refusal of diet, meals, or supplements
- current weight and percentage gained or lost
- new conditions affecting nutritional status, such as introduction of thickened liquids or new diagnosis
- new medications affecting nutritional status
- current laboratory results, if appropriate
- wound condition, stage, and progress toward healing
- current calorie, protein, or fluid requirements
- remonitoring and evaluation of care plan including revisions
- education of individual and/or family concerning the importance of nutritional interventions for healing and improving nutritional status.

Summary

Nutrition is an important consideration when treating an individual with pressure ulcers, chronic wounds, or diabetic ulcers. Early nutritional screening and assessment are key elements for diagnosing malnutrition, which can impede healing. Nutrition not only facilitates healing, but it also improves or stabilizes the individual's quality of life. The focus should be on optimal nutrition for each individual, which for some persons may be achieved by a diet that includes supplements or fortified foods and allows the individual to enjoy their favorite foods. For others, enteral and parenteral nutritional support may be necessary. The amount and type of

nutritional support provided to individuals with pressure ulcers should be consistent with both the medical goals and the individual's wishes.[72]

PATIENT SCENARIO

Clinical Data

MT is a 70-year-old woman admitted to a rehabilitation center following surgery 4 days ago for a hip fracture. She has stage IV pressure ulcer on her coccyx that she acquired while lying on her kitchen floor for several hours after she fell. Additional diagnosis includes diabetes mellitus (DM), hypertension, and osteoporosis. Her height is 60 inches, usual body weight is 225 pounds, and she currently weighs 210 pounds—a 6.5% loss in less than a week. Her intake is 50% of her 1,200 calorie diet, and she eats snack foods, regular soft drinks, and chips. Her MNA screening score is 6 indicating malnutrition, and she is referred to the RDN for a nutritional assessment.

Her biochemical data include HgbA1c of 8.0 (normal is <7% for older adults). Fasting glucose is 175 mg/dL (normal is 70 to 100 mg/d), and serum albumin is 2.0 g/dL (normal is 3.5 to 5.0 g/dL). Medications include oral hypoglycemic, diuretic, calcium, vitamin D, and pain medications. The physical therapy department is working to improve her mobility but notes her grip strength is poor, a possible indication of her malnutrition. The wound care team is addressing the appropriate treatment and support surfaces for her pressure ulcers.

Clinical Malnutrition

Acute injury–associated malnutrition (hip surgery and pressure ulcer) and chronic disease–associated malnutrition (DM)

History and Clinical Data

MNA nutritional screen indicated malnutrition. Longstanding obesity and chronic inflammation associated with DM plus current decline in food/fluid intake and historical diet of poor quality per interview with MT. Hip surgery and current stage IV pressure ulcers associated with

acute inflammatory response compounded by comorbidities.

Anthropometric Data

BMI 40.9 consistent with obesity; 6.5% weight loss not related to edema

Laboratory Data

Fasting glucose and HgbA1c consistent with DM in poor control. Low serum albumin due to the inflammation.

Dietary Intake

MT's history and current intake indicate poor quality. She also expressed dissatisfaction with her current 1,200 calorie diet. Her intake of sodium is high, carbohydrate intake is of poor quality, and her diet is low in high-quality protein, zinc, and vitamin C—all of which are required to heal her pressure ulcers.

Functional Outcome

Physical therapy notes her poor endurance during rehabilitation sessions, and her grip strength is significant for age and gender.

Nutritional Diagnosis

Severe malnutrition in context of acute injury as evidenced by reduced energy intake for 7 days and reduced grip strength for age and gender.

Nutritional Intervention

The RDN and MT worked together to develop a selective menu plan to achieve her estimated caloric needs with a focus on consistent carbohydrate choices for each meal. Per RDN's recommendation, the physician ordered a regular consistent carbohydrate diet and 30 mL of sugar-free liquid protein supplement twice a day. The supplement has 100 kcalories, 17 g of protein plus vitamin C, zinc, and arginine, which proved MT 34 g of protein in addition to the approximately 80 g in her meals. MT increased her fluid intake to at least 12 cups of fluid daily and switched from regular soft drinks to sugar-free beverages. The

physician ordered routine labs to check her glucose levels and renal function to ensure high level of protein was tolerated. Her weight is monitored weekly.

Show What You Know

1. An individual who weighs 125 pounds with a stage IV pressure ulcer and a poor appetite has a recommended daily protein requirement of:

A. 57 to 118 g.
B. 68 to 107 g.
C. 85 to 113 g.
D. 68 to 86 g.

2. Mr. B only consumes 50% of his 1,800 calorie 2-g sodium diet. He complains food has no flavor, and he continues to lose weight and his pressure ulcer is not healing. The wound care team should:

A. request RDN referral to discuss the importance of consuming his current diet to heal his wounds.
B. request RDN referral to assess his nutritional status and ask the physician to consider ordering a regular diet.
C. request the physician order a high-calorie supplement three times a day.
D. request the physician order zinc sulfate 220 mg three times a day.

3. Mr. A has a large, draining stage IV pressure ulcer, and his daily caloric requirements are estimated at 2,500 kcalories. He is unable to consume this amount of calories in three meals each day. He should be offered:

A. a high-calorie supplement with all his meals.
B. double portions of protein at each meal.
C. high-protein, high-calorie snack between meals.
D. a high-calorie supplement at breakfast.

4. Which of the following 2014 NPUAP/EPUAP/PPPIA nutrition guidelines for treatment is <u>not</u> correct?

A. Assess the adequacy of total food and fluid intake.
B. Offer 30 to 35 kcalories/kg body weight daily for an individual with a pressure ulcer.

C. Offer 25 mg elemental zinc and 500 mg ascorbic acid twice daily.
D. Offer 1.25 to 1.5 g protein/kg body weight.

5. Which of the following amino acids are considered conditionally indispensable during periods of stress?

A. Arginine and glutamine
B. Alanine and glutamine
C. Valine and arginine
D. Lysine and glutamic acid

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Pressure Redistribution: Seating, Positioning, and Support Surfaces

11

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Objectives

After completing this chapter, you'll be able to:

- discuss tissue biomechanical properties, their measurement, and their relationship to soft tissue–loading tolerance
- identify support surface characteristics related to the maintenance of tissue integrity
- describe the categories, functions, and limitations of various support surfaces
- outline an assessment process for selecting an appropriate support surface (seat cushion or horizontal support) and related interventions (positioning).

Preventing Pressure Ulcers

Multiple intervention strategies are needed to prevent and treat pressure ulcers. Managing loads on the skin and associated soft tissue is one of these strategies. A comprehensive care plan should include pressure redistribution strategies for individuals both while in bed and when seated. Properly chosen support surfaces; adequate periodic pressure

redistribution; protection of especially vulnerable bony prominences, such as the heels, sacrum, and coccyx; and consideration of special patient needs are all essential components of the care plan.

A support surface is a specialized device for pressure redistribution designed for management of tissue loads, microclimate, and other therapeutic functions. Types of surfaces include mattresses, integrated bed systems, mattress replacements, mattress overlays, and seat cushions. Unless specifically identified as a mattress or seat cushion, the term "support surface" will refer to both product categories in this chapter. Achieving a good match between the patient's needs and the performance capabilities of the support surface has a profound, positive impact on a patient's health and well-being; conversely, a poor match has an equally negative impact. Support surfaces redistribute the body's weight and protect the skin's tissue while providing for proper body alignment, comfort and, as part of a seating system, postural control during functional movement. These effects may conflict and require clinical decision making to strike a balance between protective and functional goals.

Ideally, an algorithm that incorporates an individual's characteristics, conditions, environment, and preferences should be able to guide one to formulate a recommendation for an ideal, personalized support surface. The body of evidence is improving, but the research in this area has not yet produced a validated selection algorithm that matches specific products for any given situation. Some guidance is available for some conditions, but it's not sufficient enough to replace good clinical decision-making and follow-up evaluations. Furthermore, existing clinical recommendations need to be updated regularly to reflect new research, technology, and treatment strategies as they become available.

Knowledge of a product's composition and contents is a necessary part of the selection process. Although describing the materials and components of support surface technology may be informative, it isn't always instructive. In terms of selecting a product, the information on the function or performance of the surface is most critical, regardless of composition. In a study by Krouskop and van Rijswijk, performance parameters were emphasized when identifying nine key support surface characteristics[1] (Table 11-1).

Table 11-1 Support Surface Performance Parameters

Nine parameters must be considered when evaluating the characteristics of a support surface for the patient with a wound:

- Redistribution of pressure
- Moisture control
- Temperature control
- Friction control (between patient and product)
- Infection control
- Flammability
- Life expectancy
- Fail safety
- Product reputation

Adapted from Krouskop, T., van Rijswijk, L. "Standardizing Performance-Based Criteria for Support Surfaces," *Ostomy/Wound Management* 41(1):34-44, January-February 1995. Used with permission.

Although function- and performance-based categorization of seat cushions and support surfaces isn't yet possible, the necessary data from clinical validation studies are being generated. Most significantly, standard tests for cushion and mattress performance have been and are being developed. These tests will provide researchers and industry with the tools needed to differentiate products based on relevant performance measures. This work is a global effort with participants from numerous countries. In the United States, the work is led by the Rehabilitation Engineering and Assistive Technology Association of North America's (RESNA's) Wheelchair and Related Seating Standards Committee.[2] The mattress standards development effort is led by the National Pressure Ulcer Advisory Panel's (NPUAP's) Support Surface Standard Initiative (S3I) Committee,[3] which is sanctioned by RESNA. The International Organization for Standardization (ISO) coordinates and publishes both cushion and mattress standards for worldwide use. On the clinical validation front, a recent study by Brienza et al.[4] has shown that support surface technology can have a positive effect on preventing pressure ulcers. The best we can do now is to group the devices according to the technologies and materials used in their construction and relate the characteristics of these technologies to the factors believed to have significant effects on the prevention and healing of pressure ulcers.

Soft Tissue Biomechanics

Human soft tissue consists of a variety of macrostructures, including skin,

fat, muscle, vessels, nerves, ligaments, and tendons. The relative amounts and arrangement of tissue macromolecules of the skin and supporting soft tissue are adapted to their specific functions and dictate their biomechanical properties.

In most connective tissue, fibroblasts secrete the macromolecules that make up the extracellular matrix. The matrix is made up of two main classes of macromolecules:

- polysaccharide chains of a class called glycosaminoglycans, which are usually found covalently linked to protein in the form of proteoglycans
- fibrous proteins that are either primarily structural (e.g., collagen and elastin) or primarily adhesive (e.g., fibronectin and laminin).

Glycosaminoglycans and proteoglycans form a highly hydrated, gel-like "ground substance" in which the proteins are embedded. The ground substance is analogous to glue that fills the lattice of collagen and elastin fibers, providing lubrication and shock absorbing qualities. The polysaccharide gel resists compressive forces on the matrix, while the collagen fibers along with elastin fibers provide tensile strength and resilience.

Tissue Mechanical Properties

In general, soft tissues are anisotropic, incompressible biosolid, biofluid mixtures.[5] Obviously, soft tissue contains a substantial amount of fluid and, as a result, is largely incompressible. Moreover, tissue's response to loading has both short- and long-term phases. In the short term, tissue deforms in response to load, but the fluid within tissue also moves slowly over time from areas of greater pressure to areas of lesser pressure. This time-dependent response to loading characterizes the viscoelastic behavior of tissue. This viscoelasticity influences its mechanical properties that include stress relaxation, creep, and hysteresis.[6]

These phenomena may be graphically represented as stress–strain curves. Stress is represented as the deforming force on the y axis, and the tissue strain (deformation) is plotted on the x axis. When soft tissue is suddenly deformed (strained) and the strain is thereafter kept constant, the corresponding stress induced in the tissue decreases over time. This phenomenon is known as stress relaxation (Fig. 11-1). Alternatively, creep describes the progressive tissue deformation that occurs over time when stress remains constant (Fig. 11-2). During cyclic loading, such as that

produced by a dynamic, or alternating pressure mattress, the stress–strain relationship demonstrated during the loading phase is different from that of the recovery, or unloaded, portion of the cycle. This effect is known as hysteresis.

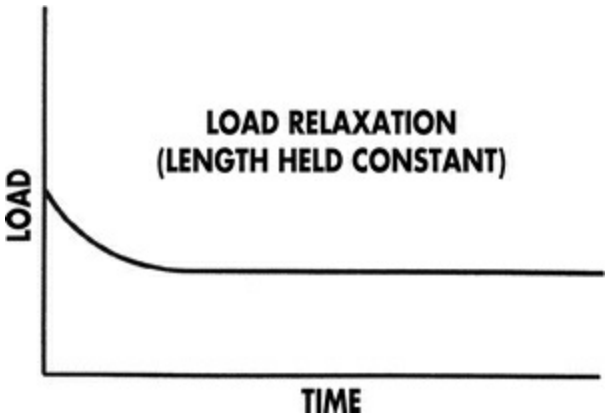


Figure 11-1. Stress relaxation phenomenon. The stress versus strain curve, shown below, illustrates the stress relaxation phenomenon. With the compression of tissue (strain) held constant, the force (stress) generated in the tissue as a result of that compression reduces over time. The degree of stress relaxation—that is, the amount of reduction in the holding force—can be determined by measuring the distance along the vertical axis between the time when the load is first applied to the time when it reaches steady state (downward sloping ends).

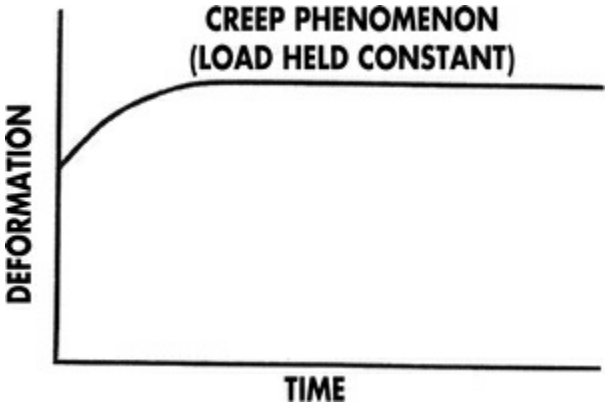


Figure 11-2. Creep phenomenon. Creep reflects the ability of tissue to resist deformation over time when the force causing the deformation remains constant. The creep phenomenon shown here indicates that the tissue progressively deforms over time without any additional force being applied. If creep were zero, the curve would be a flat line, indicating that deformation was constant over time.

Tissue Loading and Pressure Ulcer Formation

Body weight resting on bony prominences, such as the scapula, sacrum,

greater trochanters, ischial tuberosities, and heels, can cause significant concentrations of pressure at the skin's surface and in the underlying soft tissue. The pressure peaks and the pressure gradients surrounding these peaks can put the soft tissue at risk for breakdown. However, high pressure alone usually isn't sufficient to cause a pressure ulcer. Research has clearly demonstrated that the damaging effects of pressure are related to both its magnitude and duration.[7–11] Simply stated, tissue can withstand higher loads for shorter periods of time[10] (Fig. 11-3).

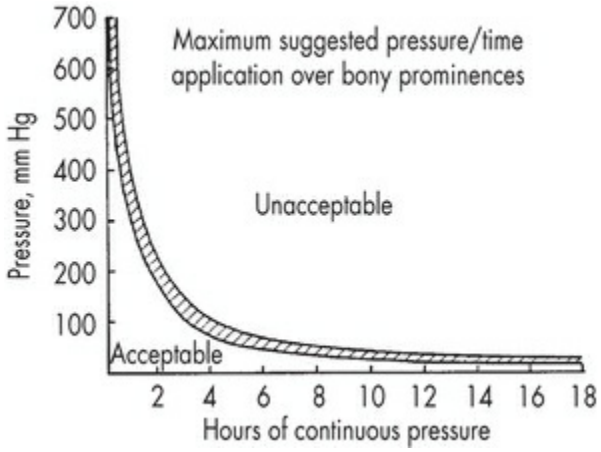


Figure 11-3. Guidelines for sitting duration. This graph provides guidelines on sitting tolerance based on the magnitude of localized pressure. (Reprinted with permission from Reswick, J., Rogers, J. *Experiences at Rancho Los Amigos Hospital with Devices and Techniques to Prevent Pressure Sores. Bedsore Biomechanics*. London, UK: University Park Press, 1976.)

How pressure and shear ultimately cause pressure ulcers is complex and not entirely understood. At the cellular level, the three mechanisms most commonly cited describing how external forces result in tissue damage are (1) ischemia resulting from tissue deformation,[9–11] (2) reperfusion injury following pressure relief,[12–14] and (3) mechanical damage to cells caused by excessive deformation.[15,16] Certainly, combinations of these factors may be significant. Several investigators have shown that in muscle tissue, external pressure causes deformation, not ischemia, which can be directly linked to injury.[17,18] Elevated skin temperature is another factor that appears to be more important than previously believed.[16,19,20]

Use of Interface Pressure to Assess Tissue

Loading

Interface pressure is the force per unit area that acts perpendicularly between the patient's body and the support surface. Interface pressure can be measured by placing a pressure sensor between the patient's skin and the support surface. This measurement approximates the pressure on the tissue. Single sensors have been used to measure local pressure over a single bony prominence; multiple sensors integrated into a mat may be used to "map" the entire body area in contact with the support surface[10,18,21–26] (Fig. 11-4).

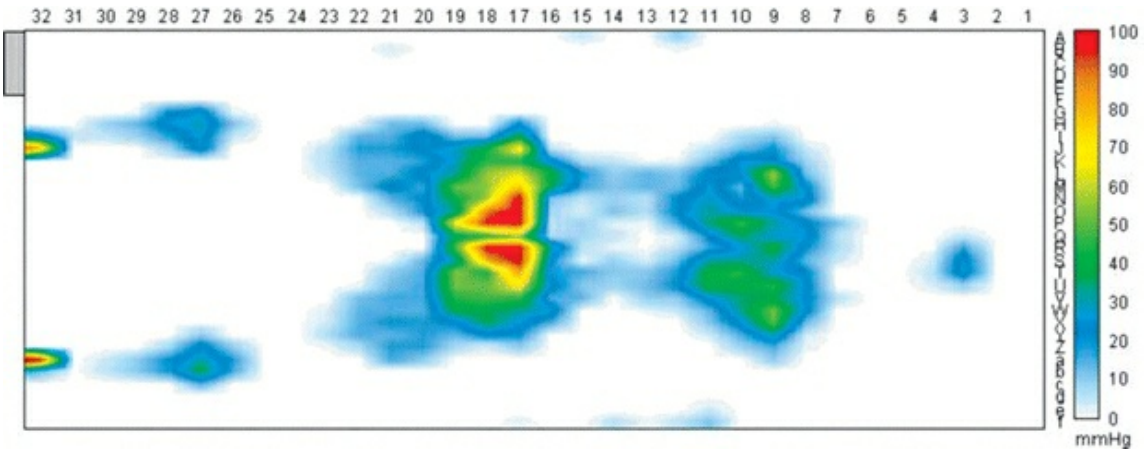


Figure 11-4. Seating, positioning, and support surfaces. Multiple sensors integrated into a mat may be used to "map" the entire body area that comes in contact with the support surface. This pressure map of a patient lying face up on a horizontal support surface shows varying degrees of pressure exerted by the patient's hees, calves, thighs, buttocks, shoulders, and head.

Interface pressure has been used extensively as a tool for predicting the clinical effectiveness of various support surfaces and for comparing products. Many research efforts have been directed toward establishing an interface pressure threshold beyond which pressure ulcers would form. However, research has not identified a specific threshold at which loads can be deemed harmful across either subject populations or various tissue body sites. This is because a tissue's loading tolerance is highly individualistic and varies according to its composition, condition, location, age, hydration, and metabolic state. As a result, interface pressure should not be used as a singular indication to use a particular support surface; however, it does have clinical utility to prevent pressure ulcers.[22,27,28] Measuring interface pressures for a particular person permits comparison of tissue loading across surfaces or postures. This can assist clinicians to identify unacceptable situations and help determine acceptable clinical

interventions individualized to the patient or client.[29]

Clinical Implications of Aging

The gross morphology of the soft tissues undergoes significant changes due to aging, including decreased moisture content and decreased elasticity manifested as rough, scaly skin with increased wrinkling and laxity. Dry, inelastic skin with larger, more irregular epidermal cells leads to decreased barrier function. These changes are reflected in the tissue biomechanical properties and have been associated with increased risk of tissue injury.

At the microscopic level, flattening of the dermal–epidermal junction (rete ridges) has been observed with the height of the dermal papillae declining by 55% from the third to ninth decade of life. As the space between the well-vascularized dermis and epidermis increases, several functional changes occur. Decreases have been reported in the area available for nutrient transfer, the number of cells within the stratum basale, and the skin's resistance to shearing. A 30% to 50% decrease in epidermal turnover during the third to eighth decade of life has also been reported. This diminution in repair rate has been quantified as both decreased collagen deposition and diminished wound tensile strength. The loss of subcutaneous fat with aging decreases our protection from injury due to pressure and shearing forces between the bony prominences and the support surface. Moreover, decreased sensory perception increases the risk of injury by mechanical forces such as pressure. And, the stiffer, less elastic, drier nature of an elderly person's skin can result in tissue that tears and bleeds more easily.

Support Surface Characteristics

Prevention of pressure ulcers is accomplished primarily by managing tissue loads. Support surfaces have been designed to reduce the effects of tissue loading by controlling the intensity and duration of pressure, shear, and friction. Also, attempts have been made to control the physical factors associated with increased risk through elimination of excess moisture and effective dissipation of heat.

Pressure Redistribution

Pressure redistribution is the ability of a support surface to distribute load over the contact areas of the human body. (This term replaces prior

terminology of pressure reduction and pressure relief surfaces.[3] The redistribution of pressure reduces the magnitude of pressure and shear forces, both of which can cause excessive tissue distortion and damage soft tissue. Pressure (stress) is defined as force per unit area; the pressure distribution is influenced by mechanical and physical characteristics of the support surface, mechanical properties of the body's tissue, and weight distribution (posture).

Immersion

Immersion is defined as the depth of penetration into a support surface.[3] The fundamental strategy for reducing pressure near a bony prominence is to allow the prominence to be immersed into the support surface. Immersion allows the pressure concentrated beneath a specific bony prominence to be spread out over the surrounding area, including other bony prominences. For example, when a person is sitting on a relatively hard cushion, a disproportionately large portion of his or her body weight is born by the tissue beneath the ischial tuberosities. On a softer surface, the ischial tuberosities and buttocks may immerse more deeply, even to the level of the greater trochanters. With greater immersion, the body weight divided by a greater surface area results in decreased average pressure. This definition of immersion doesn't distinguish between immersion resulting from compression of the support surface and immersion resulting from the displacement of a support surface's fluid components.

The potential for immersion depends on both the force–deformation characteristics of the cushion and its physical dimensions. For fluid-filled support surfaces, immersion depends on the thickness of the surface and the flexibility of the cover. For elastic and viscoelastic support surfaces, immersion depends on their stiffness and thickness. Consider how the thickness of a seat cushion might limit the potential for immersion. If the seat cushion is 1.5 inches (3.8 cm) in depth and the vertical distance between the ischial tuberosities and greater trochanters is 2 inches (5 cm), the potential for immersion isn't enough to significantly unload the ischial tuberosities.

Envelopment

Envelopment is the ability of a support surface to conform to or mold around irregularities in the body.[3] Good envelopment implies that the surface conforms to the body without a substantial increase in pressure. Examples of irregularities are creases in clothing, bedding, or seat covers,

and protrusions of bony prominences. A fluid support medium would envelop perfectly. However, surface tension plays an important role in envelopment. For example, a fluid-filled support surface such as a water bed doesn't envelop as well as water alone. The membrane containing the water has surface tension, which has a hammocking effect on irregularities of the interface. Poorly enveloping support surfaces may cause high local peak pressures, thereby potentially increasing the risk of tissue breakdown.

Pressure Gradient

Pressure gradient, also known as *pressure differential,* is defined as the change in pressure over a distance. Although various distances have been reported in the literature, pressure gradient is expressed most commonly as a change in millimeters of mercury (mm Hg) per square centimeter or square inch. When the pressure across a surface is plotted on a graph, the slope of the curve is the pressure gradient. Because the skin and other soft tissue at risk for breakdown consist of a mixture of interstitial fluid and ground substance into which structural elements are embedded, a pressure differential between adjacent regions will result in a slowing of the flow of the tissue's fluid elements from a region of high pressure to one of lesser pressure. This flow is analogous to the movement produced when one compresses the surface of a bucket of wet sand with one's hand.

Several investigators have hypothesized that the flow of interstitial fluid caused by pressure gradients is the primary factor in the development of pressure ulcers.[30,31] The flow of ground substance and interstitial fluids from an area of high pressure is believed to increase the likelihood of intercellular contact, resulting in cellular ruptures.[30–32] This theory is consistent with the classic experimental results of several researchers showing a relationship between duration of pressure application and the magnitude of pressure that results in the formation of a pressure ulcer.[10,30]

Pressure gradient is intimately linked to pressure and is affected by immersion and envelopment in a similar manner. Under certain circumstances, it's possible to have pressure gradients without high pressure, and vice versa. For example, the boundary of the contact area on a support surface necessarily demonstrates a significant pressure gradient where the pressure magnitude transitions from zero outside the area of support to a nonzero value in the supported region. Despite these significant gradients, boundary areas are typically areas of lower risk for pressure ulcer development, suggesting that pressure gradient only becomes an important factor when combined with high pressure. Further research is

needed to test and investigate this hypothesis.

Shear and Friction Reduction

The term shear can be used to reference shear stress or shear strain, and that can be a source of confusion. Shear stress refers to forces applied tangentially over an area of tissue that induce deformation (shear strain). Within tissue, stress and strain are ever-present because of the effects of gravity. Shear strain is a part of the overall tissue deformation that occurs on all supporting surfaces.[33] For example, when the head of a bed is raised or lowered, if the skin over the sacrum does not slide along the surface of the bed, or the bed does not absorb the resulting shear force by deforming in the horizontal direction, the effect will be a shearing of the soft tissue between the sacrum and the support surface. In engineering terms, the resulting shearing or deformation of the soft tissue would be referred to as "shear strain." The characteristics of the support surface affecting this potentially harmful situation are the coefficient of friction of the surface and the ability of the surface to deform horizontally.

Friction is the resistance to motion of two surfaces in contact. Friction is a force that is dependent on the characteristics of the two interface surfaces and the contact force between them. With respect to support surfaces and tissue, friction is the force that resists sliding, and this force can induce deformation, including shear strain in the tissue.[34–36]

Shear strain and frictional forces can be illustrated using the elevation of the head of the bed. When raising the head of the bed, gravity will encourage the body to slide downward to the foot of the bed. This sliding is resisted by the friction between the body and the bed linens. However, even if the skin over the sacrum does not slide along the surface of the bed, deformation of the tissue (shear strain) can occur if the bony sacrum moves with respect to the overlying skin.

Friction cannot be fully eliminated in support surfaces. In fact, friction is needed to maintain stability whether sitting, standing, or lying. Therefore, the clinical goal is to understand friction in the context or minimizing its potential damaging influences. For example, wet skin has a higher coefficient of friction than does dry skin[34–36] and, as will be discussed below, is more susceptible to damage. Pulling someone across a bed sheet during transfer exposes the tissue to significant friction. Unweighting the person before sliding will greatly minimize the friction, because, as mentioned above, friction is dependent on the force between the contact

surfaces (Fig. 11-5).

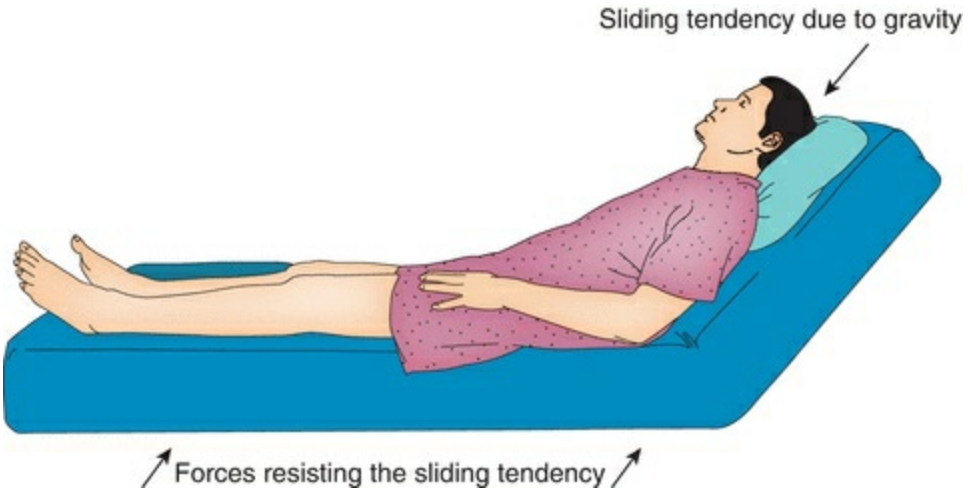


Figure 11-5. Friction and shear forces. This illustration shows the friction and shear forces acting on a person lying in bed.

Some support surface technologies protect the skin from shear better than do others. Some surfaces use a low friction interface in an attempt to minimize shear strain. Others have a low horizontal stiffness that deflects under tangential forces to minimize shear strain. Shear strain as a contributing factor for pressure ulcers is difficult to study but has been the focus of several articles.[37–39] Shear is a topic of international discussion and a task force is looking at ways to measure shear stress and strain and quantify its effects on skin.

Temperature Control

One of the extrinsic factors in pressure ulcer development, temperature, has not been definitively investigated. However, some clinical trials have shown that the application of repetitive surface loading alone induces an elevated skin temperature of 41°F or greater.[40] In addition, peak skin temperatures have been found to be proportional to the magnitude and duration of the applied pressure.[20,41,42] The conclusions of research vary depending upon the amount and duration of pressure that's simultaneously applied with varying temperatures.[20,41,42]

In addition, higher ambient temperatures have been shown to cause an increase in tissue metabolism and oxygen consumption on the order of 10% for every 1.8°F increment.[43] Thus, patients with compromised tissue already at risk for pressure ulcers may have increased demands for oxygen

in excess of their metabolic capabilities. Any increase in temperature in combination with pressure is believed to increase the susceptibility of the tissue to injury either from ischemia or reperfusion injury when the pressure is relieved.[44]

Also, increased temperature causes an exponential increase in blood perfusion, which has been associated with either an increase in core body temperature or in local skin temperature.[45,46] For example, in a study of operatively acquired pressure ulcers, the single greatest predictor of pressure ulcer development was the use of a warming blanket under the patient.[47] These findings clearly indicate the need for additional studies to definitively determine the effects of skin temperature modulation on the development of pressure ulcers. Therefore, when choosing the right support surface for the patient, its heat transfer rate is an objective performance measurement related to its ability to control temperature effects.

Animal studies have found that increased skin temperature is related to greater tissue necrosis at all layers, and a shorter period of damaging conditions is required to cause the same amount of tissue damage when the skin temperature is higher. Kokate et al.[48] first investigated increased skin temperature in a swine model. They applied 100 mm Hg of pressure for 5 hours on the back of swine at four different temperatures: 25°C, 30°C, 35°C, and 45°C. Histological examination revealed that there was severe necrosis in more layers of the soft tissue with higher temperatures. Iaizzo[49] adopted the same animal model and evaluated various combinations of skin temperature and duration of localized pressure. The histological assessment showed that temperatures as high as 45°C could cause tissue damage under localized pressure for as short as 2 hours, while sites with 25°C showed no indication of tissue damage for any duration. The protective effects of cooling the skin has also been demonstrated in humans. In particular, Tzen et al.[50] showed that temperature control reduces harmful effects of ischemia and demonstrated the effectiveness of local cooling for enhancing tissue ischemia tolerance in people with spinal cord injury[51] Lachenbruch showed that temperature is a more important factor when compared to shear relative to the intensity of the reactive hyperemic response.[37] The implication of this work is that support surfaces that control the skin temperature in the range of 25°C to 27°C facilitates pressure ulcer prevention.

Moisture Control

Moisture is another key extrinsic factor in pressure ulcer development. The sources of skin moisture that may predispose the skin to breakdown include perspiration, urine, feces, and fistula or wound drainage. Excessive moisture may lead to maceration of the skin.[52] Increases may be due to the slight increase in friction that occurs with light sweating[53] or to the increase in bacterial load resulting when alkaline sources of moisture neutralize the protection provided by the normal acid mantle of the skin.

The detrimental effect of an increase in moisture adjacent to the skin has been demonstrated by tensile tests on excised skin strips in a controlled humidity environment. In a study by Wildnauer et al.,[54] the tensile strength of the strips decreased by 75% with an increase in relative humidity from 10% to 98%. Skin with such reduced strength may be more prone to mechanical damage from shear stress and could easily be abraded.

Materials and Components Used in Support Surface Systems

The components and materials described here are the most commonly used in support surface systems and may be used alone or in combination. They include foam, gel and gel pads, fluid-filled bladders, viscous fluid, and elastomers.

Foam

Foam may be elastic or viscoelastic and may be comprised of open or closed cells. Open-cell foam is defined as a permeable structure in which the cells are interconnected and allows air or fluids to pass between them. Closed-cell foam is a nonpermeable structure comprised of isolated cells that are not connected. As a result, closed-cell foam tends to be stiffer than open-cell foam simply because it does not compress as readily due to trapped air in which there's a barrier between cells, preventing gases or liquids from passing through the foam.

Elastic Foam

Elastic foam is a type of porous polymer material that conforms in proportion to the applied load.[3] Consequently, greater loads result in predictably greater deformations, and vice versa. If time is a factor in the load versus deformation characteristic, the response is considered to be

viscoelastic, which is discussed separately. The response of support surfaces made from resilient foam is predominately elastic. Elastic foam is resilient, meaning that it returns to its nominal shape or thickness once load is removed. One indication of foam fatigue is an inability to return to its original unloaded condition.

Foam is characterized by density and stiffness. Density reflects the weight of the foam and stiffness reflects the force required to deflect the foam a given amount. Foam's stiffness and thickness impact its ability to immerse and envelop; soft foams permit greater immersion and envelopment compared to stiffer foams, but must be thicker to avoid bottoming out. Foam seat cushions are typically contoured to improve their performance.

Many foam products consist of layers of varying stiffness. This laminar design is used to optimize the performance of different foams and materials. In a laminar foam product, the softer foam is on top, thereby allowing immersion and envelopment of the body. Foam used in seat cushions can also be contoured to provide a better match between the buttocks, and the cushion increases the contact area and immersion, thereby reducing average pressure and pressure peaks[2] (Fig. 11-6). Foam is also used in combination with other materials such as elastomers, air, and fluid. Typically, a foam base offers postural stability while the elastomer, air, or fluid provides an enveloping interface. An ideal combination of characteristics for an elastic support surface is resistance that adjusts to the magnitude of compressive forces.[55,56] The support surface should be soft and thick enough to provide good immersion and envelopment but have high enough compression resistance and thickness to fully support the load without approaching maximum compression. Near maximum compression, the foam becomes much more rigid and provides too high a reactive force and a concentration of high pressure. This effect is sometimes referred to as "bottoming out." Over time and with extended use, foam degrades and loses its resilience. This decreased ability can result in higher interface pressures if it causes bottoming out. Krouskop[55] estimated that in approximately 3 years, a foam mattress wears out.

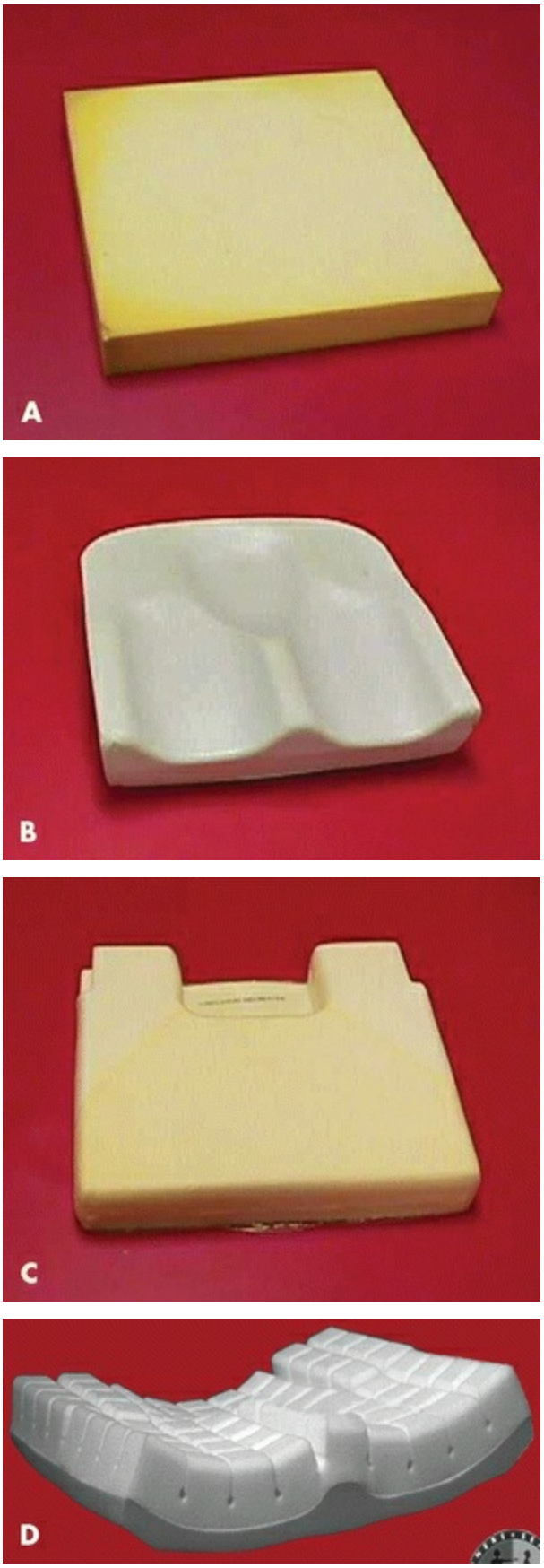


Figure 11-6. Elastic foam seat cushions. These photos show four different types

of elastic foam seat cushions: **(A)** Flat. **(B)** Coutoured. **(C)** Cut-out. **(D)** Segmented.

Foam tends to increase skin temperature because its materials and the air they entrap are poor heat conductors. Patient movement can also increase heat transfer rates. Mean temperature increases of 6.1°F (3.4°C) and a 10.4% increase in moisture at the skin surface have been recorded on foam products after 1 hour of contact.[57]

Viscoelastic Foam

Viscoelastic foam is a special type of open cell foam that compresses under load like elastic foam but has other unique qualities. Viscoelastic foam is sometimes referred to as "memory foam" because it contours to the body and its elastic response diminishes over time. This is a characteristic of stress relaxation and allows viscoelastic foam to maintain its deformed shape for a few moments before returning to its original unloaded shape (ergo, "memory foam"). Viscoelastic foam comes in a variety of stiffness so it can allow immersion similar to a similar elastic foam. The difference lies in its enveloping quality. Because of the stress relaxation, viscoelastic foam envelops better than does elastic foam. However, viscoelastic foam also exhibits creep, meaning that, over time, it will continue to deform under the same load. Furthermore, viscoelastic foam products can be temperature sensitive and become softer at temperatures near that of the human body. Mean temperature increases of 5°F have been reported for viscoelastic foam.[57] This characteristic allows the body to immerse more into the foam and can improve contact area and lower interface pressure. However, both creep and temperature sensitivity can lead to a greater potential of bottoming out. So, as with elastic foam, viscoelastic foam must be chosen according to the specific needs of the patient for both seating and mattress applications (Fig. 11-7).

Figure 11-7. Viscoelastic gel seat cushion.

Elastomers

Soft elastomers are rubber-like materials and are sometimes referred to as solid gels. For use in support surfaces, elastomers are often used on top of other materials because they tend to be heavy.

Elastomers have a high heat capacity and conductivity. This is evidenced by the many elastomeric products designed to be heated or frozen to provide relief to a sore body part. When used as a support surface, an elastomer will absorb body heat so the interface temperature will not rise as quickly.[57] This allows elastomers to feel cooler once they are sat upon. Overtime, the elastomer will eventually reach capacity and the interface temperature will rise, unless the body is repositioned—via pressure reliefs or other repositioning—to allow the heat to dissipate. Depending on the elastomer and cushion cover, moisture at the interface can also rise.[44]

Fluid-Filled Bladders and Compartments

Fluid-filled products may consist of small or large chambers filled with air, water, or other viscous fluid materials, such as silicon elastomer, silicon, or polyvinyl. The fluid flows from chamber to chamber or within a single chamber in response to movement and requires no supplemental power. The term “air-flotation” is sometimes used to describe interconnected multichamber surfaces (Fig. 11-8).

Figure 11-8. Fluid-filled products. As the photos demonstrate, fluid-filled products come in a variety of forms: **(A)** Cushion. **(B)** Mattress.

Practice Point

Be careful to maintain the correct levels of inflation in air cushions to achieve optimal pressure reduction. Underinflation causes bottoming out, and overinflation increases the interface pressure. For surfaces with viscous fluid bladders, such as seat cushions, it's important to monitor the distribution of viscous fluid and manually move it back to the areas under bony prominences if it has moved away from these areas.

Most fluid-filled products permit a high degree of immersion, allowing the body to sink into the surface. The surface conforms to bony prominences, effectively increasing the surface pressure distribution area and lowering the interface pressure by transferring the pressure to adjacent areas. These products are capable of achieving small-to-modest deformations without large restoring shear forces. In a direct comparison of interface pressures with air-fluidized and low-air-loss beds, the RIK mattress was shown to relieve pressure as effectively as the air-fluidized and low-air-loss surfaces

used in the study.[58]

Skin temperature is affected by the heat capacity and thermal conductivity of the fluid material contained in the support device. Air has a low heat capacity, whereas water has a high heat capacity. The viscous material used in the RIK mattress also has a high heat capacity, and skin interface temperature will not rise as rapidly compared to foam cushions.[58]

Given the large variety of materials used as fluid bladders, it's difficult to generalize on the moisture control characteristics of these products. However, the insulating effects of rubber and plastic used in some fluid-filled products have been shown to increase the relative humidity due to perspiration.[57]

Features of Support Surfaces

The features covered in this section can be used alone or in combination with other features. They include air-fluidized, low-air-loss, alternating pressure, and lateral rotation products.

Air-Fluidized

A support surface with an air-fluidized feature provides pressure redistribution via a fluid-like medium created by forcing air through beads as characterized by immersion and envelopment.[4] These beds were originally developed in the late 1960s for use with burn patients. These products consist of granular materials such as silicon beads encased in a polyester or Gore-Tex sheeting. The granular material takes on the characteristics of a fluid when pressurized air is forced up through them. In some models, the fluidization feature is variable, permitting individualization based on the patient's needs. These products aid in the reduction of evaporative water loss. Feces and other body fluids flow freely through the sheet; to prevent bacteriologic contamination, the bed must be pressurized at all times, and the sheet must be properly disinfected after use by each patient and at least once per week with long-term use by a single patient.[59,60]

Air-fluidized beds use fluid technology to decrease pressure through the principle of immersion while simultaneously reducing shear. Air-fluidized products permit the highest degree of immersion currently available among support surfaces. The surface conforms to bony prominences by permitting deep immersion into the surface—almost two-thirds of the body may be

immersed.[61] The immersion effectively lowers the interface pressure by increasing the surface pressure distribution area. The greater deformations possible with this technology enable the transfer of pressure to adjacent body areas and other bony prominences. Envelopment and shear force are minimized. A loose but tightly woven polyester or Gore-Tex cover sheet is used to reduce surface tension. Low surface tension enhances envelopment and minimizes shear forces.

The pressurized air in these products is generally warmed to a temperature level of 82.4°F to 95°F (28°C to 35°C); however, warming may be beneficial or harmful depending on a patient's needs. For example, heat may be harmful to patients with multiple sclerosis, but beneficial for patients in pain. The beneficial effects must be balanced against the increasing metabolic demands of the tissue.

The high degree of moisture–vapor permeability of the air-fluidized system is effective in managing body fluids (Fig. 11-9).

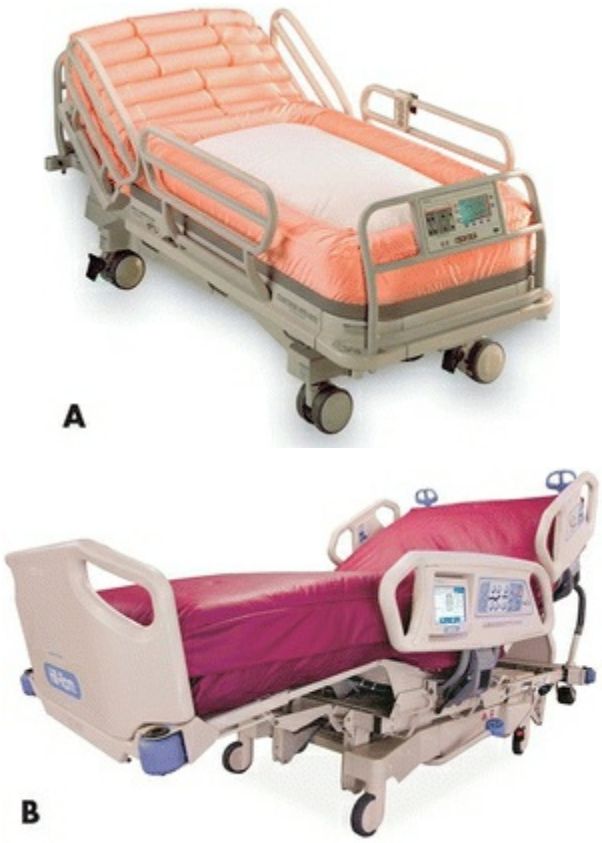


Figure 11-9. Air-fluidized **(A)** and low-air-loss **(B)** beds.

Practice Point

Air-fluidized beds are advantageous for burn patients due to their effectiveness in managing body fluids.

Low-Air-Loss

Low-air-loss is a feature of a support surface that provides a flow of air to assist in managing the heat and humidity (microclimate) of the skin[3] Low-air-loss systems use a series of connected, air-filled cushions or compartments, which are inflated to specific pressures to provide loading resistance based on the patient's height, weight, and distribution of body weight. An air pump circulates a continuous flow of air through the device, replacing air lost through the surface's pores. The inflation pressures of the cushions vary with the patient's weight distribution; some systems have individually adjustable sections for the head, trunk, pelvic, or foot areas. As with other fluid-filled surfaces, the temperature of the skin is affected by the specific heat of the fluid material. However, the constant air circulation and evaporation tend to keep the skin from overheating.

In low-air-loss systems, the patient lies on a loose-fitting, waterproof cover placed over the cushions. The waterproof covers are designed to let air pass through the pores of the fabric and are usually made of a special nylon or polytetrafluoroethylene fabric with high moisture–vapor permeability. Manufacturers have addressed the problem of skin dehydration by altering the number, size, and configuration of the pores in the covers.[50,61] The material is very smooth, with a low coefficient of friction; in addition, it's impermeable to bacteria and easy to clean.[61] Low-air-loss devices have been shown to prevent buildup of moisture and subsequent skin maceration.[62]

Alternating Pressure

Alternating pressure is a feature of a support surface that provides pressure redistribution via cyclic changes in loading and unloading as characterized by frequency, duration, amplitude, and rate of change parameters over the "active area" of the surface.[3] These systems contain air-filled chambers or cylinders arranged lengthwise or in various other patterns. Air or fluid is

pumped into the chambers at periodic intervals to inflate and deflate the chambers in opposite phases, thereby changing the pressure distribution. The frequency of the alternating pressure feature can have an effect on its use. For example, very short peak inflation, and the cycling time appear to have a dramatic effect on increasing lymphatic flow.[63]

Rather than increasing the surface area for distribution through immersion and envelopment, alternating-pressure devices distribute the pressure by shifting the body weight to a different surface contact area. This may increase the interface pressure of that area during the inflation phase.

Alternating pressure technology has the same potential as any other fluid-filled support surface to influence temperature at the interface, thus care must be taken to maintain the correct levels of inflation. The skin moisture control and temperature control characteristics of alternating-pressure surfaces also depend on the characteristics of the cover and supporting material (Fig. 11-10).

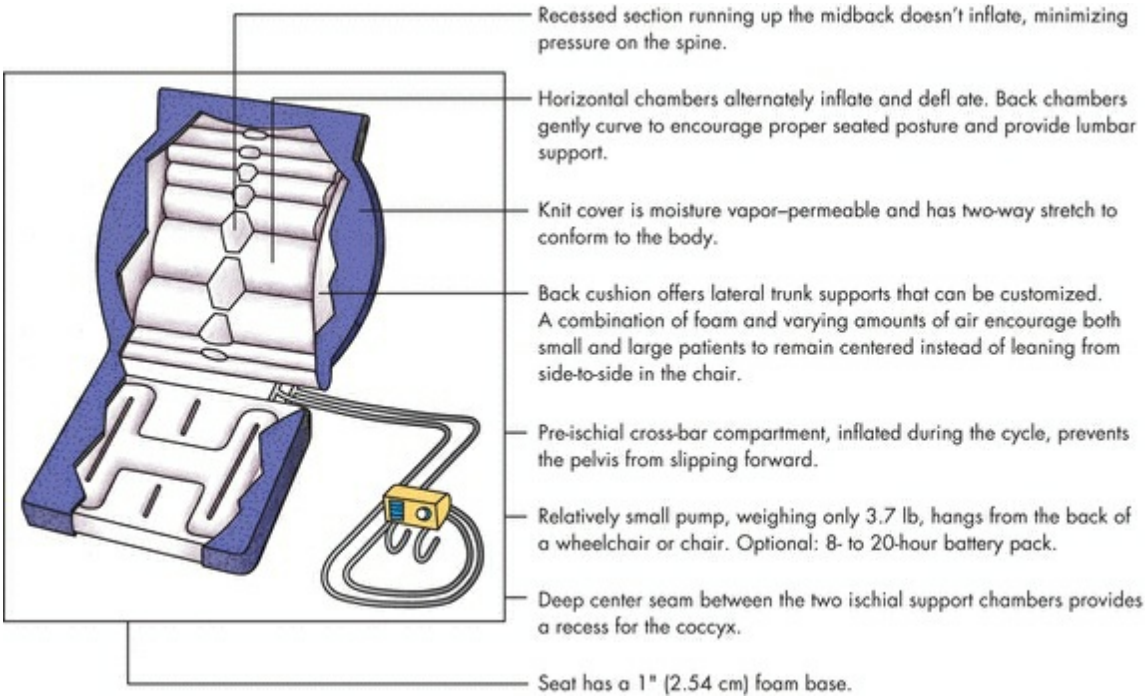


Figure 11-10. Alternating-pressure integrated cushions. The illustration shows the characteristics of alternating-pressure integrated cushions.

Lateral Rotation

Lateral rotation is a feature of a support surface that moves the patient in a regular pattern around a longitudinal axis as characterized by degree of patient turn, duration, and frequency.[3] Although these devices have been

used for several decades for other medical purposes, such as pulmonary therapy, research is conflicting on their use for pressure ulcer treatment. Lateral rotation may be continuous (i.e., on an automatic timed cycle) or manual, where the bed is rotated and locked in that position.[64] The therapy works by positioning the patient in such a way so that one lung is higher than the other to prevent pneumonia. This therapy is not intended for patients with cervical or spinal fracture, intracranial pressure instability, or long bone fractures.

According to the NPUAP, "Whenever lateral rotation features are used, the risk for shear injury exists. Shear force tangentially strains the skin (through stretching) and interrupts blood flow of the skin. Unless the individual is properly positioned and bolstered, shearing can occur with every rotation, causing a new ulcer or worsening existing ulcers"[65] (Table 11-2).

Table 11-2 Recommendations for Lateral Rotation

Lateral Rotation in Individuals Without Existing Pressure Ulcers	Lateral Rotation in Individuals With Pressure Ulcers
1. Minimize shear strain when lateral rotation features are used. (Strength of evidence = C; Strength of Recommendation = Strength of Recommendation = 👍) 2. Secure the individual with bolster pads (provided by the manufacturer) to prevent sacral shearing when lateral rotation features are selected for individuals without existing pressure ulcers. The individual should be aligned properly in the center of the surface. (Strength of Evidence = C; Strength of Recommendation = 👍)	1. Position the individual off the pressure ulcer as much as possible. (Strength of Evidence = C; Strength of Recommendation = 👍) 2. Consider alternative methods of pressure redistribution (or avoid lateral rotation beds) in individuals with sacral or buttocks pressure ulcers. (Strength of Evidence = C; Strength of Recommendation = 👍)
3. Assess skin frequently for shear injury. (Strength of Evidence = C; Strength of Recommendation = Minimize shear strain when lateral rotation features are used. 👍👍) 4. Continue to reposition the individual when using lateral rotation features. (Strength of Evidence = C; Strength of Recommendation = 👍)	3. Inspect the pressure ulcer and the periulcer skin for shear injury with every dressing change. Shear injury may appear as deterioration of the ulcer edge, undermining, and/or as increasing inflammation of periulcer skin or the ulcer. (Strength of Evidence = C; Strength of Recommendation = 👍)
5. Reevaluate the need for lateral rotation at the first sign of tissue injury. If indicated and consistent with medical needs, change to a support system with improved pressure redistribution, shear reduction, and microclimate control. (Strength of Evidence = C; Strength of Recommendation = 👍)	

Adapted from Emily Haesler, ed. National Pressure Ulcer Advisory Panel and European Pressure Ulcer Advisory Panel, Pan Pacific Pressure Injury Alliance. *Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline*. Osborne Park, Western Australia: Cambridge Media, with permission.

Matching Support to Patient Needs

Although widely used, support surfaces have neither performance standards nor criteria for function that can be tested against clinical outcomes. Indeed, the basis for effective function isn't known, or is poorly understood, for such common products as wheelchair seat cushions and horizontal support surfaces intended for skin protection and healing of wounds. Despite this, clinicians must have some basis for decision making regarding the selection of these products. The following key questions should be used to guide the decision-making process.

What Are the Patient's Specific Load Management Needs?

Regardless of body position, the first step in determining an individualized protective intervention is to perform a general physical assessment and functional evaluation. Much of this information will then be used to assess the patient's risk for pressure ulcer development.

General Physical Examination and Functional Evaluation

Patient evaluation is described elsewhere in this text; however, several additional items are germane to the selection of a support surface. These include assessing the capability of the patient for specific bed mobility (movement on the surface, ingress and egress, and ability to place supportive devices), the number of available turning surfaces, the time spent in lying in bed or sitting per day, the number of devices or pillows needed for positioning, the patient's body weight and its distribution, and the presence of contractures.

Wheelchair Cushion Selection

For selection of wheelchair cushions, the evaluation is quite extensive because cushions are part of the total seating system that also includes the wheelchair. Indeed, no cushion can perform effectively in the prevention of pressure ulcers if the wheelchair isn't properly fitted. Therefore, it's recommended that a trained seating specialist perform the seat cushion evaluation and selection.

The seating evaluation should also include a mat examination to determine the functional postural limitations of the spine, pelvis, and extremities and to determine appropriate measurements for wheelchair fitting. An extensive functional examination is also required to consider the seating and mobility needs of the individual in the immediate, intermediate, and community environments.[66–68]

Strategies for maintaining tissue integrity can be extremely complicated for spinal cord–injured patients, elderly people, and other populations with degenerative neuromuscular conditions or diseases. For example, a patient's ability to sit unsupported can be characterized by the amount of external support needed to maintain posture: hands-free, hands-dependent, or prop-sitting with external support only.[67] This capability has significant implications for compensatory functional postures, ability to reposition, and the method used for intermittent pressure relief. Figure 11-11 illustrates three common and effective strategies for intermittent pressure relief.

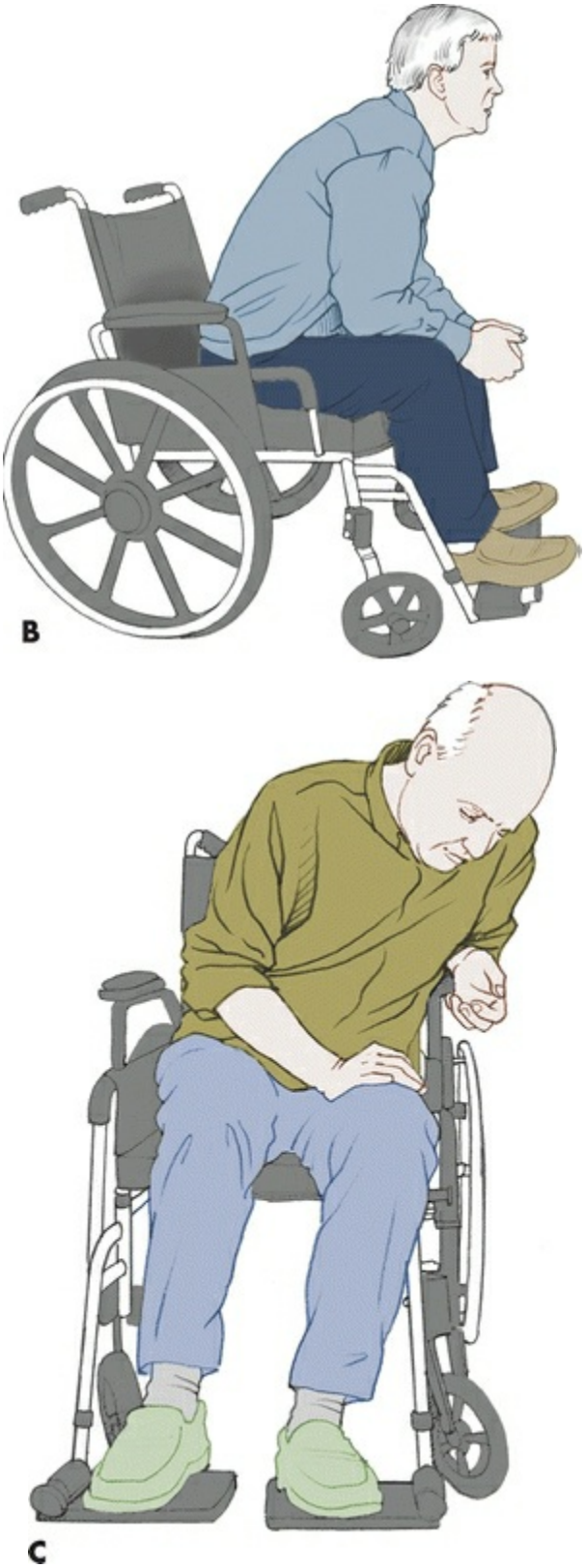


Figure 11-11 (A) A push-up pressure relief can completely off-load the buttocks but requires sufficient arm strength and trunk control. **(B)** A forward lean can unweight the ischial tuberosities. **(C)** A side-lean pressure relief off-loads the contralateral side and so must be done in both directions.

Specialty Mattress Selection

When selecting a specialty mattress, a patient's weight and the distribution of that weight are important factors. Indeed, each mattress overlay, replacement, or integrated bed unit has weight limits. For heavier patients, bariatric (Bari) beds should be used. However, for patients approaching the manufacturer's recommended weight limit, the distribution of the weight should be examined. For example, patients who are heavier in the hip region, but who don't exceed the manufacturer's weight limit, may, in fact, need to be placed on a bariatric product in order to achieve effective pressure redistribution. Patients with contractures may have their weight dispersed unevenly. For example, contractures could pull the heels toward the groin or increase flexion between other body parts creating special needs for tissue management beyond what a mattress can provide. For more information, refer to NPUAP/EPUAP/PPPIA guideline on support surfaces for Bariatric patients.[65]

In addition, patient mobility and activity levels are important consideration. If the goal is to have the patient independently get into and out of bed, this may not be possible with some support surfaces. The patient's needs may also change over time. A therapeutic mattress will not help the person in the chair all day. Therefore, revaluation of needs as the person's condition changes is important.

Assessing Risk

The most commonly used risk assessment scales for prediction of pressure ulcer incidence are the Braden, Norton, and Waterlow scales.[69–72] The sensitivity and specificity of these scales vary depending on the population setting and the position of the patient's body. For example, different Braden cutoff scores are associated with different settings (nursing home vs. intensive care unit). Risk assessment scales specifically designed for wheelchair users have not been developed.

Bergstrom and colleagues[73] reported that mattress selection based on categorizing patients via a pressure ulcer risk assessment scale produced both efficacious and cost-effective results. Patients of a large tertiary care hospital scoring nine or less on the Braden pressure ulcer risk assessment scale were provided a group 2 support surface (low-air-loss mattress) as a preventive measure. Those scoring above nine were evaluated and provided with the most appropriate surface according to individual patient needs. Results indicated that not only did pressure ulcer incidence and prevalence

drop by more than 50% when patients were categorized according to pressure ulcer risk but costs associated with overlays, replacement mattresses, and low-air-loss beds also decreased.

Similar results have been realized by studies that selected wheelchair cushions from a set of cushion alternatives based on risk assessment. Krouskop and colleagues[74] described how a seating clinic assigned risk to their clients with spinal cord injury by using such factors as gender, interface pressure, lifestyle, and stability. In 80% to 90% of the clients, cushions were selected from three alternatives, with the remaining clients being provided with other cushion types.

When assessing risk, remember that proper follow-up care is necessary to prevent pressure ulcers regardless of the cushion or bed prescribed. Skin redness/discoloration often occurs because of positioning and use and doesn't necessarily indicate that a poor surface choice was made.

Clinical Judgment

Clinicians should know how to evaluate bed or cushion performance, which includes an assessment of how adequately the product provides pressure redistribution, or if the patient is "bottoming out" or actually having soft tissue in close proximity to the hard undersurface. Clinicians should also observe any powered products for loss of power, sensor malfunctions, or disconnected hoses. Frequently checking the product's performance is especially important for patients who are unable to express their discomfort due to cognitive or communication impairment.

Interface Pressure Mapping

Interface pressure mapping—comparison of a patient's relative responses from one surface to another—can be an effective clinical tool to aid in the selection of a support surface for a specific patient. Pressure mapping may also be used to determine the relative effectiveness of modifications to the wheelchair and other positioning devices or to obtain information about pressure relief for patients with spinal cord injuries. For example, Henderson and colleagues used a pressure mapping system to compare three methods of relieving pressure in seated individuals with spinal cord injuries.[75] The positions studied were tilted back 35 degrees, tilted back 65 degrees, and forward-leaning seated posture. The results indicated that the greatest pressure relief over the ischial tuberosities was seen in the forward-leaning position, followed by the 65-degree backward tilt position.

Observing the change in pressure distribution on the mapping display allows patients without sensation to observe the effects of various weight-shifting methods and learn to consciously integrate them into their seated behavior.

Using Clinical Practice Guidelines

Clinical practice guidelines offer recommendations, based on scientific evidence and the professional judgment of expert panels, about how healthcare professionals can provide quality care. Several clinical practice guidelines include recommendations on support surface selection: NPUAP/EPUAP/PPPIA,[65] WOCN,[76] and PVA.[77]

How Does the Product Function and How Well Does It Perform?

Answers to what the product does and how it performs should be sought from a variety of sources. Sources of information for support surfaces include marketing materials, controlled clinical trials, and objective indirect data from laboratory testing or clinical studies (interface pressure and other physiological responses).

Laboratory Testing

What a support surface does has been largely determined by studies using laboratory methods to measure variables believed to be clinically relevant in pressure ulcer formation. For example, Krouskop's[78] study of foam mattress overlays provided the following recommended specifications for selection based on the results of independent laboratory testing:

- a thickness of 3 to 4 inches (7.5 to 10 cm)
- a density of 1.3 to 1.6 pounds per cubic foot as an indicator of the amount of foam in the product
- a 25% indentation load deflection (ILD) equal to about 30 pounds (the amount of force required to compress the foam to 75% of its thickness as an indicator of the foam's compressibility and conformability)
- a modulus of 2.5 or greater (the ratio of 60% ILD to 25% ILD).

Support Surface and Cushion Standards

Standards have been developed for wheelchair seat cushions and a for other support surfaces.[79–81] Publishing national or international standards for support surfaces requires that uniform test methods be developed to quantify clinically relevant characteristics. Simply stated, to make valid comparisons among products, characteristics and properties need to be measured using the same test and under the same conditions. Testing conditions should model clinically relevant parameters as closely as possible. The requirement that the tests be repeatable across laboratories in different countries means that standardized tests typically use models rather than human subjects. Clinicians, therefore, must consider test results as relative measures.

The results of standard tests often don't include pass–fail criteria. Just as there are valid reasons to purchase an automobile with an engine that only gets 15 mpg over another with a fuel efficient engine that gets 25 mpg, so are there reasons to purchase support surfaces with, for example, a lower pressure distribution characteristic but higher moisture dissipation ability over a product with a higher pressure distribution characteristic but lower moisture dissipation ability. The primary objectives of the standard tests are to characterize their different functional properties and to permit comparisons of performance among products with similar functions.

The range of products on the market places a heavy burden on clinicians to keep abreast of new technology; therefore, patients, clinicians, vendors, manufacturers, third-party payers, and researchers all benefit from standard terminology, definitions, and test methods. Clinicians would benefit from a mechanism to objectively match a seat cushion's or support surface's characteristics to the needs of their patients. Vendors benefit by being able to clearly describe products from different manufacturers in a manner understood by clinicians and patients. Testing standards aid manufacturers by guiding new product development and assisting in the redesign of existing products. In addition, standards promote quality assurance within manufacturing processes. The final potential beneficiaries of standards are third-party payers because the seat cushion and support surface market is a payer's market; that is, payer reimbursement drives the market. A validated system to test and objectively characterize support surfaces will give funding agencies an objective means for making funding decisions. In fact, standards form the basis for certain tests used by funding agencies to classify or categorize products.

Seat Cushion Standards

Standards for seat cushions are being developed by the ISO. In the United States, the effort is organized through the RESNA as an accredited standards organization for the American National Standards Institute.[79–81] One of the four interrelated working groups focuses on tissue integrity management and has developed test methods that address key performance features of cushions, including load deflection and hysteresis, frictional properties, lateral and forward stiffness, sliding resistance, impact damping, recovery, loaded contour depth and overload deflection, water spillage, and biocompatibility.[82] Additional tests measure heat and water vapor transmissibility and stability of properties with use (fatigue). Although all these tests have clinical relevance, a subset is described below. And, even though tests have been designed for seat cushions, the constructs are described in relation to both cushions and horizontal support surfaces to illustrate key concepts.

Load Deflection

Materials used in mattresses and cushions support the body by compression (foam and air), deflection (gel and viscous fluid), or tension (bladder material and fabric). Material stiffness impacts how materials deform to accommodate the body. Load deflection tests typically involve loading a cushion or support surface with a standardized indenter that mimics a part(s) of the body. Deflection into the support surface is measured as the weight on the indenter is increased.

Clinically, materials can be too stiff or too soft. When too stiff, the body does not immerse, and high pressures or instability can result. When too soft, materials can "bottom out," leading to poor support and high pressures. Foams are made with different stiffness ratings, called indentation force deflection. Many products use a combination of foam with a softer material positioned on top of a more stiff material. This configuration permits deflection of the top surface while protecting against bottoming out through the use of the stiffer bottom layer.

Because the amount of air impacts the stiffness of a support surface or cushion, products that use air are often adjustable. Too much air leads to an overly stiff surface, and too little air can lead to bottoming out. In addition to body weight, the amount of tissue, the type of tissue (hypotonic, normal, hypertonic), and a person's posture or position all influence how stiff a surface must be to adequately support a person

Frictional Properties

Friction is the result of a relationship between materials; so standardized tests for friction measure the sliding forces of one material on another. In terms of cushions and support surfaces, these standardized tests concentrate on cover materials, such as fabrics and bed linens.

Sliding Resistance

Unlike friction tests, which focus on cover material, sliding resistance tests measure the influence of the entire system, including the cover and all support materials. All users of support surfaces and cushions have to transfer onto and off the bed or wheelchair. For people who can't fully transfer, certain materials and designs facilitate easier transfers. However, if a support is too easy to slide on, stability on that surface can be compromised. Standardized tests of sliding resistance involve loading the surface with an indenter modeled on the human body or buttocks and pulling it forward or sideways. The force required to slide the indenter on the surface is measured and reflects sliding resistance.

Loaded Contour Depth and Overload Test

This test measures the immersion of a standardized indenter into a cushion surface. Clinically, the test provides two key pieces of information about seat cushions: the initial contour of a cushion and the amount of deflection that may occur when someone sits on it. The overload portion of the test adds 33% more weight and measures additional immersion of the indenter. A cushion that has "bottomed out" will not deflect further under the additional weight. A support surface should maintain a margin of safety that allows additional cushioning during overload conditions. Certain functional movements and postural adjustments, such as leaning and reaching, impart an overload condition on the surface.

As noted, several standardized tests have been developed for wheelchair cushions by the ISO. A similar approach focusing on horizontal support surfaces began years ago by the National Pressure Ulcer Advisory Panel (NPUAP). Most of these tests are or will be voluntary. Therefore, all stakeholders should request test results as a means to compare performance across products and also to encourage this voluntary testing and disclosure.

Product Effectiveness

Decades of research have produced moderate and low levels of evidence upon which to base clinical decisions concerning how and when to apply

support surfaces for prevention The Cochrane review of the clinical literature was able to provide only weak statements regarding the relative efficacy of various surface types under specific conditions.[83] The effectiveness of a support service product is measured by two methods: its efficacy in use by patients and in comparison to similar products. Rather than basing comparisons on functional classification, most studies have compared classes of products based on the product's ability to redistribute pressure.

Considering the limitations ascribed to interface pressure measurement, it may be more useful to categorize devices according to their ability to evenly distribute pressure over the contact surface area rather than ascribe any significance to the magnitude of pressure measured at a particular location. However, the most common comparison has been the use of interface pressure to compare a product against a "standard" hospital bed or mattress. When reviewing this literature, remember that the "standard" support surface probably varies from study to study. Most studies also vary with regard to patient population (for example, orthopedic or neurologic) and setting studied (e.g., acute care, intensive care, long-term care, or home care). All three independent variables (products compared, subject population, setting) affect both study outcomes and the interpretation of results. Finally, when comparing performance studies, remember that treatment studies, in which subjects already have ulcers, are fundamentally different from prevention studies.

Prevention Effectiveness

Generally, studies have shown that nonpowered, reactive support surfaces (foam, air, gel, and combinations of these materials) are more effective in preventing pressure ulcers than a "standard" hospital mattress. (The support surface category called "reactive" includes, but is not limited to, surfaces that have been described by the phrase "constant low pressure (CLP)" in the past. The reactive category is broader than the old CLP category because it also contains surfaces with powered features such as low-air-loss). Generalization of the results of studies comparing different pressure redistribution products is difficult. Most comparative studies of various nonpowered reactive products demonstrated no differences in the prevention of pressure ulcers compared to each other.[83,84]

Conclusions from research investigating the more complex technology, including low-air-loss and alternating-pressure products, are similar. Evidence suggests that both low-air-loss and alternating-pressure surfaces

are more effective than are "standard" mattresses, but comparative studies of the performance of low-air-loss and alternating-pressure products are inconsistent regarding the clinical superiority of one over the other. Moreover, comparisons of alternating-pressure to constant low-pressure products have not produced definitive differences. Caution should be taken in attempting to generalize the results from these studies because large variations in performance characteristics exist among surfaces within the same category. For example, the abilities of a support surfaces with low-air-loss features to remove moisture from the surface of the product can vary by an order of magnitude.

Because the evidence is not definitive, clinicians must carefully read the original study to generalize the results to a specific clinical situation. When considering a product's clinical applicability, the characteristics of the population, setting, and products must closely match one's clinical situation and the limitations of the study must be known. For example, consider the design of alternating-pressure products where such variables as cell height or bladder thickness and cycle timing and frequency can significantly affect product performance. One can't necessarily generalize the performance of one alternating-pressure product against another. Similarly, the results of a support surface study within acute care might not produce similar results in a home care setting using the same support surfaces.

Evidence about specific cushions and their respective effectiveness is insufficient. Contradictions also exist in the literature regarding the clinical benefits of cushions designed to reduce the risk of sitting-acquired pressure ulcers. Most research has used indirect outcomes, such as interface pressure or blood flow. Relatively few studies have measured direct outcomes related to specific types of cushions,[4,22,85,86] but these have not resulted in definitive findings of efficacy of one product over another. One facility performed two studies targeting elderly wheelchair users. The first study found no difference in the incidence of pressure ulcers in users of flat foam compared with custom-contoured foam cushions, and a subsequent study found that more users of flat foam (41%) developed ulcers compared with users of a contoured foam-viscous fluid cushion (25%), but this difference did not reach statistical significance. In a study tracking interface pressure and wheelchair cushions in elderly users, Brienza and colleagues[22] found that interface pressures were higher for subjects who developed sitting-acquired pressure ulcers compared with those who didn't develop ulcers. No definitive relationship was found between interface pressures and cushion types across these subjects.

Brienza et al.[4] performed an RCT in the long-term care setting on 232 nursing home residents comparing pressure ulcer incidence in participants given a properly sized wheelchair and randomized into a treatment group (n = 113) provided an air cell–based, viscous fluid and foam cushion; or a gel and foam cushion or a control group (n = 119) given a segmented (cross-cut) foam cushion (n = 119). The control group had a significantly greater incidence of ischial tuberosity pressure ulcers (6.7% vs. 0.9%, p = 0.04).

Treatment Effectiveness

Widely varying subject populations and care settings have complicated the ability to compare results across studies investigating the effectiveness of support surfaces in the treatment of pressure ulcers. Furthermore, a number of treatment outcome measures have been used to judge effectiveness, such as the relative or actual reduction in ulcer size (area or volume), the percentage of ulcers healed within a specified time period, and the time until wound closure. Different operational definitions have also been used for wound status, such as "healed" and "closure," making it difficult to compare equitably.

Generally, studies targeting support surfaces for ulcer treatment have produced results similar to those targeting prevention. Low-air-loss and air-fluidized surfaces have been shown to improve treatment outcomes more effectively compared with "conventional" treatment[87,88] and nonpowered foam alternatives.[89] Results of alternating-pressure surface studies are inconsistent with some studies showing a treatment effect and others showing none. No clinically significant treatment differences have been shown among similar products.

What Other Patient Needs Must Be Met?

While load management and product function are critical areas to consider in matching patient support needs, other pressure redistribution options must also be considered. These options include repositioning, heel protection, managing heel pressure, encouraging ambulation, and managing skin microclimate.

Turning and Repositioning Schedules

The frequency of repositioning required to prevent ischemia is variable and unknown, yet regular repositioning is believed to help deter the deleterious

effects of pressure by decreasing the duration of exposure.[90,91] Through the process of repositioning, the body's weight is redistributed, and new pressure areas are introduced. To provide effective pressure relief, both pressure and time must be considered. For example, in 1959, Kosiak recommended that repositioning be done in intervals of 1 to 2 hours based on the interface readings from healthy subjects.[9]

Turning schedules have been studied empirically and experimentally. Bliss[92] studied turning schedules in a spinal injury ward and found that 2 hours was adequate for some, whereas others required more frequent, and some, less frequent, turning. Two important aspects of these findings are that some patients exhibited redness after 2 hours and that many patients disliked frequent turning.

In an experimental study by Knox and colleagues,[91] variables such as temperature, pressure, and redness were monitored while people rested on a mattress for 60, 90, and 120 minutes. Some subjects exhibited redness after each of the intervals, leading the researchers to conclude that a 2-hour turning schedule might not be sufficient.

Such theoretical evidence points directly to the duration of loading as the way to maintain tissue integrity. If, however, the above experiment is only one example of patients experiencing redness after less than a 2-hour turning schedule, then it cannot be construed as the answer for all clinical practices.

While the every-2-hour turn schedule has traditionally been entrenched in clinical practice, new research has challenged this long-held belief. Recent findings from two randomized controlled trials have provided evidence that the frequency of repositioning for persons who are on a viscoelastic foam mattress may be extended for longer than every 2 hours without increasing the incidence of pressure ulcers. In a study of 838 nursing home patients who were turned every 4 hours on a viscoelastic mattress, DeFloor et al.[93] reported a reduction in stage II and more severe pressure ulcers. Vanderwee et al.[94] also studied nursing home patients who were also on a viscoelastic foam mattress (n = 235). The time in lateral position of 2 hours was compared with 4 hours in a supine position. Turning these patients every 2 hours did not result in fewer pressure ulcers compared with the group turned every 4 hours.

In addition, no knowledge exists as to how turning schedule should be affected by support services. Therefore, the best approach is to evaluate and reevaluate each patient to best determine an appropriate repositioning/turning schedule.

Positioning

According to the NPUAP/EPUAP/PPPIA guidelines[65] for repositioning individuals in bed a number of recommendations exist in regard to positioning for management of tissue loads.

1. Use the 30-degree tilted side-lying position (alternately right side, back, left side) or the prone position if the individual can tolerate this and her/his medical condition allows

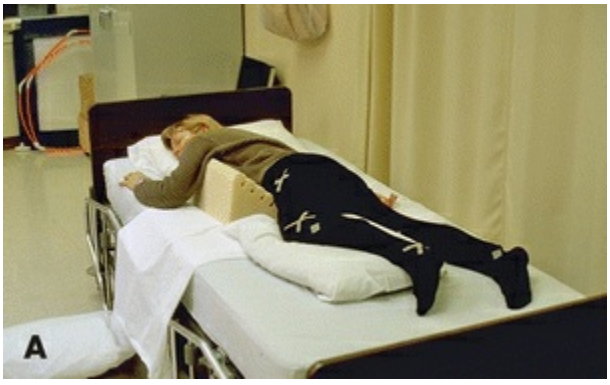
1.1. Encourage individuals who can reposition themselves to sleep in a 30 to 40 side-lying position or flat in bed if not contraindicated

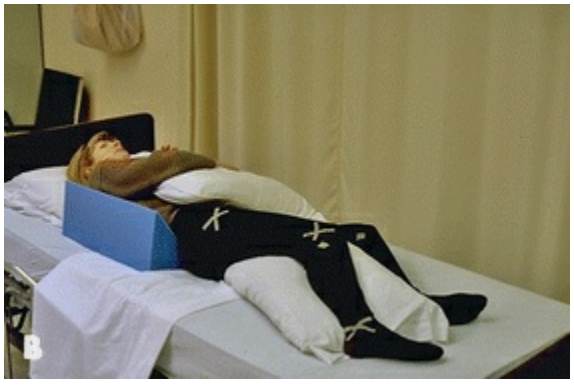
1.2. Avoid lying postures that increase pressure, such as the 90 side-lying position or the semirecumbent position

2. Limit head-of-bed elevation to 30 for an individual on bedrest unless contraindicated by medical condition or feeding and digestive considerations

2.1. If sitting in bed is necessary, avoid head of bed elevation of a slouched position that places pressure and shear on the sacrum and coccyx

See Figure 11-12 for some recommendations. Note the use of pillows and towels to separate and protect bony prominences. Additional positioning technique includes blocking the feet and knees in a flexed position to prevent shear forces created when the patient slides down in bed.





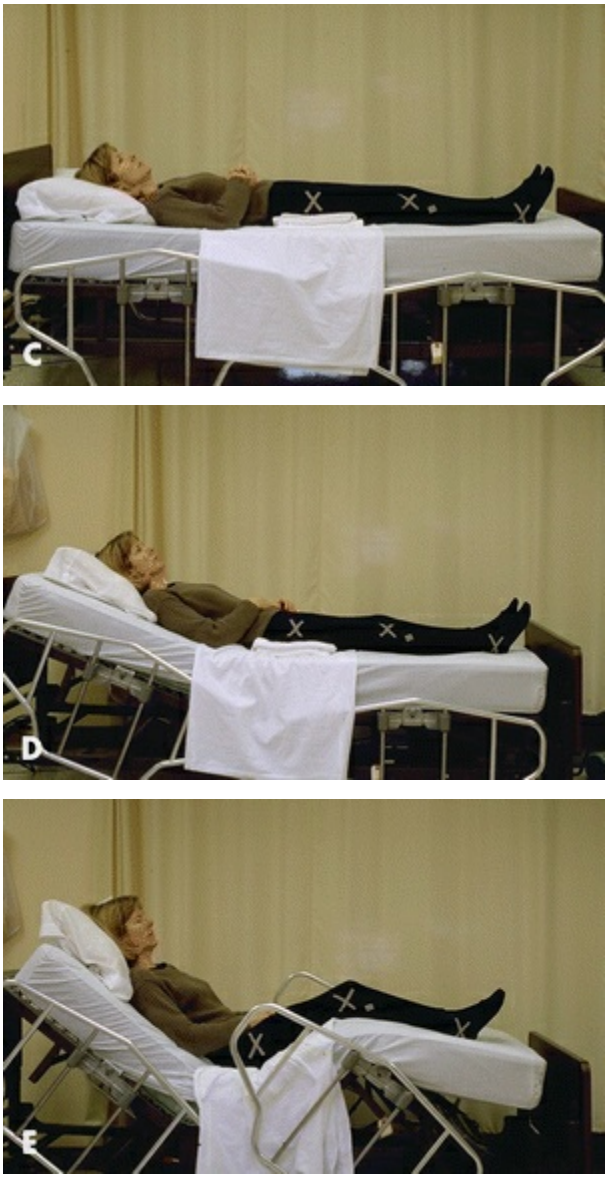


Figure 11-12. Horizontal positioning. The following illustrations show how to position patients properly on horizontal surfaces. **(A, B)** 30-degree rotation from prone and supine positions, respectively. **(C, D)** Head of the bed elevated 30 degrees or less with unilateral sacral relief and feet blocked, respectively. **(E)** Head of the bed elevated 30 degrees with knees flexed to prevent shearing at the sacrum.

Small shifts in weight can also be accomplished by positioning. For instance, foam wedges or pillows, used to position the patient on his or her side, can be altered slightly every 15 minutes. Pulling them out gradually over 1 to 1½ hours shifts the weight slightly. A patient who is in a wheelchair, he still requires shifts in his or her body weight. If possible, this patient should be taught to shift his or her weight every 15 minutes with repositioning every hour. (See Figure 11-11 and Box 11-1.)

Box 11-1 NPUAP/EPUAP/PPPIA Positioning Guidelines: The Following Are Some of the Recommendations in the 2014 Guidelines

General repositioning

Reposition all individuals at risk of, or with existing pressure ulcers, unless contraindicated. (Strength of evidence = A; Strength of Recommendation = 👍👍

Repositioning frequency: consider the pressure redistribution support surface in use when determining the frequency of repositioning. (Strength of evidence = A; Strength of Recommendation = 👍

Repositioning techniques: reposition the individual in such a way that pressure is relieved or redistributed. (Strength of evidence = C; Strength of Recommendation = 👍👍

Repositioning individuals in bed: Use the 30-degree tilted side-lying position (alternately, right side, back, left side) or prone position if the individual can tolerate this and her/his medical condition allows. (Strength of evidence = C; Strength of Recommendation = 👍

Prone position: Use a pressure redistribution surface to off-load pressure points on the face and body while in the prone position. (Strength of evidence = C; Strength of Recommendation = 👍 Additional recommendations are available in the guidelines.[65]

Adapted from Haesler, E., ed., National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel and Pan Pacific Pressure Injury Alliance. *Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline*. Osborne Park, Western Australia: Cambridge Media, 2014, with permission.

Heel Protection

The heel presents unique challenges for pressure-reducing interventions due to its small radius of curvature and its thin layer of subcutaneous tissue between the skin and calcaneal structures. This small contact area affords minimal protection from the pressure exerted by the weight of the foot and,

frequently, a portion of the lower limb. The lower limb is approximately one-sixth of the total body weight, so, even if a small proportion of this rests on the heel, high interface pressure may result, even on air support systems. Therefore, the 2014 NPUAP/EPUAP/PPPIA guideline[65] recommends using additional pressure-reducing devices for immobile patients' heels.

In addition, heel ulcers are especially painful and are among the most difficult to heal. Indeed, the sequelae of heel pressure ulcers can be devastating, possibly resulting in infection and, in extreme cases, amputation of the foot. Also, the costs associated with heel pressure ulcers increase proportionally with morbidity and length of hospital stay, underscoring the importance of prevention in high-risk patients.

NPUAP/EPUAP/PPPIA Clinical practice guideline[65] recommend heel suspension devices that elevate and off-load the heel in such a way as to keep the heels off the bed surface and avoid placing pressure on the Achilles tendon. Pillows should not be used as they don't protect against footdrop and, due to patient movement, pillows require time and diligent positioning to maintain proper suspension (Fig. 11-13).

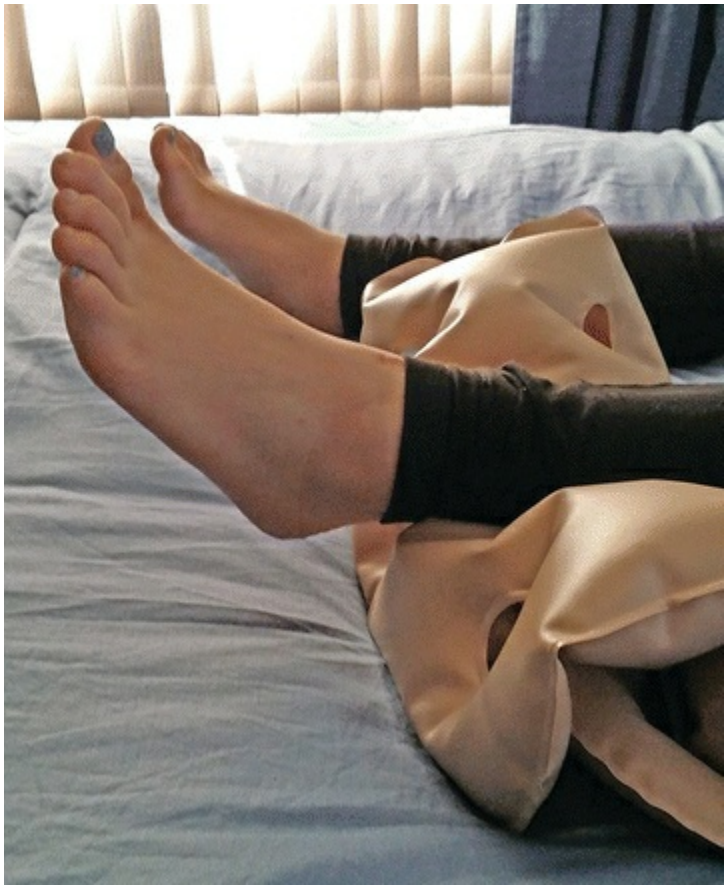


Figure 11-13. Floating heels off the bed. (Photo courtesy of S. Baranoski.)

Evidence for Managing Heel Pressure

Although relatively few studies of heel protection devices have been completed in the past decade, the majority of those studies that have been published have examined the pressure distribution capabilities of heel wraps, heel dressings, pillows, water-filled gloves, and various specialty heel products using interface pressure or pressure ulcer incidence as the primary outcomes.[95,96] We will examine these devices here.

As with other support surfaces, most heel protection products consist of a combination of materials and incorporate multiple strategies to optimize their therapeutic function. The distribution of pressure on a heel support surface depends on the relative fit between the heel and the surface, the mechanical properties of the heel tissue and the device, and the distribution of weight in the body part (heel). An ideal pressure distribution is one in which soft tissue shape isn't altered relative to its unloaded condition.[97]

In a study designed by Zernike to investigate the efficacy of clinically "familiar" heel devices, the preventive use of routine nursing care, hydrocolloid dressings, egg-crate foam, polyester-filled heel boots, and foam footdrop splints were compared in geriatric, orthopedic patients at risk for pressure ulcers.[97] In a second study, a combination of 4″ × 4″ gauze pads and an absorbent pad held in place with a gauze roll was compared with a laminated foam boot.[94,98] Although the statistical analyses of both studies were limited, the egg-crate boot and laminated foam boot were more effective in preventing pressure ulcers. Other methods in these studies either increased incidence or, as with the footdrop splints, proved too uncomfortable despite their ability to effectively suspend the heel. What's notable about Zernike's study[97] is that deterioration of the heel tissue ensued despite routine nursing care (heel observation every 2 to 3 hours and direct pressure relief and repositioning of patients' heels). In a similar study, however, the prompt reporting of heel discomfort by patients to the nursing staff, with subsequent simultaneous use of heel elevation with a pillow or bath blanket under the calf and a quilted heel protector, resulted in zero incidence of pressure ulcers in 30 hip replacement patients.[98]

Flemister[99] also examined heel interface pressures with use of both a foam and polyester heel protector in seven patients assessed at moderate-to-high risk for pressure ulcer development. The foam heel protector marginally reduced heel interface pressures (1.3 mm Hg), whereas the polyester protector actually resulted in a significant increase in interface pressure.

While there are a variety of heel protective devices available, their effectiveness seems to vary across the board. The few studies that have been performed suggest that while matching the device to the patient may be challenging, with careful consideration of patient needs and characteristics, it can be done.

What's Available in This Setting?

A patient's needs can change over the course of his or her illness. A product that's appropriate for acute care use may not work in the patient's home. Clinical judgment should consider therapy goals (ease of getting into or out of the bed for therapy or mobility), ability of the patient to move himself or herself in the bed, other complications (heel breakdown, pulmonary complications), and body weight and weight distribution.

What's Practical?

The issue of practicality relates to the overall care plan, the goals of load management (prevention vs. treatment), how complicated the product is to use, and whether it will be operated by a healthcare professional, family member, or the patient. Many hospitals, long-term care facilities, and home care agencies have developed product selection guidelines that are usually presented as an algorithm or graph. These guidelines are typically based on the availability of equipment from previous purchases in accordance with other published guidelines. Another common method used in selection and purchasing decisions is the subjective assessment of equipment based on a trial use in the clinical setting. This gives the staff a chance to use the equipment in a variety of situations and judge its performance.

How Easy Is the Product to Use?

Equipment that's easy to use has been associated with successful compliance. Awkward design or difficult assembly may cause the patient and his or her family to abandon using the equipment or increase the likelihood of misuse. Directions must be clear and obvious. For example, if the product is powered, it should have a battery backup to facilitate transferring patients to other locations as needed.

What Service and Maintenance Is Available?

A 24-hour call service with on-site repair or replacement is essential.

What Type of Alarm System Does the Product Have?

Visual alarms are rarely sufficient, especially if the alarm is obscured under the bed. In the home-care setting, visible alarms are of little value unless there's constant attendance.

Is the Equipment Easily Maintained?

If the equipment must be deflated for storage, reinflation time may be critical as well as the ability to deflate the equipment in the event of a cardiac arrest. Additionally, if the product isn't a personal use item, one must consider how long it takes to clean and ready the item for subsequent use.

What Operating Mechanism and Space Requirements Does the Product Have?

If the product has some form of movement, is cessation possible to facilitate such procedures as bedpan usage and hygiene care? Is the product the correct size for the home or setting where it will be used? Is the floor structure sound and capable of holding the weight of the bed? Will the bed fit through the door?

How Much Does the Product Cost?

Technology has produced some innovative treatment products, but costs are high for patients, insurance companies, and hospitals. Prevention is more cost-effective than is any other treatment. Available technology (support surfaces) may provide solutions when used correctly by properly educated staff. Indeed, one recent study demonstrated that educated staff can reduce the number of pressure ulcers better than noneducated staff. The same study indicated that the cost of care is significantly less under the supervision of educated staff[100]

The cost of support surfaces varies. Third-party reimbursement (Medicare may provide coverage), rental versus purchase, and cost–benefit issues need to be evaluated carefully. Some devices are only available on a rental basis. Although reimbursement is an issue in any setting, operational costs are of particular importance if the product is used in the home care setting where costs may be absorbed by the patient.

Consideration should also be given to the projected number of days that a support surface will be in use. More patients are being placed on advanced technology surfaces for considerably longer periods of time. Cost-effectiveness may be measured by relating the cost of the product to its efficacy.

Practice Point

Consider these patient factors before purchasing support surfaces:

- Ease of use
- Operational costs of the equipment
- Service contracts and backup service
- Alarm systems
- Daily maintenance
- Operating mechanisms and space requirements

When managing the allocation of specialty mattress and bed systems, a continuous process of evaluation and reevaluation should be employed to ensure that the patient's needs are reassessed on a regular basis.

Summary

Understanding the nature of pressure ulcer etiology, the factors affecting pressure redistribution, and other physical factors associated with the use of specific support surface products and heel protection products is a necessity for nursing and other healthcare personnel involved with tissue integrity management. As standardized test methods are developed and used for support surface products, the clinical validation of specialized protective devices and the development of more specific clinical practice guidelines will be possible. Until then, matching products to patient needs is a challenging process and must be based on the available evidence regarding the performance characteristics of existing support surfaces. Continued research in heel protection is needed.

Heels in patients at risk for pressure ulcers require additional protection beyond the use of specialty beds and mattress overlays. It is important to keep in mind that pillows don't protect against footdrop,

require time and attention to positioning details, and can't protect the heels continuously due to patient movement. To provide continuous heel suspension via heel protective devices, clinicians must consider proper fit, turning schedules and number of turning surfaces, patient position, the presence of additional equipment, and the performance characteristics of the product when selecting heel protection devices.

PATIENT SCENARIO

Clinical Data

Ms. CM is a 35-year-old woman with a T11-14 spinal cord injury. She lives alone, works full-time, and drives her own car. She presents in clinic with a right ischial pressure ulcer that will not heal and requests a new cushion. Inspection confirms an ischial ulcer that appears clean and well dressed.

Postural evaluation revealed a flexible left pelvic obliquity and minimal-to-moderate posterior pelvic tilt. Interface pressures measured on her current cushion were consistent with her posture, with higher pressure under the left ischial tuberosity and lesser pressure under the right ischial tuberosity, where the ulcer exists. Overall, the pressure profile was deemed acceptable. Subsequent discussion revealed that Ms. CM spends between 12 and 14 hours per day in her wheelchair and is able to achieve full pressure relief using a push-up technique. Ms. CM reported doing a push-up a couple times per day. A request to demonstrate a transfer showed good technique, adequate clearance of the buttocks over the drive wheels, and acceptable impact when landing. Ms. CM also reported that she sits on her Jay wheelchair cushion while driving.

A decision was made to try to increase pressure relief frequency and to change the technique from a push-up to a forward lean. By using a forward lean, Ms. CM was able to maintain pressure relief for a greater period of time. Her clinician asked her to limit her sitting to 9 hours per day with pressure relief every 30 minutes. This suggestion allowed Ms. CM to continue working while attempting to reduce her sitting time.

When Ms. CM returned for follow-up in 2 weeks, inspection showed little change in the appearance of the pressure ulcer.

In her description of a typical day, Ms. CM stated: " … and then I

usually go upstairs to bed." This statement prompted the clinician to ask if her bedroom was on the second floor and how she got up the stairs. Ms. CM responded: "I bump my way up and down the stairs." This was obviously a significant finding as this technique can repeatedly load the buttocks in a dangerous manner. Several interventions were discussed, including the use of a protective cushion during stair ascent and home modification to install a stair lift.

Case Discussion

This case highlights several important issues. A postural assessment done in concert with interface pressure mapping revealed that the side opposite of a pelvic obliquity was the site of an ulcer. This is typically not the case as the lower side usually receives higher pressures. Weight shift technique and frequency were addressed, but they did not have an impact. Nonetheless, it was an important intervention that may prove beneficial in the future. Finally, investigating all the places that Ms. CM "sits" proved to offer the key piece of information. Because highly functional wheelchair users may sit on a variety of surfaces, clinicians cannot presuppose that the cushion or wheelchair is always the causative factor in pressure ulcer formation.

Show What You Know

1. The feature of a support surface primarily indicated for pulmonary therapy that provides rotation about a longitudinal axis as characterized by degree of patient turn, duration, and frequency is called:

A. low-air-loss.
B. alternating pressure.
C. air-fluidized.
D. lateral rotation.

2. The selection of an appropriate support surface depends on:

A. the clinical condition of the patient.
B. the characteristics of the support surface.
C. the characteristics of the care setting.
D. all of the above.

3. Support surfaces are designed to:

A. lower pressure.
B. eliminate pressure.
C. redistribute pressure.
D. relieve pressure.

4. The ability of a support surface to distribute load over the contact areas of the human body is:

A. immersion.
B. envelopment.
C. pressure gradient.
D. pressure redistribution.

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Pain Management and Wounds

12

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Objectives

After completing this chapter, you'll be able to:

- define and identify the components of wound-associated pain
- describe the similarities and differences of pain associated with various types of chronic wounds
- utilize two validated patient tools for chronic wound-related pain
- assess the advantages and disadvantages of wound pain treatment modalities.

Etiology and Definitions of Pain

Pain has an element of blank;
It cannot recollect
When it began, or if there were
A day when it was not.

—EMILY DICKINSON

As clinicians, we have a tendency to identify various wound types as having *characteristic* pain of a specific type or amount. However, pain is what the patient states it is—not what we believe it to be. Our responsibility

as clinicians is to assess the patient's pain accurately and treat it adequately, without judging the patient or doubting that the pain is as described. Pain is often more important to patients than it is to clinicians, with surveys indicating that pain is the most important parameter for many patients but is often only the third or fourth priority for clinicians.

There are several definitions of pain in the literature. Both the 1979 International Association for the Study of Pain (IASP) Subcommittee on Taxonomy[1] and the Agency for Healthcare Research and Quality (AHRQ, formerly the Agency for Healthcare Policy and Research, or AHCPR)[2] support a common definition of pain. They have defined pain as *an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage*.[1,2]

Another commonly used pain definition is that of McCaffery and colleagues, [3,4] who state that "pain is whatever the experiencing person says it is and exists whenever he says it does."

The inability to communicate verbally or behaviorally does not negate the possibility the person is experiencing pain. This definition of pain encompasses the subjective component and acknowledges the patient as the best judge of his or her own pain experience. Experts in the field of pain have come to accept that the patient's self-reporting of pain, including its characteristics and intensity, encompasses the most reliable assessment. This belief that the patient in pain is his or her own best judge is also accepted as the basis for pain assessment and management by such regulatory agencies as the Joint Commission, formerly known as the Joint Commission on Accreditation of Healthcare Organizations[5] as well as such professional organizations as the American Pain Society (APS).[6]

Practice Point

Pain is what the person says it is and exists whenever he or she says it does. The true etiology of pain isn't known. More research is needed to learn the true cause of the individual patient's pain.

Types of Pain

Pain can be nociceptive or neuropathic or have components of both as

commonly experienced with wound-associated pain. Nociceptive pain can result from ongoing activation of primary afferent neurons by noxious stimuli, with a normally functioning nervous system. Neuropathic pain is initiated or caused by a primary lesion or dysfunction of the nervous system.[7]

The two types of nociceptive pain are somatic and visceral. Somatic pain arises from bone, skin, muscle, or connective tissue. It's usually gnawing, aching, tender, or throbbing and well localized. Pressure ulcer pain is usually somatic in nature. Visceral pain arises from the internal organs such as the gut or from an obstruction of a hollow viscous organ, as occurs with a blockage of the small bowel. Visceral pain is poorly localized and is commonly described as cramping. Both types of nociceptive pain respond well to nonopioid and opioid pain medication.

The origin of neuropathic pain is from an abnormal processing of the sensory input by the peripheral or central nervous system (CNS). The pain is typically described as having burning, stabbing, stinging, shooting, or electrical characteristics. Diabetic neurotrophic foot ulcer pain and the pain of shingles are examples of neuropathic pain. Neuropathic pain responds more readily to adjuvant agents, including antidepressants (e.g., tricyclic, serotonin–norepinephrine reuptake inhibitors, SSRI) or anticonvulsant therapy. Tricyclic antidepressants (TCA), such as amitriptyline, nortriptyline, or desipramine, are good choices because of their antinoradrenaline activity. Amitriptyline is a first-generation tricyclic agent with almost equal antinoradrenaline, antihistamine, antiserotonin, and antiadrenergic actions. Nortriptyline is a second-generation tricyclic that has higher antinoradrenaline activity at a lower dose, with fewer side effects such as double vision, dry mouth, and urinary retention. Desipramine has the same advantages as nortriptyline with less drowsiness. Duloxetine is an SSRI that has been approved for the treatment of painful peripheral diabetic neuropathy. The analgesic effect is likely related to augmentation of the inhibitory pain pathways in decreasing the perception of pain. If antidepressive agents are not tolerated or provide inadequate relief of neuropathic pain at reasonable dosages, then anticonvulsants, such as gabapentin and its derivative pregabalin, should be considered. Gabapentin has demonstrated a reduction in neuropathic pain.[8] Pregabalin has also proved to be useful in the treatment of neuropathic pain, with studies demonstrating a benefit in painful postherpetic neuropathy[9] and painful diabetic neuropathy.[10] Both gabapentin and pregabalin require dose adjustments in patients with renal disease.

Pain can also be acute (intermittent) or chronic (persistent). Acute pain has a distinct onset, with an obvious cause and short duration, and is usually associated with acute wounds, subsiding as healing takes place. Chronic pain can be from a chronic wound or other long-term disease, such as cancer. If it persists for 3 months or more, chronic pain is usually associated with functional and psychological impairment. Chronic pain can fluctuate in character and intensity.

The American Geriatric Society (AGS) [11] supports the terminology of "persistent" pain rather than "chronic" pain to circumvent the negative stereotypes that have been associated with the word "chronic." The AGS Clinical Practice Guideline, "The management of persistent pain in older persons," states: "Unfortunately, for many elderly persons, chronic pain has become a label associated with negative images and stereotypes commonly associated with long-standing psychiatric problems, futility in treatment, malingering, or drug-seeking behavior. Persistent pain may foster a more positive attitude by patients and professionals for the many effective treatments that are available to help alleviate suffering."[11]

Persistent and acute wound-associated pain can occur at the same time; similarly, nociceptive and neuropathic pain may occur simultaneously. Wound-associated pain is often a combination of nociceptive and neuropathic pain. It may be compounded by an inflammatory process that occurs from local tissue damage due to surgery, infection, trauma, or other inflammatory conditions. Inflammation and infection are characterized by redness, heat, and swelling that have been associated with an increased sensitivity to pain in and around the wound site.[12–14] Inflammation or infection-associated pain usually resolves when the condition that provoked the pain is controlled. Local tissue ischemia has also been implicated as a contributing factor to increased pain sensation in the acute phase of wounds.[12]

Regardless of the types and etiologies, pain can be debilitating and associated with sleep disturbance, poor appetite, functional decline, and/or psychosocial maladjustment. Patients suffering from pain reported decreased quality of life that extends beyond its physical component.

Practice Point

Reframing the phrase "the patient complains of pain" to "the

patient reports pain" may help to foster a more positive and objective way for practitioners and caregivers to connect with the patient's experience of pain. Use the term *persistent pain* rather than *chronic pain*.

The Persistent (Chronic) Pain Experience

Krasner[15–17] has conceptualized pain in chronic wounds as the chronic wound pain experience. Within this model, pain is divided into three categories: noncyclic, cyclic, and chronic pain. *Noncyclic* or *incident* pain is defined as a single episode of pain that might occur, for example, after wound debridement. *Cyclic* or *episodic* pain recurs as the result of repeated treatments, such as dressing changes, debridement, or turning and repositioning. *Chronic* or *continuous* pain is persistent and occurs without manipulation of the patient or the wound. For example, the patient may feel that the wound is throbbing even when he or she is lying still in bed and with no treatment occurring at the local wound site.

Practice PointInterventions for Noncyclic Wound Pain

- Identify and develop a pain treatment plan for potentially painful procedures.
- Administer topical or local anesthetics.
- Consider an operating room procedure under general anesthesia rather than bedside debridement for large, deep ulcers.
- Administer opioids and/or nonsteroidal anti-inflammatory drugs before and after procedures.
- Assess and reassess for pain before, during, and after procedures.
- Avoid using wet-to-dry dressings that can cause pain and trauma upon removal.
- Consider alternatives to surgical/sharp debridement, such as transparent dressings, hydrogels, hydrocolloids, hypertonic saline solutions, or enzymatic agents.[14]

Practice PointInterventions for Cyclic Wound Pain

- Perform interventions at a time of day when the patient is less fatigued.
- Provide analgesia 30 to 60 minutes before dressing change.
- Assess the patient for pain before, during, and after dressing changes.
- Provide analgesia 30 to 60 minutes before whirlpool.
- If the patient's dressing has dried out, thoroughly soak the dressing—especially the edges—prior to removal.
- Observe the wound for signs of local infection.
- Gently and thoroughly cleanse or irrigate the wound to remove debris and reduce the bacterial bioburden, which can cause contaminated wounds to become critically colonized or infected. Infection will increase the inflammation and pain at the wound site.
- Avoid using cytotoxic topical agents.
- Avoid aggressive packing. (Fluff; do not stuff!)
- Avoid drying out the wound bed and wound edges.
- Protect the periwound skin with sealants, ointments, or moisture barriers.
- Minimize the number of daily dressing changes.
- Select pain-reducing dressings that include moisture balance for healable wounds and avoid aggressive adhesives.
- Avoid using tape on fragile skin.
- Splint or immobilize the wounded area as needed.
- Utilize pressure-reducing devices in bed or when seated in a chair.
- Provide analgesia as needed to allow positioning of patient.
- Avoid trauma (shearing and tear injuries) to fragile skin when transferring, positioning, or holding a patient.

Practice PointInterventions for Persistent (Chronic) Wound Pain

- Use all of the interventions listed for noncyclic and cyclic wound pain.
- Control edema.
- Control infection.
- Monitor wound pain while the patient is at rest (at times when no dressing change is taking place).
- Control pain to facilitate healing and positioning.
- Provide regularly scheduled analgesia, including opioids, patient-controlled analgesia, and topical preparations such as lidocaine gel 2%, depending on the severity of pain.
- Attend to non–wound-associated pain from:
  - Comorbid pain syndromes such as contractures and neuropathic pain associated with diabetes/other neuropathies
  - Iatrogenic device insertions, such as central lines, venous puncture sites, catheters, feeding tubes, blood gas drawing, or other equipment or procedures
- Address the emotional component of the pain or the patient's suffering:
  - What does the wound represent to the patient?
  - What does pain mean? Is it associated with loss of function?
  - Has the wound altered the patient's body image?
  - Did unrelieved pain alter the patient's mental status or behavior?

Pain and Wound Types

The type of pain a patient experiences depends largely on the type of wound present. Pain can occur in patients with acute and chronic wounds and can be related or unrelated to the wound or its cause. Clinicians should determine whether pain is generalized, regionalized, or related directly to the wound bed. Regional pain often relates to the wound cause. Localized

wound pain may relate to local wound manipulation, treatment modalities, or infection.[18] Gardner and Frantz[18] identified increased wound-associated pain as a potential symptom of infection. This section discusses various types of wounds and the types of pain that accompany them.

Pressure Ulcer Pain

Pain at the site of a pressure ulcer is supported by pressure ulcer experts and anecdotal reports by clinicians, although few studies concerning pressure ulcer pain have been published. At its first conference in 1989, the National Pressure Ulcer Advisory Panel (NPUAP) stated that "pressure ulcers are serious wounds that cause considerable pain, suffering, disability, and even death."[19] van Rijswijk and Braden[20] reevaluated the AHCPR Treatment of Pressure Ulcer guidelines in light of studies published after the guidelines were released in 1994[21] and reaffirmed the panel's first recommendation about assessing pressure ulcer patients for pain. Based on additional evidence from studies supporting pain reduction with the use of moisture-retentive dressings, however, van Rijswijk and Braden[20] proposed that the 1994 AHCPR recommendations concerning pain and pressure ulcers be rewritten.

The etiology of pain in patients with pressure ulcers is often unknown. Pieper[22] quotes the work of Rook[23] and suggests that the common sources of pressure ulcer pain are from the "release of noxious chemicals from damaged tissue, erosion of tissue planes with destruction of nerve terminals, regeneration of nociceptive nerve terminals, infection, dressing changes, and debridement." In stage III or IV pressure ulcers, this pain may come from ischemic necrosis of the tissue triggered by a deep tissue injury or shear forces. Superficial stage II ulcers may be associated with skin surface pain from moisture or friction.

According to a study by Szors and Bourguignon,[24] pressure ulcer pain depends not only on the stage of the ulcer but also on whether a dressing change is taking place at the time the assessment is made. The majority of patients in their study (88%) reported pressure ulcer pain with dressing changes; a lower number of patients (84%) had persistent pain at rest. Patients rated the pain from sore to excruciating. Seventy-five percent rated their pain as mild, discomforting, or distressing; 18% rated their pain as horrible or excruciating. Clinicians need to ensure adequate pain control for patients with persistent pain with long-acting pain and breakthrough medication; they must also time the breakthrough medication so that it is

effective against the pain experienced at dressing change. In addition, appropriate cleansing and debridement methods and suitable dressings need to be chosen that will minimize wound surface pain and trauma at the time of removal and reapplication.

In 2009, Pieper and colleagues[25] published a NPUAP position paper based on a systematic review of the pressure ulcer pain literature of which 15 studies met their inclusion criteria. Four studies were on topical medication treatment and the others on various other pain treatments, assessment, and the pain experience. Their findings included choosing dressings that minimum pain. They were unable to find studies regarding nutrition and pressure ulcer pain but did discuss the potential of nausea and decreased appetite for patients with pressure ulcers experiencing pain. These authors were also able to identify gaps in the research literature for special groups such as neonates, children, bariatric, and end of life patients. In 2010, Langemo and Black published a NPUAP white paper that includes recommendations for assessing, preventing, and treating pressure ulcer pain in the population of 300 million individuals who receive palliative or end of life care.[26]

Arterial Ulcer Pain

Pain associated with peripheral vascular disease can be due to intermittent claudication or to rest pain with advanced disease that may be more prominent at night with leg elevation. Intermittent claudication pain results from physical exertion or exercise-induced ischemia and has been described as cramping, burning, or aching. The blood flow with exertion is inadequate to meet the needs of tissues. Patients can employ several tactics to relieve pain. The most important are to stop smoking, start gradual and regular exercises, lose weight, and have vascular risk factors treated.

Nocturnal pain may have the same symptoms but usually precedes the occurrence of rest pain. Rest pain occurs—even without activity—when blood flow is inadequate to meet the needs of tissues in the extremities. These types of pain are described as a sensation of burning or numbness aggravated by leg elevation where gravity no longer can facilitate local blood flow. The pain is constant and intense and isn't easily relieved by pain medications. Pain can sometimes be alleviated by stopping the activity or exercise and placing the legs in a dangling or dependent position (head of bed on blocks) to promote gravity-assisted blood flow. Some patients find relief with gentle massage, while other wear socks to help maintain warmth.

Venous Ulcer Pain

Venous ulcer pain can have several possible sources:

- Edema due to extravasated fluid from the capillaries
- Inflammation of woody fibrosis: acute or subacute lipodermatosclerosis
- Bacterial damage
    - Superficial increased bacterial burden (NERDS©)[27,28]
    - Deep and surrounding skin cellulitis (STONES©)[27,28]
- Inflammation of the veins: superficial and deep phlebitis

The range of venous ulcer pain is extensive; the patient may report mildly annoying pain, a dull ache, or sharp, deep muscle pain. Pain is more intense at the end of the day secondary to edema resulting from the legs being in a dependent position, and this dependent edema is often aggravated by standing, sitting, or crossing the legs. The pathophysiology of venous disease is related to reduction or occlusion of blood return to the heart. Incompetent superficial, perforating, or deep veins can cause pooling of fluid in the legs leading to pitting edema and resultant pain. To minimize pain, patients should be instructed to elevate the legs when sitting and encouraged to wear support stockings. Stocking selection is based on accurate individualized measurement, and their effectiveness relies on putting them on before the legs are placed on the floor in the morning. Other clinical management goals that help to minimize venous disease–related edema include the avoidance of prolonged sitting, weight reduction, and smoking cessation.

Thrombus formation in the deep veins can lead to leg swelling and pain, mimicking an infection or acute lipodermatosclerosis. The patient may report localized tenderness and pain over the long and short saphenous veins. Increased bacterial burden in the superficial wound bed can lead to delayed healing and localized pain. Clinicians should look for three or more NERDS[27,28] signs: ***n***onhealing wounds; increased ***e***xudate; ***r***ed, friable granulation tissue; new ***d***ebris or slough on the surface; and an unpleasant ***s***mell or odor. (For more information about NERDS and STONES, see Chapter 7, Wound Bioburden and Infection.) When venous disease has been present for a long period of time, the veins become leaky with fibrin extravasation into the dermis (woody fibrosis). In addition, red blood cells can leak into the tissue, causing staining that's often referred to as hemosiderin and hyperpigmentation. The woody fibrosis does not go away

at the end of the day, and patients can have acute and chronic inflammatory changes within the woody fibrosis, leading to acute and chronic lipodermatosclerosis-type pain.

Neuropathic Ulcer Pain

Neuropathy is the most common complication of diabetes. The amount of pain present depends on the severity of the neuropathy. Unlike stimulus-dependent nociceptive pain, neuropathic pain is spontaneous. The patient may state that the pain interferes with his or her entire life—especially the ability to sleep. The affected extremity may feel like it's asleep ("a block of wood") or have the "pins and needles" pain that occurs after a part of the body has "fallen asleep" and starts to wake up. The quality of pain can be burning, stinging, stabbing, or shooting and may include increased skin sensitivity to nonnoxious stimuli (allodynia) and itching. True pain relief is accomplished primarily with pharmacologic intervention. All pain needs to be assessed adequately to ascertain the most effective treatment modality. If a patient reports excessive pain in a neuropathic limb that hasn't had pain before, an infection or acute Charcot joint changes may be developing.

Patients with diabetes lose protective sensation after 10 to 15 years (sooner with poor blood glucose control). This loss of protective sensation allows these individuals to undergo sharp surgical debridement without nociceptive pain, although they may have referred pain in the leg or foot. If persistent nociceptive pain develops in a neuropathic limb, it usually means there's disruption of the deeper structures. In a person with a foot ulcer, clinicians should check for underlying osteomyelitis. If a patient has a tender, swollen foot without ulceration and an increase in skin surface temperature, there's a strong possibility of a Charcot foot. Occasionally, a patient may have both osteomyelitis and a Charcot foot.

Practice Point

Determining whether pain in a person with diabetes is the result of neuropathy or is associated with peripheral vascular disease or infection is extremely important because patients with diabetes have a high incidence of peripheral vascular disease. In addition, pain in a painless foot usually indicates disruption of the deeper structures and a strong possibility of associated osteomyelitis, Charcot foot, or even both conditions coexisting.

Understanding Wound Pain

Most of our understanding of wound pain comes from literature about other diseases.[29] Clinicians are increasingly acknowledging that pain is a major issue for patients suffering from many different types of wounds.[29] Several consensus statements and other documents regarding wound pain during dressing changes are available to help clinicians manage this type of pain properly.

Practice PointPain: What We Know, What We Don't Know

McCaffery and Robinson[4] reported on nurses' self-evaluation of their knowledge about pain.

- Observable changes in vital signs must be relied upon to verify a patient's report of severe pain: False (answered correctly by 88.4%).
- Pain intensity should be rated by the clinician, not the patient: False (answered correctly by 99.1%).
- A patient may sleep in spite of moderate or severe pain: True (answered correctly by 90.6%).
- Intramuscular (IM) meperidine is the drug of choice for prolonged pain: False (answered correctly by 85.6%).
- Analgesics for chronic pain are more effective when administered as needed rather than around the clock: False (answered correctly by 92.7%).
- If the patient can be distracted from the pain, the patient has less pain than he or she reports: False (answered correctly by 94.7%).
- The patient in pain should be encouraged to endure as much pain as possible before resorting to a pain relief measure: False (answered correctly by 98.4%).
- Respiratory depression (<7 breaths per minute) probably occurs in at least 10% of patients who receive one or more doses of an opioid for relief of pain: False (answered

correctly by 60.5%; clinicians tend to exaggerate the risk of respiratory depression with opioid use; according to McCaffery and Robinson, the risk is <1%).

- Vicodin (hydrocodone 5 mg and acetaminophen 500 mg) is approximately equal to the analgesia of one-half of a dose of meperidine 75 mg IM: False (correctly answered by 48.3%).
- If a patient's pain is relieved by a placebo, the pain isn't real: False (answered correctly by 86.1%).
- Beyond a certain dose, increasing the dosage of an opioid such as morphine won't increase pain relief: False (answered correctly by 57.2%).
- Research shows that promethazine reliably potentiates opioid analgesics: False (correctly answered by 35.1%).
- When opioids are used for pain relief under the following circumstances, what percentage of patients is likely to develop opioid addiction?
    - Patients who receive opioids for 1 to 3 days: Answer is less than 1% (correctly answered by 82.8%).
    - Patients who receive opioids for 3 to 6 months: Answer is less than 1% (correctly answered by 26.7%).

International Guidelines

The European Wound Management Association (EWMA)[30] has developed a position document on wound pain titled "Pain at wound dressing changes." The document is subdivided into three sections:

- Understanding wound pain and trauma from an international perspective[31]
- The theory of pain[32]
- Pain at wound dressing changes: A guide to management[33]

In the first section of the document, Moffat and colleagues[30] surveyed 3,918 healthcare professionals from the United States and 10 countries in Western and Eastern Europe. The survey respondents indicated that pain prevention was the second highest ranking consideration at dressing change, with trauma prevention being first.[30] Pain from leg ulcers was ranked as the most severe pain compared with other wound types, and dressing removal caused

the greatest pain.[23]

A copy of this EWMA position document[30] can be found on the Internet and is available in Dutch, English, French, German, Italian, and Spanish (Table 12-1).

Table 12-1 EWMA Suggestions for Preparing the Wound Pain Environment[30,34–36]

Prepare, Plan, Prevent

- Choose an appropriate nonstressful environment, close windows, turn off mobile phones, etc.
- Explain to the patient in simple terms what will be done and the method used.
- Assess the need for skilled or unskilled assistance, such as help with simple hand holding.
- Be thoughtful in positioning the patient to minimize discomfort and avoid unnecessary contact or exposure.
- Avoid prolonged exposure of the wound (e.g., waiting for specialist advice).
- Avoid any unnecessary stimulus to the wound and handle wounds gently, being aware that any slight touch can cause pain.
- Involve the patient throughout; frequent verbal checks and use of pain tools offer real-time feedback.
- Consider preventive analgesia.

Another international consensus statement was developed by the World Union of Wound Healing Societies (WUWHS).[31] This document, entitled "Principles of best practice: Minimizing pain at wound dressing related procedures," outlines common pain management challenges, myths, and misunderstandings that are useful for improved clinical practice during wound dressing changes (Table 12-2). This document also includes helpful suggestions for care planning and treatment interventions (Table 12-3).

Table 12-2 Dispelling Myths About Wound Pain

In its international consensus statement,[31] WUWHS outlines several myths related to wound pain.

- **Myth:** Wet-to-dry dressing is still the gold standard for wound care.
- **Fact:** Adherent gauze can disrupt delicate healing tissue and provoke severe pain.
- **Myth:** Transparent films are the best dressing for treating and reducing the pain of skin tears and other minor acute wounds.
- **Fact:** The misuse of transparent films is a common cause of skin tears.
- **Myth:** Using paper tape is the least painful way to secure a dressing.
- **Fact:** Heightened nerve sensation in a wide area around a wound can make adhesive tape painful to remove.
- **Myth:** Pulling a dressing off faster rather than slower reduces pain at dressing changes.
- **Fact:** This method has the potential to inflict tissue damage and traumatic pain.
- **Myth:** Using a skin sealant on periwound skin reduces the risk of pain and trauma.
- **Fact:** Skin sealants only create a thin topical layer and do not protect deeper dermal layers.
- **Myth:** People with diabetic foot wounds do not experience pain.
- **Fact:** There may be some loss of peripheral nerve sensation, but sensitivity in the area can be heightened.
- **Myth:** Pain comes from the wound. The surrounding tissue nerves play little role.
- **Fact:** Spinal cord responses to incoming pain signals can give rise to abnormal sensitivity in the surrounding area (allodynia).
- **Myth:** The only way to treat wound pain is by oral analgesic, 30 to 60 min before dressing changes.
- **Fact:** An oral analgesic can give some relief but should not be seen as a single solution. A full pain assessment must be used to evaluate and fine-tune any prescribed therapy.

Table 12-3 WUWHS Procedural Wound Pain Interventions

The following planning and treatment suggestions from WUWHS[31] can be helpful in caring for patients with wound pain:

- Be aware of the current status of pain
- Know pain triggers, and avoid them where possible
- Know and use pain reducers (when possible and not contraindicated)
- Avoid unnecessary manipulation of the wound
- Explore with the patient simple distraction techniques, such as counting up and down, focusing on the breath entering and leaving the lungs, or listening to music
- Reconsider management choices if pain becomes intolerable, and document as an adverse event
- Observe the wound and surrounding skin for evidence of infection, necrosis, or maceration
- Consider the temperature of the product before applying it to the wound
- Avoid excessive pressure from a dressing, bandage, or tape
- Follow the manufacturer's instructions when using a dressing or technology
- After the procedure, assess comfort of intervention and/or dressing/bandages applied.

Ongoing evaluation and modification of the management plan and treatment intervention is essential as wounds change over time. More advanced nonpharmacologic techniques that require specialist training or skilled personnel, such as the use of hypnosis or therapeutic touch, may be considered.

Lastly, a third international document (not a consensus statement) addresses the management of pain associated with burns. This document is titled "The management of pain associated with dressing changes in patients with burns" and can be found in the electronic wound care journal, *World Wide Wounds*.[32]

Pain Research: Pressure Ulcers

Nursing Management

Nursing management of patients with pressure ulcers sometimes does not adequately address the component of pain management. Hollingworth[29] determined that nurses' assessment, management, and documentation of pain after completing a wound dressing change was inadequate. Likewise, in a qualitative study that examined the reflections of 42 general and advanced practice nurses, Krasner[33] identified three distinct patterns nurses used to address pressure ulcer pain in their patients—nursing expertly, denying the pain, and confronting the challenge of pain:

1. Nursing expertly

   - Reading the pain
   - Attending to the pain
   - Acknowledging and empathizing with the patient

2. Denying the pain

   - Assuming that it doesn't exist
   - Not hearing the cries
   - Avoiding failure

3. Confronting the challenge of pain

   - Coping with frustration
   - Being with the patient[33]

Krasner[16,17,33] suggested that clinicians use this information to provide more patient-centered sensitive care for patients with pressure ulcer pain.

The Patient's Pain Experience

Few studies (four quantitative and five qualitative) have been published concerning the pain experience of patients with pressure ulcers. Variability in pain perceptions can be influenced by many contextual and psychological (patient-centered) factors. Woo[37] examined 96 patients with chronic wounds to determine whether anxiety or anticipatory pain played a role in the intensity of pain experienced at dressing changes. He uncovered a direct relationship between anxiety related to impending pain that subsequently intensified the experienced pain intensity. With heightened anxiety, environmental and somatic signals are brought to the patient's attention,

sharpening the degree of sensory receptivity and reducing pain tolerance. Woo documented that certain individuals who are insecure in their relationship with others were susceptible to experiencing anxiety and intensified pain.[37]

The first study to quantify pain by pressure ulcer stage was completed by Dallam and colleagues,[14] who documented the perceived intensity and patterns of pressure ulcer pain in hospitalized patients. The study population was diverse, with 66% being white (non-Hispanic) and the remainder being black (non-Hispanic), Hispanic, or Asian. Of the 132 patients enrolled in the study, 44 (33.3%) were respondents and 88 (66.7%) were nonrespondents because they couldn't communicate responses to the instruments (language and other cognitive barriers). Two different scales were used to measure pain intensity: the Visual Analog Scale (VAS) and the Faces Pain Rating Scale (FPRS). (See the next section for additional discussion of these pain scales.) The authors identified a high degree of agreement between the two pain scales. They also noted that the FPRS was easier to use for patients who were cognitively impaired or for whom English was their second language.

The major findings of this important study include the following[14]:

- The majority of patients with pressure ulcers had ulcer-related pain (68% of respondents reported some type of pain).
- Most patients didn't receive analgesics for pain relief; only 2% (n = 3) of patients in this population were given analgesics for pressure ulcer pain within 4 hours of the pain measurement.
- Patients who couldn't express pain or respond to pain scales may still have had pain.
- Patients with deeper pressure ulcer stages (stages III and IV) had more pain.

Some procedures, such as surgical debridement or wet-to-dry dressing changes, may increase pain. While the study didn't identify the interventions that might be most effective in controlling pain, patients whose beds had static air mattresses rather than regular hospital bed mattresses and those whose wounds were dressed with hydrocolloid dressings had significantly less pain.[14] The study also demonstrated that patients are able to differentiate between ulcer site pain, generalized pain, and other local pain sites such as IV and catheter sites.[14] Some cognitively impaired patients were able to indicate the presence of pain and respond to pain intensity

scales.

Both Dallam et al.[14] and Szors and Bourguignon[24] discovered that many patients suffer with untreated or undertreated pressure ulcer–associated pain. Dallam and colleagues[14] determined that only 2% of patients with pressure ulcer pain received analgesia. Four years later, Szors and Bourguignons[24] evaluation documented little improvement in the administration of pain-relieving medication: only 6% of patients with pressure ulcer pain had analgesics prescribed to address their pain.

Both studies reflect the need for clinicians to realize the potential for pain from pressure ulcers. Because only 44 of the 132 patients with pressure ulcer pain could respond to pain scales, Dallam and colleagues[14] recommend that pressure ulcer pain should be suspected even when the patient can't report pain. Both studies recommend further research to identify interventions that can relieve pressure ulcer pain and the associated suffering.

Franks and Collier[38] conducted a study in the United Kingdom in which they compared home care patients with (n = 75) and without (n = 100) pressure ulcers. Interestingly, they documented that patients with pressure ulcers had less pain than did those who did not have pressure ulcers. The authors speculated that perhaps pressure ulcer pain might not be the problem, as previously presumed, or that pain control was somehow more effective for patients receiving home care. An alternative explanation is that the home care patients with pressure ulcers did not have the same comorbid conditions as previously reported in hospital populations or that the comparator conditions evaluated in home care may have had greater pain (e.g., infected wounds).

In a quantitative pain study of 128 chronic wound patients, Ayello and colleagues[39] found that more than half of the patients with venous ulcers had pain (54%), almost one-third with diabetic neurotropic or neuroischemic ulcers had pain (30%), and one-quarter of those with pressure ulcers (25%) had pain.

Langemo and colleagues[40] published a qualitative phenomenological study about pain in pressure ulcer patients. They interviewed eight adults, half with active pressure ulcers at the time of the study and the other half with healed pressure ulcers. Seven themes were identified:

1. the perceived etiology of the pressure ulcer
2. life impact and changes
3. psychospiritual impact

4. extreme painfulness associated with the pressure ulcer
5. the need for knowledge and understanding
6. the need for and stress related to numerous treatments
7. the grieving process.

The fourth theme—extreme pain—was subdivided into three categories: intensity of pain, duration of pain, and analgesic use. Patients commonly referred to the intensity of pain from pressure ulcers with descriptors such as "it burned," "feeling like being stabbed," "sitting on a bunch of needles," or "stinging." Some examples of statements by actual study respondents include a woman with a stage II pressure ulcer who said, "I felt like somebody was getting a knife and really digging in there good and hard." In the words of another male respondent, "They (pressure ulcers) are very painful because no matter what way you put your bottom, it hurts."[40]

Respondents also commented on the duration of the pain, with statements such as "the majority of the time, even when I was lying down, it hurt." Pain continued to be a problem even after the pressure ulcer had healed. As one respondent stated, "Every now and again, it still hurts. But there is nothing there. This time there is nothing really there." The fear of addiction resulting from analgesic use was expressed by some respondents. One respondent with a stage IV pressure ulcer on the buttock commented, "I was constantly in pain and was taking morphine and other types of painkillers to try and ease the pain."

Another qualitative study reported about the pain of 10 pressure ulcer patients.[41] Although Rastinehad identified 22 themes, lack of communication and painful treatment interventions were the two most common complaints.[38] Some patients related accounts of communication failures that contributed to stress, tension, and anxiety.[41]

The European Pressure Ulcer Advisory Panel (EPUAP) funded a phenomenological study by Hopkins et al. [42] who identified endless pain as one of the three main themes in older people living with pressure ulcers. The eight patients in this study were all over age 65 and had stage III or IV pressure ulcers for more than 1 month. None had spinal cord injuries, as suggested by Langemo and colleagues[40] for future qualitative pressure ulcer pain studies. The four subthemes of endless pain were constant pain presence, keeping still, equipment pain, and treatment pain. For some patients, keeping still reduced their pain: "I don't dare move because everything then gets worse. I lie very still." For others, pain was exacerbated by pressure-relieving equipment as well as dressing changes.

All but one of the patients described their endless pain in a graphic way. "You put a bit of weight on your heel and (it) feels as though it's burst open."[40]

Chronic wound studies[43,44] and the studies cited in this chapter emphasize the importance of adequate pain assessment and treatment.

Pain Assessment

Despite the APS's identification of pain as "the fifth vital sign,"[6] it isn't always included in the assessment of a patient's pressure ulcer. Dallam and colleagues[14] urged that pain be added to the assessment of pressure ulcers and that a patient's pain status be assessed during dressing changes as well as when the patient is at rest. They also cautioned clinicians to remember that the absence of a response or an expression of pain doesn't mean that the patient doesn't have pain. Despite research about the pain experience,[14,40,41,43–45] assessment of pain in persons with pressure ulcers continues to be underreported.[39] Documentation of pain assessment may vary by chronic wound type, as patients with venous ulcers (63%) and diabetic foot ulcers (53%) in one study were more likely to have their pain assessment recorded compared with those with pressure ulcers (45%).[39]

Two assessment guides include pain as part of pressure ulcer assessment. The AHCPR[21] treatment guidelines include an example of a sample pressure ulcer pain assessment guide in which there is a place to check either yes or no regarding the presence of pain. Ayello's[46,47] ASSESSMENT mnemonic asks the clinician to quantify the patient's pain experience, including the presence of pain, when the pain occurs (e.g., is it episodic or constant), and if the patient is receiving measures for pain relief. The caregiver checks one of the following boxes under T = tenderness to touch or pain:

- no pain
- pain present on touch, anytime
- pain only when performing ulcer care.[46,47]

The mnemonic PQRST, which outlines the specific questions to ask the patient, is another useful tool for assessing a patient's pain[14] (Table 12-4).

Table 12-4 Essential Pain Assessment Elements

Use the PQRST mnemonic (shown below) to assess your patient's pain.
P = Palliative/provocative factors
• What makes the pain worse? • What makes it better?
Q = Quality of pain

• What kind of pain are you experiencing?
• Would you describe it as:
  • gnawing, aching, tender, throbbing (nociceptive)?
  • burning, stinging, shooting, stabbing (neuropathic)?
  • or any combination thereof?
  • Do you have other symptoms with the pain, such as fever, chills, nausea, or vomiting?

R = Region and radiation of pain
• Where is the pain? • Does the pain travel or remain in the same spot?
S = Severity of pain
• Would you describe your pain as none, mild, moderate, severe, or excruciating? • Rate your pain on a scale from 0 to 10, with 0 representing "no pain" and 10 being "the worst imaginable pain." • How would you rate the pain intensity at its worst, best, and now?
T = Temporal aspects of pain
• Is the pain better or worse at any particular time of the day or night? • When does it start or when does it stop? • Is it intermittent or constant, or does it occur only when you're moving?

A complete and thorough pain assessment enables the clinician to develop an effective pain treatment regimen and evaluate its effectiveness (Table 12-5). The American Society for Pain Management Nursing offers a position statement and clinical practice recommendations for pain assessment in specific patient populations—groups that clinicians may not always identify as needing pain assessment. These groups include patients with advanced dementia, infants and preverbal toddlers, and intubated and/or unconscious patients.[48] The Hartford Institute for Geriatric Nursing has produced a series on pain assessment called "Try This."[49] These one-page (front and back) documents provide a succinct summary that covers the important points on pain assessment in older adults and in patients with dementia.[49]

Table 12-5 Additional Pain Assessment Elements

Include the following additional elements in your initial assessment plan and treatment:

- Detailed history consisting of:
  - medication usage
  - treatment history
  - previous surgeries and injuries
  - impact on quality of life and activities of daily living.
- Physical examination, emphasizing the body system involved in the pain complaint (e.g., the musculoskeletal or neurologic system).
- Psychosocial assessment, including family history of depression or chronic pain.
- Appropriate diagnostic workup to determine the cause of pain and to rule out any contributing, treatable causes.

A thorough pain assessment enables the clinician to develop an effective pain treatment regimen and evaluate its effectiveness.

Practice Point

Pain is the fifth vital sign.

Pain Intensity Scales

Pain intensity scales use a simple verbal, visual, or numeric measure to help determine how much pain the patient is experiencing. The gold standard for assessing pain intensity is self-report and the utilization of standard pain intensity instruments.[50,51] Pain intensity scales are one-dimensional, quantitative measures designed to measure the sensory aspect of a patient's pain and to obtain a more objective approximation of pain by minimizing inaccuracies.[51]

The use of pain intensity scales to quantify pain levels and determine patients' responses to pain treatments has been mandated by The Joint Commission for use in all hospitals.[5] Two of the most widely accepted and utilized pain assessment scales are the Numeric Pain Intensity Scale and the Faces Pain Rating Scale (FPRS).[52] Another commonly used scale is the Visual Analog Scale (VAS), which consists of a 10-cm line that has no

numbers on it. At one end is the term "no pain," and at the other end is the phrase "pain as bad as it could possibly be."[2]

Numeric Pain Intensity Scale

The Numeric Pain Intensity Scale is considered the gold standard for pain assessment for adults and children over age 7.[2,52] This scale is a 10-cm line with the words "no pain" at one end, "worst possible pain" at the other end, and the numbers 0 to 10 running from one end of the scale to the other (Fig. 12-1). The patient is asked to select the number on the scale that represents the level of pain he or she is experiencing. Zero indicates no pain, 5 indicates moderate pain, and 10 indicates the worst possible pain.[2] Patients can often relate these anchors to 10 being slamming the car door on your thumb and 5 being a bee sting (personal communication Gary Sibbald). The Numeric Pain Intensity Scale is sometimes presented verbally[2,52]; however, visual presentation may help to standardize the process of pain assessment and assist hearing-impaired patients. In addition, the scale has been translated into many languages.[7]

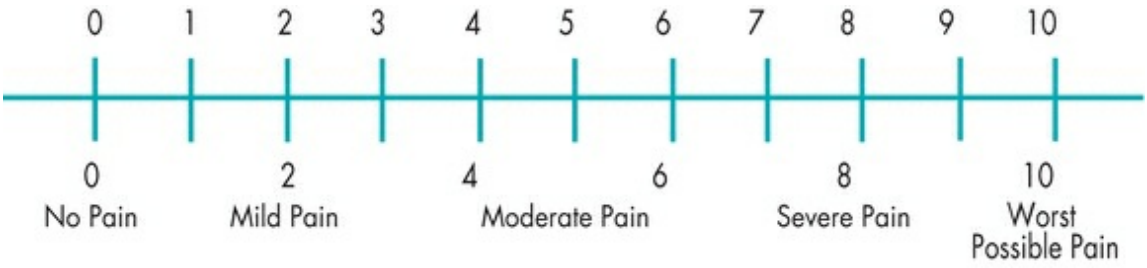

Figure 12-1. Numeric pain intensity scale. The Numeric Pain Intensity Scale is considered the gold standard for pain assessment and may be used for adults and children over age 7.

The Numeric Pain Intensity Scale aids in the adequate assessment and treatment of pain. It also helps clinicians choose the appropriate classification of pain medication recommendations based on any given patient response.[19,52,53] The scale can also help determine whether the patient has a response to the interventions if the numbers show a downward trend on repeated assessments.

Faces Pain Rating Scale

The FPRS[54] consists of six faces that range from a happy, smiling face (no

pain) to a crying, frowning face (worst pain). The first face on the scale, which is numbered 0, represents the absence of pain; the next face, numbered 1, represents very little pain; and so forth. The last face on the scale indicates extreme/worst pain. The patient is asked to choose the face that most closely reflects his or her own pain at that point in time (Fig. 12-2). The FPRS is preferred over other pain intensity scales for use with children.[52] The word "hurt," which appears on the FPRS, is the preferred word to use when assessing children, as children may not respond to the word "pain." In an older person, the word "ache" may be more useful in obtaining a response, as some older persons may be stoic and not admit to "pain." The validity and reliability of this scale in adult patients has not been established, although in a geriatric population a high degree of agreement was found between the FPRS and the VAS ($r = 92$; $p < 0.5$).[55] The FPRS has been used with cognitively impaired patients and with those for whom English is their second language. A high degree of consistency has been noted between the VAS and FPRS when utilized in the populations tested.

Figure 12-2. FACES pain rating scale. The FACES Pain Rating Scale may be used for children ages 3 and older, for cognitively impaired patients, and for persons less familiar with English. This scale is not always culturally or gender appropriate, as not everyone with a pain score of 10 will cry. (From Hockenberry-Eaton, M., Wilson, D. *Wong's Essentials of Pediatric Nursing*, 8th ed. St. Louis, MO: Mosby, 2009. © Mosby, Inc. Reprinted with permission.)

After the initial pain assessment has been completed, reassessment should be performed at regular intervals. Reassess the patient after administration of pain medication or nondrug pain-relieving interventions to ensure that optimal pain relief has been achieved.

Pain Management

Accurate and continuous pain assessment is the foundation of successful pain management.[7,50] However, evidence supports the fact that pain is often poorly assessed. Of physicians with patient care responsibilities in oncology, 76% rated poor pain assessment as the number one barrier to adequate pain management.[8] Donovan et al.[56] found that of the 58% of hospitalized patients reporting excruciating pain, fewer than half had a member of the healthcare team ask them about their pain or note the pain in their records. The use of pain assessment measures has been shown to improve pain management for patients.[55,57] However, problems using the pain assessment scales in everyday practice persist. One problem includes the lack of clinicians' knowledge and familiarity in the use of pain rating scales. Training is required for clinicians to administer pain scales and offer adequate patient instructions on the possible responses to the pain scale questions.

After pain has been identified, its cause should be determined and treated. "The goal of pain management in the pressure ulcer patient is to eliminate the cause of pain, to provide analgesia, or both."[20] Practical ways of treating pain, depending on the specific chronic wound etiology, have been described by McCaffery and Robinson[4] and Freedman and colleagues.[58]

Dressing changes, debridement, wound edema, infection, turning, and positioning are some of the factors that can cause wound-associated pain. An appropriate plan of action can be implemented after the specific cause of pain has been identified. For example, if the pain results from dressing changes, administering pain medication prior to dressing changes or switching to a different type of dressing may be indicated. According to Bergstrom and colleagues,[21] "Besides medications, pain may be treated with physical and occupational therapy to decrease muscle spasms, decrease contractures, and aide in selecting less painful methods of wound debridement and cleaning. Proper seating, positioning, and adaptive equipment may also help to decrease pain." The optimal way to treat the pain associated with pressure ulcers requires more research, but clinicians can assess the cause of the ulcer, patient-centered concerns, and all the components of local wound care to minimize pain at each step of their care plan.

Practice Point

Pain management should include interventions that:

- treat the cause
- address patient-centered concerns
- educate the patient
- improve activities of daily living (ADLs) and quality of life
- minimize pain and trauma by incorporating local wound care measures for cleansing, debridement, and moist interactive dressings
- assess and treat critical colonization/deep–surrounding infection
- provide palliation to the dying patient
- decrease or eliminate pain with minimal adverse effects
- minimize the patient's dependency on healthcare workers and family members

Pain Medication

The World Health Organization[59] (WHO) developed a three-step analgesic ladder for the treatment of cancer pain that has been accepted for use in patients with nonmalignant pain[8] (Fig. 12-3). The WHO approach advises clinicians to match the patient's reported pain intensity of 0 to 10 with the potency of the analgesic to be prescribed, starting with nonopioid analgesics for nociceptive pain and progressing to stronger medications if pain isn't relieved. For example, a patient who reports a pain score of 1 to 3 (mild pain) should receive a nonopioid with or without an adjuvant. If the patient reports a score of 4 to 6 (moderate pain), an appropriate low-dose single or combination opioid with or without an adjuvant should be administered. If the patient's pain score is 7 to 10 (severe pain), he or she should be given a strong opioid with or without an adjuvant. A low-dose single opioid is an opioid given at a low dose that is not combined with another drug within the same pill. An example is of a low-dose single opioid is oxycodone 5 mg. A combination opioid is an opioid, which is combined with another drug. An example of a combination opioid is oxycodone with acetaminophen or codeine with acetaminophen.

Combination opioids are frequently referred to as "weak opioids," whereas opioids such as morphine, hydromorphone, and fentanyl are referred to as "strong opioids." The clinician should bear in mind that "weak" and "strong" opioids may not apply to all cases, as what is considered weak for one person may be strong for another. For example, an 80-year-old frail person with a pain score of 10/10, who has never used an opioid, may or may not respond to a combination opioid of oxycodone 2.5 mg/acetaminophen 325 mg. Giving a patient an opioid is based not only on the level of pain but also on comorbid conditions, frailty, and previous analgesic history. Any opioid may be potent or cause harm if used indiscriminately or at levels that are inappropriate to the patient and his or her clinical circumstances. An AGS panel reminds us that recommended age-related doses are not available for most analgesics; therefore dosing for most patients requires initiation at low doses, followed by careful upward titration with frequent reassessment for dosing, pain relief, and side effects.[60]

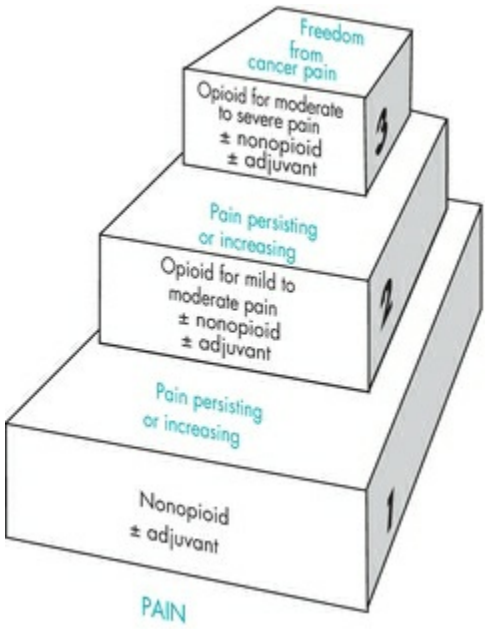

Figure 12-3. WHO analgesic ladder. This analgesic ladder, developed by the World Health Organization (WHO)[59] for pain management in patients with cancer, may be used as a guideline to manage mild through severe wound pain. (Copyright © World Health Organization, 2003. Reprinted with permission.)

An adjuvant medication is a drug that has a primary indication other than pain but is analgesic for some painful conditions.[7] Examples of adjuvant medications are anticonvulsants or TCA (Table 12-6). Adding an

adjuvant medication is most useful in addressing the burning, stinging, shooting, or stabbing symptoms of neuropathic pain. Using a combination of drugs such as an opioid and a nonopioid can enhance pain relief because the two drugs work synergistically. The opioid works on the CNS to alter the perception of pain, and the nonopioid works on the periphery to block painful impulses. Using a combination method may decrease the need for higher doses of opioids.

Table 12-6 Adjuvant Agents

Drug Class	Drug Name	Indications
Tricyclic antidepressants	Amitriptyline Desipramine Nortriptyline	• Multipurpose • Any chronic pain • Lower level of sedation • Neuropathic pain
Anticonvulsants	Carbamazepine Clonazepam Gabapentin Pregabalin Valproic acid	• Burning, neuropathic, lancinating pain
Systemic local anesthetics	Lidocaine Mexiletine	• Gnawing, Aching, Tender, Throbbing
Topical anesthetics	Capsaicin EMLA cream Lidocaine gel Lidocaine 1% Lidocaine 4% Lidocaine patch 5%	• Analgesic for intact skin (use on wound periphery prior to dressing changes) • Before changing vacuum-assisted closure dressing (instill solution through the tubing, with the pressure at 50 mm Hg, and clamp tubing for 15 to 20 min)[a] • Saturate gauze for 15 to 20 min prior to dressing change • Postherpetic neuralgia • Stump pain

[a]Systemic absorption and toxicity can occur in moderate-to-large wounds. Lidocaine products should not be used in patients who are taking class 1 antiarrhythmic drugs such as tocainide or mexiletine.

Step 1: Nonopioid Analgesics

Acetaminophen or nonsteroidal anti-inflammatory drugs (NSAIDs) should be initiated as first-line therapy. Acetaminophen is now available as an injectable for IV use. Its indication is for the management of mild-to-moderate pain and with an adjuvant opioid for severe pain. It is useful when parenteral analgesia is clinically warranted, or there is compromise of GI absorption, or an inability to take oral analgesic. It offers 100% bioavailability and can be used in children 2 years of age or older. Parental acetaminophen can be used preoperatively, intraoperatively, or postoperatively. As with PO acetaminophen, it should not be used in doses

higher than prescribed given its hepatotoxic profile. Acetaminophen and NSAIDs should be administered on a regular rather than an as-needed basis to increase their effectiveness and maintain a constant level of the medication in the blood. If one group of NSAIDs doesn't work, try another group (Table 12-7).

Table 12-7 Examples of Nonopioid Analgesics

- Acetaminophen
- Aspirin
- Tramadol[a]
- Nonsteroidal anti-inflammatory drugs
  - Celecoxib
  - Ibuprofen
  - Ketorolac
  - Salsalate

[a]Tramadol is not classified as an opioid, but it is centrally acting. It may cause any of the side effects experienced with opioids, including abuse potential and withdrawal. Dose adjustments must be made for those who have renal impairment and/or liver disease. It also lowers seizure thresholds.

NSAID groups include:

- salicylates: aspirin, diflunisal, choline magnesium trisalicylate, salsalate
- propionic acids: naproxen, ibuprofen, fenoprofen, ketoprofen, flurbiprofen, suprofen
- acetic acids: indomethacin, tolmetin, sulindac, diclofenac
- oxicams: piroxicam.

Clinicians must remember that NSAIDs have increased side effects in older people, including gastrointestinal bleeding, decreased renal function, and aggravation of congestive heart failure, and so should be used with caution in persons over age 65.[60] NSAIDs should also be avoided in patients with platelet disorders such as thrombocytopenia or who are on anticoagulation therapy. NSAIDs are generally not recommended for patients of any age, with kidney disease, heart failure, or cirrhosis or if the patient is using diuretics. There is an increased risk of bleeding in patients with a history of previous esophageal or gastrointestinal bleeding. All NSAIDs, including gels and patches, carry a black box warning. The warning is as follows:

NSAIDs may cause an increased risk of serious cardiovascular thrombotic events, myocardial infarction, and stroke, which can be fatal. This risk may increase with duration of use. Patients with cardiovascular disease or risk factors for cardiovascular disease may be at greater risk.

Low-dose, single-agent opioids such as oxycodone (5 mg) may be safer for patients with moderate pain who cannot take NSAIDs because of their side effect profile.

TCA, such as amitriptyline, imipramine, nortriptyline, and desipramine, have been shown to relieve neuropathy and postherpetic neuralgia but are contraindicated in patients with coronary disease (although they may be taken cautiously in coronary disease patients at low doses of 10 to 30 mg in a single night-time dosage).[8] According to an AGS panel, older patients may experience anticholinergic effects of TCAs that may include visual, urinary, gastrointestinal, and cardiovascular changes including orthostatic changes and atrioventricular blockade.[60] Hydroxyzine is a sedating H1 antihistamine that also has analgesic, antiemetic, and mild sedative properties. These drugs may help to induce sleep in patients with chronic pain and ordered along with other neuropathic agents as adjuvants for nociceptive pain.

Diabetic patients with neuropathic pain or other patients with conditions arising from peripheral nerve syndromes may benefit from the use of certain anticonvulsants, such as gabapentin, pregabalin,[9,10] phenytoin, carbamazepine, sodium valproate, or clonazepam.[8] Clonazepam, although pharmacologically classified as a benzodiazepine, not as an anticonvulsant, has anticonvulsant, muscle relaxant, and anxiolytic properties.

Opioids vary in strength from mild to very strong and are available in different forms, including oral, oral–transmucosal, rectal, transdermal, subcutaneous, IV, and IM.[61,62] The oral form is preferred for long-term use. However, for preprocedural use or for some patients with postprocedural pain from debridement, the IV route may allow for better pain control and an ability to increase the dosage more quickly as needed. Whether an oral or IV route is used, doses should be scheduled on a regular basis to avoid breakthrough pain. If breakthrough pain occurs when the patient is on a long-acting (sustained-release) opioid regimen, a short-acting (immediate-release) opioid may be given in conjunction with the long-acting opioid to provide pain relief. Breakthrough pain can occur spontaneously but is most likely to occur when the patient is moved, if a dressing change is required, if tubes are being manipulated, or if the patient has an increase in activity. When using opioids around the clock, the clinician should assess the patient

for pain caused by an "analgesic gap" or "end of dose failure." An example of this type of pain is when a patient is on a rescue dose for breakthrough pain every 6 hours as needed, but the medication only provides relief for 4 hours because of its pharmacologic properties. Shortening the interval at which the rescue dose can be used to every 4 hours as needed may correct this problem. Note also that approximately 15% of patients who are on long-acting opioid preparations require dosing every 8 hours rather than every 12 hours (Table 12-8).

Table 12-8 Examples of Combination Opioids

- Acetaminophen with codeine
- Hydrocodone with acetaminophen
- Hydrocodone with ibuprofen
- Oxycodone with acetaminophen
- Oxycodone with ibuprofen

Constipation can be one of the most common side effects with the use of opioid analgesics. This side effect can be easily remedied by taking stool softeners and laxatives and increasing fiber and fluid intake (especially water). It's better to anticipate constipation and treat it before it happens.

Other common side effects of opioids include sedation, nausea, vomiting, itching, urinary retention, and sensory or motor deficits. Many clinicians undertreat pain because they fear respiratory depression. While respiratory depression may occur with the use of opioids, it is uncommon in patients who are appropriately dosed and monitored. Patients at risk for developing respiratory depression include those with advanced disease states and/or comorbid conditions such as frailty, chronic obstructive pulmonary disease, congestive heart failure, sleep apnea, and kidney and/or liver disease. Respiratory depression can be decreased or eliminated with the use of lower initial doses that are increased gradually while monitoring the patient's vital signs, especially the quality and rate of respiration and the patient's sedation status.

Evidence-Based Practice

Avoiding Addiction: The Four A's of Opioid Treatment

Caregivers tend to underutilize opioid analgesics because of

the fear of **addiction.** Caregiver education in this area is very important, especially in the home care setting where the caregiver is the one deciding when to give the patient pain medication. Addiction to opioids should be a concern, but the fear of addiction has been greatly exaggerated. Studies have shown that the incidence of addiction in patients who take an opioid for acute pain relief is about 1%. The length of time on the analgesic and the amount given are irrelevant to the risk for addiction.[23]

While the incidence of addiction is low, the potential for **abuse** does exist. Therefore, clinicians should remain diligent in monitoring patients who are taking opioids to assess their risk for abuse. Patients with a history of nonopioid substance abuse, including cigarettes and alcohol, and mental health disorders are at higher risk of developing opioid abuse.[62] In addition, there are reports of rising prescription drug abuse with greater use of opioids for the treatment of chronic pain.[63–65] These type of reports have prompted the FDA to develop a multiagency federal effort to address Risk Evaluation and Mitigation Strategies (REMS) for extended-release and long-acting opioids.[66] These strategies include proposed class wide labeling changes that will help to insure their safe and appropriate use.[67] In keeping with the paradigm of protecting patients from potential harm associated with chronic opioid use, the AGS panel and others recommend that patients be assessed for risk factors related to problematic use of pain medications by using initial assessment tools such as the ORT (Opioid Risk Assessment Tool) or the SOAPP-R (Screener and Opioid Assessment for Patients with Pain-Revised).[60,63] An indication that abuse is developing is if the patient begins to crave the drug for reasons other than pain relief. When patients are using opioids, it is important to for the clinician to assess and document the 4 A's of opioid treatment as follows:

1. **Analgesia** (Is the pain score going down?)
2. **Activities of daily living** (Is there improvement in functional ability, positioning?)
3. **Adverse effects** (Are there mental status changes, such as confusion, or other effects, such as sedation, jerky motions, etc.?)
4. **Aberrant behaviors** (Does the patient report lost

prescriptions, lost pills?)

Meperidine is not recommended for the management of persistent (chronic) pain. Disadvantages include the hazards of the normeperidine metabolite of meperidine. Repeated doses of meperidine can lead to an accumulation of normeperidine, which causes CNS excitability and toxicity. This toxicity will be manifested in the patient by twitching, numbness, seizures, and hallucinations.[2] Coma and death are also possible. According to the APS,[8] "although the oral doses of meperidine have about one quarter of the analgesic effectiveness of similar parenteral doses, they produce just as much of this toxic metabolite." The APS also warns that patients with compromised renal function are particularly at risk for the accumulation of this toxic metabolite.

Step 2: Opioids For Mild-to-Moderate Pain

Opioids combined with an NSAID, or acetaminophen, or low-dose single-agent opioids, such as oxycodone immediate release 5 mg, may be used if step 1 is ineffective (Table 12-8). Propoxyphene has been withdrawn because of its cardiac toxicity profile. The FDA has concluded that the safety risk of propoxyphene outweighs its benefits for pain relief at the recommended doses and advised healthcare professionals to stop prescribing and dispensing it. The generic Darvon is no longer available in the United States. Codeine may cause excessive constipation, nausea, and vomiting.[50]

Step 2: Opioids for Moderate to Severe Pain

Low-dose opioids, including morphine and morphine-like agents, may be added to step 1 when it is ineffective (Table 12-9). An adjuvant drug such as the tricyclic agents (nortriptyline, desipramine) or antiepileptics (gabapentin, pregabalin) may also be used, if there is concurrent neuropathic pain, as well as somatic or visceral pain. The prescriber as well as the nurse caring for the patient should be aware of potential side effects and possible drug interactions. This includes knowledge of age and disease-related potential hazards and risks of all drugs used.

Table 12-9 Examples of Opioids: Morphine and Morphine-Like Agents

- Morphine
- Methadone
- Fentanyl
- Hydromorphone
- Levorphanol
- Oxycodone

Practice PointHelpful Hints for Using Opioids

- Fentanyl
  - Oral–transmucosal fentanyl works in 10 minutes, which is convenient for dressing changes, but its use for this purpose is considered off-label. This drug is FDA approved only for cancer-related pain. Oral–transmucosal fentanyl should only be used in patients who are opioid tolerant (patients whose total dose of opioids is equal to 60 mg of morphine a day, for a total of 1 week or longer).
  - Fentanyl patches should be started at the lowest dose for patients who are opioid naive (have never used opioids). Opioid-naive patients need careful monitoring. In frail patients, even the lowest dose may cause sedation or other unwanted side effects.
  - It will take 18 to 24 hours for the first patch to reach maximum effect. If immediate pain relief is required, a short-acting opioid can be used along with the patch and then discontinued, if needed, when the patch begins to reach its maximum effect. A rescue dose of a short-acting opioid may still be needed for breakthrough pain once the long-acting dose has been titrated.
- Use short-acting agents (at 10% to 15% of total daily dose) for breakthrough pain.
- If a patient is on both a long-acting agent and a short-acting (immediate release) agent for breakthrough and both agents are opioids for severe pain, the long-acting dose may needed to be titrated up, if the patient remains in pain over time, is

without side effects and is requiring more than three breakthrough doses daily.
- Don't wait for constipation to begin. Start the patient on stool softeners and laxatives to avoid this common adverse effect.
- To taper or discontinue opioid analgesics, decrease the dose by 25% every 2 to 3 days. Monitor for pain or withdrawal symptoms, as these would indicate that the tapering is too rapid.

Morphine is one of the drugs of choice for chronic pain largely because it is more cost-effective than meperidine, may be administered via many different routes, and is easily titrated. It has predictable and treatable side effects. The side effects of nausea and constipation are usually treated with antiemetic drugs and laxatives, respectively.

More recently, a pegylated derivative of the mu-opioid receptor agonist naloxone (naloxegol) has been used to treat opioid-induced constipation with promising results. Other side effects are usually dose related and can be resolved with dose adjustments.[7] Some patients cannot tolerate the side effects of morphine even with dose adjustments, and some experience significant side effects such as a skin rash or changes in mental status; the drug should be discontinued in these patients, and a pain management consultation should be sought to determine an appropriate alternative analgesic.

Morphine is not for use in patients with renal disease (glomerular filtration rate <30 mL/min) owing to the rapid accumulation of nondialyzable metabolite that can be neurotoxic.[68] Patients who have liver disease may also be sensitive to the effects of morphine because it takes longer for the damaged liver to metabolize and eliminate the drug. Patients with asthma may have increased wheezing with morphine use and should be monitored carefully. Any patient whose daily dose of narcotic agents exceeds the equivalent of 120 mg of morphine should have a specialized pain consultation.[61]

Although studies are limited, some researchers are exploring the effects of topical opioids in the treatment of painful skin ulcers. In their report on nine patients with open skin ulcers caused by a variety of medical conditions, Twillman and colleagues[69] documented that pain at the ulcer site was decreased when a morphine-infused gel dressing was used. The researchers reported remarkable efficacy in eight of the nine patients

studied and believe further research is needed in this area because so many patients stand to gain pain relief.

Nonpharmacologic Treatment Modalities

Management of pain from wounds can require a combination of pharmacologic and nonpharmacologic treatments; the latter may include the use of music, massage, and relaxation techniques. Pain associated with dressing changes and debridement can be minimized by allowing patients to call "time-outs." Many other nonpharmacologic treatments can also be used prior to, and in conjunction with, medications. These include physical and occupational therapy, repositioning the patient, providing support surfaces, and optimizing local wound care with materials that minimize pain.

Practice Point

Symptoms of withdrawal or abstinence from opioids include:

- tachycardia
- hypertension
- insomnia
- diaphoresis
- piloerection
- enlarged pupils
- nausea and diarrhea
- abdominal pain
- body aches
- increased sensitivity to pain
- yawning
- runny nose
- anxiety

The presence of these symptoms may indicate that the opioid medication is being tapered too quickly, and not necessarily that the patient is addicted to it.

Physical and Occupational Therapy

Physical and occupational therapy services may be a valuable asset to utilize in conjunction with pharmacologic therapy. Passive and active range-of-motion exercises should be taught to the patient and his or her caregivers. Additional measures include the following:

- Patients with peripheral vascular disease may benefit from a walking program to facilitate development of collateral circulation in the lower extremities.
- Application of a transcutaneous electrical nerve stimulation unit may help to decrease pain, particularly in patients with chronic or acute wounds. It's believed that the electrical stimulation provided by the unit helps to inhibit pain transmission cells.
- Hot or cold packs can be applied to decrease spasms in the affected area.
- Stretching exercises help to decrease contractures.
- Exercise helps to decrease muscle spasms with massage.

Local Pain Management

The use of appropriate dressings and dressing techniques can help to relieve pain during and between dressing changes. The international survey conducted by Moffatt et al.[34] highlights the importance of low-pain dressing changes. All respondents agreed that gauze dressings cause the most pain, while pain is noticeably less severe with the use of hydrogels, hydrofibers, alginates, and soft silicone dressings.

When administering wound care, choose products carefully to provide the patient with a pain-free experience. (See Chapter 9, Wound Treatment Options.) Patients who express discomfort despite careful product selection should be given medication prior to dressing changes.

Moist wound dressings can be left in place for a longer period of time than wet-to-dry dressings. This reduces the frequency of dressing changes, thereby decreasing the opportunities for the patient to experience pain associated with dressing changes.

Practice Point

Wet-to-dry dressings can desiccate a wound, thus causing pain on removal. (Removing viable stuck tissue from the wound surface

leads to bleeding, trauma, pain, and delayed wound healing.) Avoid these dressings in favor of moisture-retentive dressings to promote a healing environment and patient comfort.

The following interventions will ease the dressing change process and manage pain for your patients.

Treating the Cause/Aggravating Factors

- Provide pressure redistribution for your patients.
- Keep the patient's heels off the bed at all times.
- Control edema to avoid decreased blood flow to the wound, which may lead to additional pain.
- Eliminate or decrease pain from other possible pain sources.

Addressing Patient-Centered Concerns

- Keep in mind that pharmacologic management is the gold standard for moderate-to-severe pain. Give pain medication around the clock, if necessary, to keep the pain under control.
- Instruct the patient and his or her family regarding pain management to alleviate fear of addiction with the use of opioids.
- Explain the role that pain control plays in improved wound healing.
- Outline the dressing change procedures to the patient before proceeding with dressing changes.
- Allow the patient to select the time for dressing changes, if appropriate.
- Assess for pain and medicate the patient before and after dressing changes or debridement.
- Invite the patient or family members to participate in dressing changes, as indicated.
- Offer the patient distraction techniques, such as conversation, television, and videos during dressing changes.
- Inform the patient that he or she may call a "time-out" if pain is present during dressing changes.
- Ensure that the patient has adequate rest and sleep. Lack of rest and sleep will decrease the patient's pain threshold, decrease his or her mental performance, and increase the emotional response to pain.
- Teach the patient to substitute worry beads, a pet rock, bean bag,

tapping, rubbing, or gentle slapping for scratching.

- Instruct the patient in relaxation techniques and the use of visual imagery when encountering a potentially pain-provoking situation.
- Reevaluate the pain management plan when needed. Document the effectiveness of the analgesic or other treatments for pain relief with a pain score. This will help to assess whether the pain management program is working.
- Address other factors, such as loss of function, inability to perform ADLs, and possible changes in body image to help the patient deal with ancillary problems that might contribute to his or her pain.

Cleansing and Debridement

- Assess pain when the patient is at rest to provide adequate pain control.
- Apply warm normal saline solution or low cytotoxicity wound cleaners to clean wounds. (Cytotoxic solutions such as (Betadine) iodine solutions or hydrogen peroxide not only deter wound healing, but they may cause burning, adding to the patient's discomfort.)
- Use moist wound therapy to enhance autolytic debridement as an alternative to surgical or sharp debridement to eliminate pain associated with sharp debridement.
- Change dressings in a timely manner. Excess exudate on periwound skin or dressings that are allowed to dry on a wound may increase the patient's pain.
- Protect the periwound skin with skin barrier wipes, film-forming liquid acrylates, or ointment (petrolatum or zinc oxide) to prevent excoriation, trauma, maceration, or dermatitis that can delay wound healing, increase wound size, and increase patient discomfort. Avoid using strong adhesive tape on elderly patients or patients with fragile skin.

Alternative Pain Management Methods

Many natural pain control methods and therapies may be implemented to ease pain, stress, and anxiety. These methods can improve one's outlook, attitude, and quality of life. Alternative therapies, when used in conjunction with pain medications, may enhance the beneficial effects of pain medication.

Laughter. Laughter helps you breathe deeper, lowers your blood

pressure, and changes your mood.

Acupuncture. The application of needles to specific areas of the body may decrease or eliminate pain and has been used for more than 2,500 years.

Environment. Having the room at a comfortable temperature, avoiding bright lights, and keeping the room quiet may help to decrease pain.

Distraction. Playing cards, watching television, visiting with friends, petting an animal, and writing about his or her feelings can help the patient focus attention on something other than the pain.

Music. Music increases blood flow to the brain and increases energy, which in turn causes an increase in the production of endorphins (a natural body chemical similar to morphine) that work to decrease or eliminate pain and anxiety.

Magnets. Magnets may effect changes in cells or body chemistry that can produce pain relief. The use of magnets, which dates back to ancient Egypt and Greece, is popular with athletes, who report their effectiveness in controlling pain.

Capsaicin. Capsaicin, a chemical found in chili peppers, is the primary ingredient in many pain-relieving creams for the treatment of neuropathic pain. The local burning sensation it produces replaces the pain sensation.

Summary

Pain scores derived from patient-completed validated pain assessment scales can be useful as the basis for assessing and treating chronic wound-associated pain. The scales enable the clinician to accurately assess the patient's pain, thereby facilitating effective treatment modalities to help decrease the wound-associated pain. Pain is detrimental for patients because it can exhaust them, reduce their ability to perform ADLs, add to feelings of decreased worth as a person, affect their interactions with loved ones and friends, deter wound healing and, overall, diminish quality of life and as well as quality of death for patients who are in the process of dying. As clinicians, we are obligated to provide adequate pain relief for our patients by selecting appropriate pain control treatment modalities.

PATIENT SCENARIO

Clinical Data

Mr. KL is a 62-year-old male who is hospitalized for infected leg ulcer. He reports pain associated with this ulcer. His self-report pain level is 10/10. The pain extends beyond the wound edges and is present during rest and increases with dressing changes. He describes the pain as deeply aching down to the muscle with unbearable burning as if he was burning alive. He states he is "saddened and terrified by the burning feeling" and further describes it as the way that his "buddies must have felt when they were wounded or killed during the war." He further adds "I didn't burn then, but I am burning now."

The leg ulcer has been present for 9 months, progressively worsening. The leg wound is a shallow 4 × 6 cm on the lateral aspect of the lower leg. The wound bed is composed of red, friable granulation tissue and yellowish, malodorous slough on the surface. The wound is associated with edema, which worsens at the end of the day; inflammatory woody fibrosis; and surrounding cellulitis. The patient reports increased exudate and odor. The pain affects his walking, sleeping, and performing ADLs.

Mr. KL lives alone. He is a Vietnam War Veteran. He is a ½ pack day smoker for 40 years and continues to smoke "3 to 4 cigarettes a day." He admits to a remote history of marijuana use and denies illicit and or prescription drug use/abuse. He reports he is fearful of opioids because of their additive properties. He drinks 1 to 2 beers on the weekends, but abstains from hard liquor. He has had no psychiatric comorbid conditions, but states he sometimes suffers "pain of his soul." He does not use sleeping medications, benzodiazepines, or any other sedating medications or psych meds. Mr. KL's other comorbid conditions include hypertension, hyperlipidemia, osteoarthritis, stage III chronic kidney disease, and a history of DVTs. His analgesic history is as follows:

- Acetaminophen with 30% relief
- Codeine with acetaminophen with 30% relief
- Tramadol with adverse effects of confusion and had to stop medications
- Short-acting morphine 15 mg q 8 hours PRN with 60% relief, which lasts only 4 hours past dosing

Mr. KL has a nonhealing painful ulcer that is infected and associated

with surrounding cellulitis, consistent with a venous ulcer.

Case Discussion

Initial Pain Assessment and Treatment Rationale

Mr. KL should avoid the use of codeine and morphine as its metabolite can accumulate in patients with renal disease and cause neurotoxicity and respiratory depression. Likewise, NSAIDs should be used with caution because they can aggravate renal disease.

The recommended treatment for this patient is to use a combination of pain relief modalities, such as drug and nondrug related modalities. Because this patient is fearful of opioid use, it is important to educate the patient about risks, benefits, and alternatives to opioid use. Every effort should be made in using multimodal strategies and minimize the use of opioids. If an opioid must be used, it should be used at the lowest dose possible, which is consistent with pain relief and maximizing function.

Nondrug Strategies

The wound is treated locally for reducing the bioburden and controlling the exudate with cleansing of debris and autolytic debridement. The surrounding cellulitis is treated with systemic antibiotics. Reducing infection and exudate is helpful in pain reduction. The patient is instructed to avoid long periods of sitting to avoid pooling of fluids in the leg, when sitting the patient should elevate his leg, as edema increases the pain locally. Smoking cessation was also discussed with the patient. Wet to dry dressing, strong adhesive tapes are avoided, because these can increase pain at dressing change. A Doppler study was performed, given the report of muscle pain and history of DVTs and found to be negative.

As there was some concern related to possible psychosocial issues and unresolved emotional components of pain (pain of the soul) and possibly survival guilt (I didn't burn then, but am burning now) and tearfulness and reports of sadness, the patient agreed to a psych evaluation.

Nonopioid Strategies

To ameliorate the burning sensation in and around the ulcer, treatment should include a tricyclic antidepressant, starting at the lowest dose possible, or gabapentin starting at 100 mg at bedtime and slowly titrated to 300 mg daily (renal dosed based on creatinine clearance).

The patient asked if he could take acetaminophen for his arthritis pain instead of using the oxycodone and was instructed that he should avoid drinking alcohol if he wanted to use acetaminophen and should not exceed 1,000 mg q 8 hours a day (3,000 mg/d) because of the hepatotoxic effects. This includes taking into account all other products such as allergy or cold medications that contain acetaminophen that he may also be using. The patient is reminded that the FDA acetaminophen manufacturers have warned that having 3 drinks a day while using acetaminophen can cause severe liver damage.

Opioid Strategies

For his chronic pain, a low-dose fentanyl patch 12 mcg was ordered because the patient reported a pain score of 10/10 affecting his ability to sleep, ambulate, and perform ADLs. A pain score of 10/10 is considered severe pain and requires rational use of opioids for pain relief when other treatment strategies have failed. The fentanyl patch is long acting and provides around-the-clock relief and is changed every 72 hours. Prior to starting the opioid treatment, a urine toxicology screen was performed and found to be negative. The patient was given an Opioid Risk Tool (ORT) and scored 2/10. The patient signed an opioid use agreement, covering his knowledge of risks, benefits, and alternates treatments and safety issues related to safely using and storing the opioid patches.

Because this patient's pain is mostly chronic, it requires communicating realistic expectations. It is important to discuss that complete freedom from pain may not be achievable. The focus should be on increasing function and mobility and relieving associated psychological stressors, as well as minimizing the risk of opioid misuse, abuse, and addiction that can be associated with long-term analgesic opioids.

For breakthrough pain, oxycodone 5 mg (without acetaminophen) was ordered q 6 hours PRN.

Follow-up Pain Assessment and Treatment

The patient was assessed for pain and functional status. He was examined and found to be using the fentanyl patch that was located on his right upper chest and requiring the oxycodone only prior to dressing change. He reported a pain score of 3/10 with the use of the fentanyl patch and the gabapentin titrated up to 300 mg twice a day. He reported that he could live with this pain level.

After the infection cleared, the patient did not require the acetaminophen but said he would still use it for his arthritis pain and would like to try it prior to dressing change to see if it would be helpful so that he could avoid the oxycodone altogether. He reported that he was sleeping and walking better and able to get up and down the stairs with less discomfort. The oxycodone was titrated down to 5 mg, 30 minutes prior to dressing change. The patient was started on a compression dressing.

The patient was evaluated by psychiatry and was not found to have a major depressive disorder but agreed to attend the chronic pain support group available at the hospital and reported that it was helpful to feel that he was not alone with his pain.

Opioid-induced constipation was treated with a combination of Colace, Senna, and Dulcolax.

Show What You Know

1. Which of the statements listed below most accurately defines pain? Pain is:

A. an objective finding based on prolonged elevation of the patient's blood pressure and pulse rate.
B. a state of discomfort evidenced by the person being unable to sleep.
C. a physical consequence of wound care.
D. whatever the experiencing person says it is.

2. Which of the following statements best describes the Numeric Pain Intensity Scale? It is a:

A. 10-cm line with the words "no pain" at one end and "worst possible pain" at the other end.
B. series of faces ranging from smiling to frowning.

C. rainbow of colors starting with green and ending with red.
D. decision tree for determining which medications to give to a person experiencing pain.

3. According to the WHO analgesic ladder, which medications should you use initially for relief of mild pain?

A. None
B. Nonopioid with or without an adjuvant
C. Opioid with or without an adjuvant
D. Opioid

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PART II

Wound Classifications and Management Strategies

Pressure Ulcers

13

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Objectives

After completing this chapter, you'll be able to:

- discuss the significance of pressure ulcers as a healthcare problem
- explain the etiology of a pressure ulcer
- describe how to complete a risk assessment tool
- discuss strategies for pressure ulcer prevention
- define pressure ulcer classification systems
- discuss strategies for treating a patient with pressure ulcers
- state how to determine pressure ulcer prevalence and incidence.

Pressure Ulcers as a Healthcare Problem

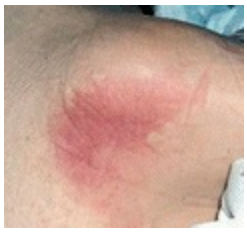
Pressure ulcers are a global healthcare concern and require an interdisciplinary approach to care and management.[1,2] All clinicians need to be responsible for the prevention and treatment of pressure ulcers.

Over the centuries, pressure ulcers have been referred to as decubitus ulcers, bedsores, and pressure sores. The term *pressure ulcer* has become the preferred name because it most closely describes the etiology and resultant ulcer. The 2014 pressure ulcer clinical guideline written by the National Pressure Ulcer Advisory Panel (NPUAP), European Pressure

Ulcer Advisory Panel (EPUAP), and the Pan Pacific Pressure Injury Alliance (PPPIA) released this common definition: "A pressure ulcer is a localized injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear. A number of contributing or confounding factors are also associated with pressure ulcers; the significance of these factors is yet to be elucidated." Pressure ulcers are usually located over bony prominences (such as the sacrum, coccyx, hips, heels) and are classified according to the extent of the type of observable tissue damage.[1] Relying just on *depth* of tissue damage rather than tissue type may be misleading because pressure ulcers in locations where there is little adipose tissue, such as the ear, may be shallow but still extend through the subcutaneous tissue[1] (Box 13-1).

Box 13-1 International NPUAP/EPUAP/PPPIA Pressure Ulcer Classification System

Category/Stage I: Nonblanchable Erythema



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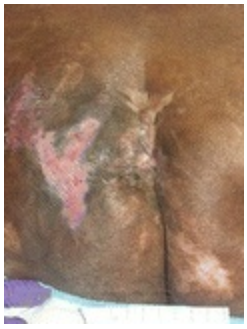
- Intact skin with nonblanchable redness of a localized area, usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its color may differ from the surrounding area.
- The area may be painful, firm, soft, warmer, or cooler as compared to adjacent tissue. Category/Stage I may be difficult to detect in individuals with dark skin tones. May indicate "at-risk" persons (a heralding sign of risk).

Category/Stage II: Partial-Thickness Skin Loss



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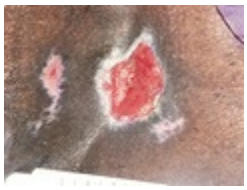
- Partial-thickness loss of dermis presenting as a shallow open ulcer with a red-pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled blister. Presents as a shiny or dry shallow ulcer without slough or bruising.* This Category/Stage should not be used to describe skin tears, tape burns, perineal dermatitis, maceration, or excoriation.

Category/Stage III: Full-Thickness Skin Loss



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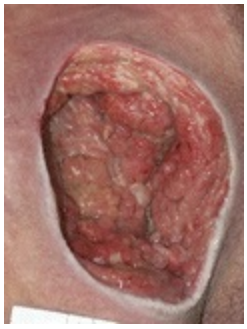
- Full-thickness tissue loss. Subcutaneous fat may be visible, but bone, tendon, or muscle are *not* exposed. Slough may be present but does not obscure the depth of tissue loss. *May* include undermining and tunneling.
- The depth of Category/Stage III pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have subcutaneous tissue, and Category/Stage III ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep Category/Stage III pressure ulcers. Bone/tendon is not visible or directly palpable.

Category/Stage IV: Full-Thickness Tissue Loss



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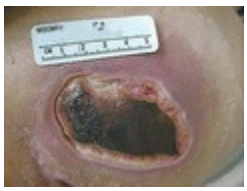
- Full-thickness tissue loss with exposed bone, tendon, or muscle. Slough or eschar may be present on some parts of the wound bed. Often includes undermining and tunneling.
- The depth of a Category/Stage IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have subcutaneous tissue, and these ulcers can be shallow. Category/stage IV ulcers can extend into muscle and/or supporting structures (e.g., fascia, tendon, or joint capsule), making osteomyelitis possible. Exposed bone/tendon is visible or directly palpable.

Unstageable: Depth Unknown



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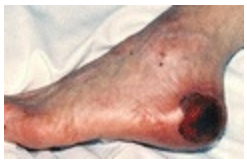
- Full-thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, gray, green, or brown) and/or eschar (tan, brown, or black) in the wound bed.
- Until enough slough and/or eschar is removed to expose the base of the wound, the true depth, and therefore Category/Stage, cannot 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.

Suspected Deep Tissue Injury: Depth Unknown



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- 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, or warmer or cooler as compared to adjacent tissue.
- Deep tissue injury may be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. The wound may further evolve and become covered by thin eschar. Evolution may be rapid, exposing additional layers of tissue even with optimal treatment.

*Bruising indicates suspected deep tissue injury.
From Haesler, E, ed., National Pressure Ulcer Advisory Panel (NPUAP), European Pressure Ulcer Advisory Panel (EPUAP), Pan Pacific Pressure Injury Alliance (PPPIA). *Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline*. Perth, Australia: Cambridge Media, 2014:40-41. Used with permission.

The incidence and prevalence of pressure ulcers are truly enigmatic. Pressure ulcers aren't a reportable event in all healthcare settings, so data are speculative at best. We do know, however, that the numbers are significant enough to warrant national healthcare initiatives in the United States as well as other countries. As early as 1989, the US federal government focused its attention on pressure ulcers with the appointment of a panel charged with developing the Agency for Health Care Policy and Research (AHCPR) guidelines.[3,4] Since that time, clinical practice guidelines regarding pressure ulcers have been released by several other organizations, including the Wound, Ostomy and Continence Nurses Society (WOCN),[5] the Registered Nurses' Association of Ontario (RNAO),[6] the Wound Healing Society (WHS),[7] the American Medical Directors Association (AMDA),[8] and the Association for the Advancement of Wound Care (AAWC).[9]

Long-term care in the United States has long been regulated regarding pressure ulcers as mandated by Federal Tag (F-Tag) 314.[10] In addition, the Agency for Healthcare Research and Quality (AHRQ; formerly AHCPR) stated in its 2008 statistical brief report that there was nearly an 80% increase in hospital stays in which pressure ulcers were noted, even though the total number of hospitalizations for the reported time period (1993–2006) increased by only 15%.[11] Given the attention from regulatory and quality improvement agencies on pressure ulcer occurrence, preventing pressure ulcers should be a priority of patient care across all care settings.

The financial costs associated with pressure ulcers to all institutions and facilities aren't precisely known. According to the Centers for Medicare and Medicaid Services (CMS), the average cost of pressure ulcers for hospitalized patients in 2007 was $43,180.[11] Published estimates of treatment costs vary across hospitals, long-term care, and home care settings; the one certainty is that pressure ulcers do create a financial burden for the facility, patient, and family alike. Pressure ulcers cost institutions valuable staff time, supplies, and reputation.

Pressure ulcer practices should be evidence based. However, adequate research-based, randomized clinical studies to support all of our current practices do not exist. For example, in the NPUAP/EPUAP/PPPIA clinical practice guideline, there are 575 recommendations, where the strength of the evidence for each recommendation is ranked with a letter based on the type of studies. Recommendations given an A are "supported by direct scientific evidence from properly designed and implemented controlled trials on pressure ulcers in humans (or humans at risk for pressure ulcers), providing statistical results that consistently support the recommendations (level 1 studies required)."[1] In the NPUAP/EPUAP/PPPIA clinical practice guideline, there are six recommendations at the A level of strength of evidence (Box 13-2).[1] Wound care interventions and modalities have often been based on an "it works for me" attitude. New to the NPUAP/EPUAP/PPPIA clinical guideline is an indication of the strength of each recommendation. Based on consensus voting by the experts who developed the guideline, a system using thumbs up or thumbs down is given to indicate how confident a healthcare professional can be that the recommendation will improve patient outcomes.[1] There are five different possibilities of strength of recommendation to help prioritize interventions. For example, one thumb up () means a weak positive recommendation;

probably do it, while two thumbs up () means a strong positive recommendation; definitely do it.[1] We need to encourage healthcare providers to participate in research studies so that we'll have more evidence in the future to direct and improve our clinical decision-making process, thereby improving patient outcomes.

Box 13-2 NPUAP/EPUAP/PPPIA Clinical Practice Guideline Recommendations at Strength of Evidence = A

- Offer high-calorie, high-protein nutritional supplements in addition to the usual diet to adults with nutritional risk and pressure ulcer risk, if nutritional requirements cannot be achieved by dietary intake. (Strength of Evidence = A; Strength of Recommendation = )
- Reposition all individuals at risk of or with existing pressure ulcers, unless contraindicated (Strength of Evidence =A; Strength of Recommendation = )
- Consider the pressure redistribution support surface in use when determining the frequency of repositioning. (Strength of Evidence = A; Strength of Recommendation = )
- Use a high-specification reactive foam mattress rather than a non–high-specification reactive foam mattress for all individuals assessed as being at risk for pressure ulcer development. (Strength of Evidence = A; Strength of Recommendation = )
- Consider the use of direct contact (capacitive) electrical stimulation to facilitate wound healing in recalcitrant Category/Stage II pressure ulcers as well as any Category/Stage III and IV pressure ulcers. (Strength of Evidence = A; Strength of Recommendation = )
- Regularly reposition the older adult who is unable to reposition independently. There is no evidence of the

superiority of one higher-specification foam mattress over an alternative higher-specification foam mattress. (Strength of Evidence = A; Strength of Recommendation = )

Haesler E, ed. National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel and Pan Pacific Pressure Ulcer Injury Alliance. *Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline*. Perth, Australia: Cambridge Media 2014. Used with permission.

Wound Etiology

How do pressure ulcers occur? This is an interesting and challenging question. Literature reviews demonstrate several etiologies of pressure ulcers. Earlier reviews focused on a model of pressure ulcer development caused by pressure-induced capillary closure cutting off blood supply leading to tissue ischemia, injury, and death. More recent research, using techniques such as magnetic resonance imaging (MRI), has documented cellular distortion and damage from pressure due to mechanical loading and deformation of soft tissues near bony prominences.[1] There is also a renewed appreciation for the effects of shear in damaging deeper tissue and the emerging role that microclimate (moisture and temperature) in rendering tissue less tolerant of the effects of pressure might affect.[1,12]

The recent international NPUAP/EPUAP/PPPIA clinical practice guideline states that "pressure ulcers develop as a result of the internal response to external mechanical load."[1] Simply stated, it is how a person's soft tissue responds to sustained pressure (mechanical loading). The latest explanation from the NPUAP/EPUAP/PPPIA[1] as to the etiology of pressure ulcers involves the interplay of mechanical loading and an individual's tissue tolerance. Mechanical load is a complex process that research is still contributing to our understanding. How mechanical loading affects tissues varies and depends on several characteristics including tissue size, shape, stiffness, strength, and diffusion properties as well as the magnitude and length of time that mechanical loading is applied to the tissue.[1] The pressure gradient[1,4,13–16] has been used to explain how pressure translates into tissue death (Fig. 13-1). External pressure is transmitted from the epidermis inward toward the bone as well as by counter-pressure from the bone. As a result, the loaded soft tissues, including skin and deeper tissues (adipose

tissue, connective tissue, and muscle), will deform, resulting in strain and stress within the tissues.[1]

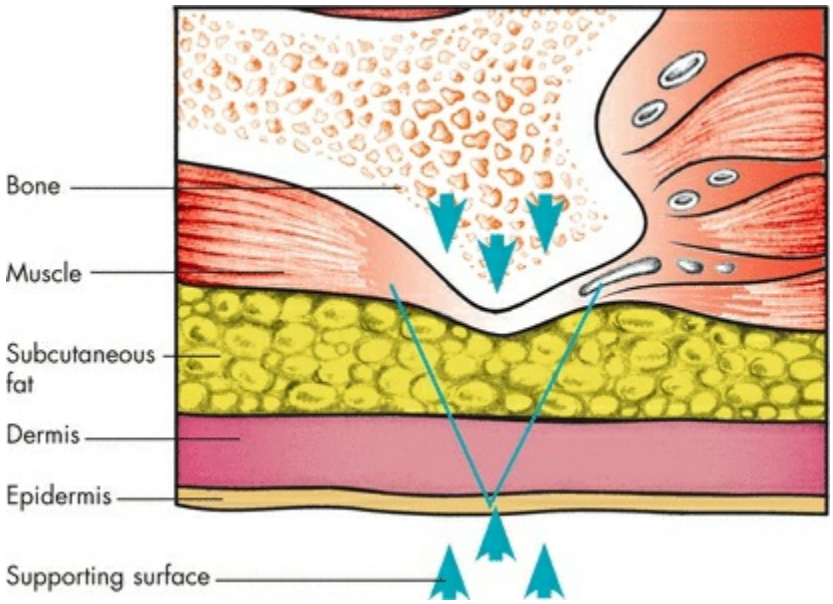

Figure 13-1. Pressure gradient. In this illustration, the V-shaped pressure gradient results from the upward force (upward arrowheads) exerted by the supporting surface against downward force (*downward arrowheads*) exerted by the bony prominence. Pressure is greatest on tissues at the apex of the gradient and lessens to the right and left of this point.

Practice Point

Muscle tissue dies first from pressure. Look at a variety of extrinsic and intrinsic factors that could put your patient at risk for pressure ulcers.

Body tissues differ in their ability to tolerate pressure. The blood supply to the skin originates in the underlying muscle. Muscle is more sensitive to pressure damage than skin tissue.[1] Tissue tolerance is further compromised by extrinsic and intrinsic factors. Extrinsic factors that are gaining momentum as to the role they may play in pressure ulcer development include moisture, and temperature, and are usually collectively referred to as microclimate.[1] NPUAP/EPUAP/PPPIA[1] suggests that microclimate plays a role in the development of Category/Stage I and II

pressure ulcers. They also suggest that another intrinsic factor, tissue perfusion (or lack thereof), and the resulting ischemia may be another important part of explaining how pressure ulcers occur.[1] A 2013 comprehensive systematic review of risk factor research by Coleman and colleagues helps explain the complex interaction between increased pressure intensity/duration and the extrinsic and intrinsic factors that affect tissue tolerance to pressure.[1,17] Based on their review of 54 studies of 34,449 patients in both acute care and the community, they identified three primary independent predictors for pressure ulcer development: mobility/activity, perfusion (including diabetes), and skin/pressure ulcer status.[17] Activity and mobility limitations create the necessary conditions for pressure ulcers to develop (i.e., unrelieved pressure). Individuals who are bedbound, chairfast, and unable to effectively reposition themselves fall into these risk factor categories and should be considered at risk. Epidemiological studies show that limitations in activity and mobility are independently predictive of pressure ulcers. Changes in sensory perception may further impair movement.

Once the conditions for increased pressure intensity and duration are established, intrinsic and extrinsic factors affecting tissue tolerance contribute to pressure ulcer development. According to epidemiological evidence, these factors fall under several categories, which include factors affecting perfusion and oxygenation (e.g., hypotension, hemodynamic instability, peripheral vascular disease, diabetes, vasopressor drugs, need for supplemental oxygen); poor nutritional status (e.g., decreased intake of nutrients—especially protein, weight loss, low albumin); and skin moisture (e.g., urinary or fecal incontinence, excessive sweating, or wound drainage).[1] There is less evidence to support the following advanced age, friction, immunity, poor general health status, and increased body temperature.[1]

Shear has the potential to damage deeper tissue. Shear and friction are two separate phenomena, yet they often work together to create tissue ischemia and ulcer development.

Friction is a force that is parallel to the skin surface and may damage the epidermis causing blisters but not pressure ulcers.[1] The tissue injury resulting from friction may look like a superficial skin insult.

Shear (shear stress) is "the force per unit area exerted parallel to the plane of interest"[1] while shear strain is the "distortion or deformation of tissue as a result of shear stress."[1] You can think of shear stress and strain

as pulling the bones of the pelvis in one direction and the skin in the opposite direction (Figs. 13-2 and 13-3). The deeper fascia slides downward with the bone, while the superficial fascia remains attached to the dermis. This insult and compromise to the blood supply create ischemia, reperfusion injury, lymphatic impairment, and mechanical deformation of tissue cell[18,19] and lead to cellular death and tissue necrosis. Shear and friction go hand in hand—you'll rarely see one without the other.

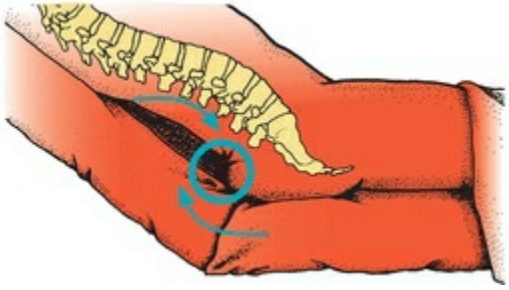

Figure 13-2. Shearing force. Shear injury is a mechanical force parallel, rather than perpendicular, to an area of tissue. In this illustration, gravity pulls the body down the incline of the bed. The skeleton and attached deep fascia slide within the skin, while the skin and superficial fascia, attached to the dermis, remain stationary, held in place by friction between the skin and the bed linen. This internal slide compromises blood supply to the area and deforms or distorts tissue.

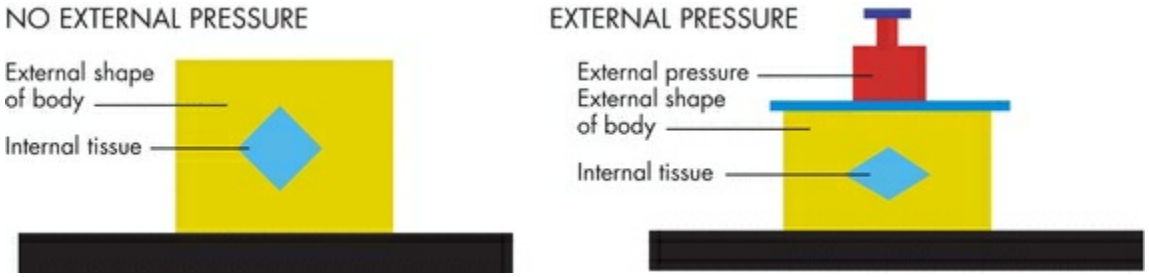

Figure 13-3. Effects of shear on tissue. ©2006, C.W.J. Oomens. Used with permission.

Practice Point

You won't see shear injury initially at the skin level because it occurs underneath the skin. You will see friction injury. Elevation of the head of the bed increases shear injury in the deep tissue and may account for the number of sacral ulcers we see in practice.

Exactly what causes pressure ulcers remains controversial.[20] Theories on the etiology of pressure ulcers need continued research.

Most pressure ulcers occur in the lower part of the body over bony prominences such as the sacrum, coccyx, ischial tuberosities, greater trochanters, heels, iliac crests, and lateral and medial malleoli[13,21] (Fig. 13-4). Other areas, where pressure ulcers may be overlooked, include the occiput (especially in infants and toddlers; see Chapter 22, Pressure ulcers in neonatal and pediatric populations), elbows, scapulae, and ears (especially in patients using nasal oxygen cannulas).

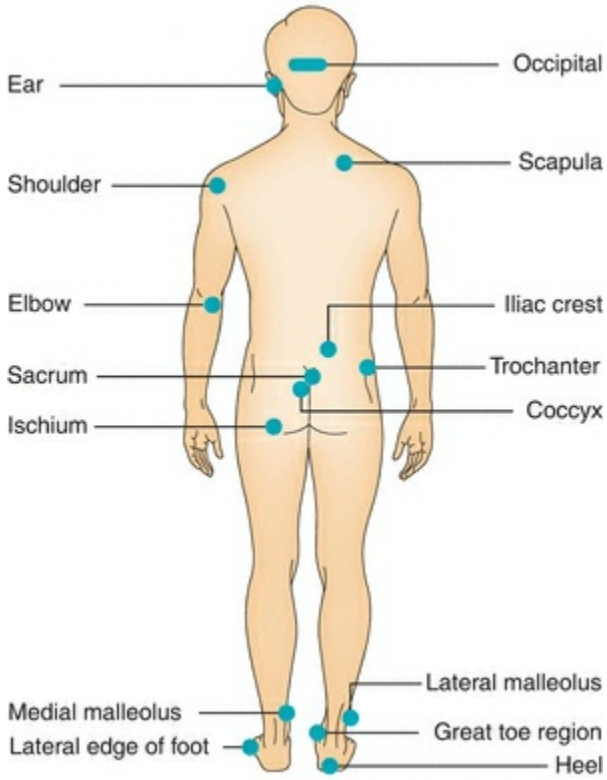

Figure 13-4. Pressure ulcer sites. Shown in this illustration are the most common sites where pressure ulcers develop.

Several national surveys demonstrated that the most common site for pressure ulcers among patients in acute care facilities is the sacrum, with the heels being second.[21] The incidence of heel ulcers has increased incrementally over the past decade, creating a need for prevention protocols targeting the heels. The HEELS© mnemonic[22] can be used to care for heels at risk for pressure ulcers (Box 13-3). A growing clinical problem is pressure ulcers caused by medical devices (Fig. 13-5). In a survey of 86,932 patients in acute care facilities, 9.1% of all pressure ulcers

identified were device related, with the ear being the most common site (20%).[21] Devices that are commonly associated with pressure ulcers include respiratory devices such as oxygen tubing (ears), nasotracheal tubes (ET, mouth and lips), continuous positive airway pressure (CPAP) masks, and biphasic positive airway pressure (Bi-PAP) (bridge of nose, face); orthopedic devices including cervical collars (neck and head), halo devices, external fixators, plaster casts; and other medical devices such as splints, braces, urinary catheters, fecal containment devices, venous or dialysis catheters, heel suspension devices, thromboembolic deterrent hose, restraints, graduated compression stockings, and intermittent pneumatic compression device sleeves (lower extremities).[1] Any tube under pressure can create pressure damage. Edematous patients are at particularly high risk.[23] Pressure ulcers on the mucosa from medical devices are not staged using the NPUAP International Pressure Ulcer Classification System. A position paper and helpful posters to use to educate clinicians about mucosal pressure ulcers can be found on the NPUAP Web site.[24]

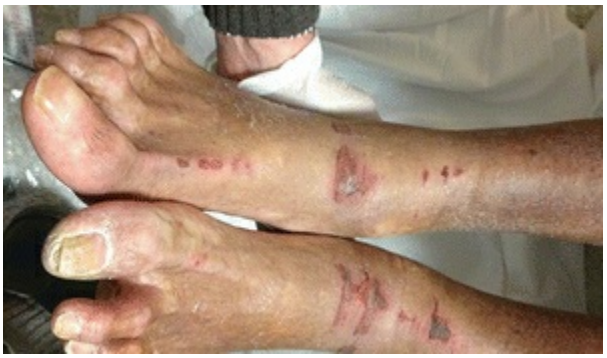

Figure 13-5. Medical device injury. © J. M. Levine, MD

Box 13-3 HEELS © Mnemonic

Have foot or leg movement?
Evaluate heels and sensation.
Evaluate foot drop risk.
Limit friction.
Suspend heels with devices as needed.

Used with permission from Cuddigan, J., Ayello, E.A., Black, J. "Saving Heels in Critically Ill Patients," *World Council of Enterostomal Therapists Journal* 28(2):16-24, 2008. Copyright © 2005, Ayello,

Cuddigan, and Black.

Practice Point

Careful observation of the sacrum/coccyx and heels is warranted because these are the most frequent sites of pressure ulcers.

Prevention

Preventing pressure ulcers is of vital importance. Elements of pressure ulcer prevention include identifying individuals at risk for developing pressure ulcers, preserving skin integrity, treating the underlying causes of the ulcer, relieving pressure, paying attention to the total state of the patient to correct any deficiencies, and educating the patient and his or her family about pressure ulcers.

Risk Factors and Risk Assessment

Risk assessment is used to identify patients at risk and the type of risk. Identifying individuals at risk for pressure ulcers enables clinicians to make decisions about when to begin using preventive measures. This is important for the most effective use of resources because the type of risk guides the intensity and cost of preventive interventions. Risk assessment is governed by the regulations in effect in a particular setting. For example, the Outcome and Assessment Information Set (OASIS-C) guidelines for home care recommend that a structured approach to risk assessment be conducted on all home care clients.[25,26] MDS 3.0 describes several methods for determining a long-term care resident's pressure ulcer risk, which may include a formal assessment instrument, a clinical assessment conducted by the clinician of the skin, comorbidities, health and functional status, an existing stage 1 or higher pressure ulcer, a scar over a bony prominence, or a nonremovable dressing or device.[27–29] The NPUAP/EPUAP/PPPIA guideline considers adults that have a medical device to be at risk for pressure ulcers based on a B strength of evidence and a two thumbs up strength of recommendation for this practice.[1] They recommend that the skin under or around a medical device(s) should be inspected at least twice daily for signs of pressure-related injury.[1]

Risk assessment is a comprehensive process that some clinicians in the past thought only included use of the many validated pressure ulcer risk assessment tools that are available, including the Norton Scale,[30] the Gosnell Scale,[31] the Braden Scale,[32] and the Waterlow Scale.[33] The NPUAP/EPUAP/PPPIA clinical guideline emphasizes that these risk assessment tools are only part of a systematic and complete assessment for pressure ulcer risk.[1] Deciding which scale to use can be challenging. Reviewing the reliability (consistency) and validity (accuracy) of each scale should always be the first step in the decision-making process. Reliability for risk assessment scales is usually described in terms of interrater reliability. According to Ayello and Braden,[34] "a common measure of inter-rater reliability for a risk assessment tool is percentage agreement, which looks at the percentage of instances in which different raters assign the same score to the same patients. Validity, or accuracy, is measured by the ability of the tool to correctly predict who will or won't develop a pressure ulcer." There are several risk assessment tools used to identify those at high risk. Chou and colleagues identified over 747 full text articles and, based on 120 studies, eventually included in their review that while there was no difference among the instruments in diagnostic accuracy, the Braden, Norton, and Waterlow Scales did identify patients at increased risk but were weak predictors.[35] Furthermore, they urged more research to better understand how these tools compared with clinical judgment regarding pressure ulcer incidence. A 2014 meta-analysis by Garcia-Fernandez and colleagues of risk assessment tools has provided new insights regarding risk assessment tools.[36]

Predictive validity is dependent on the sensitivity and specificity of the tool. Sensitivity is "the percentage of individuals who develop a pressure ulcer who were assessed as being at risk for a pressure ulcer. A tool has good sensitivity if it correctly identifies true positives while minimizing false negatives. Specificity is the percentage of individuals who don't develop a pressure ulcer who were assessed as being not at risk for developing an ulcer. A tool has good specificity if it identifies true negatives and minimizes false positives."[34,37] Because of the amount of clinical research supporting their reliability and validity, the Norton,[30] Braden,[23] and Waterlow[33] Scales are primarily used in clinical practice to determine pressure ulcer risk assessment.

Braden Scale

The Braden Scale is the most commonly used pressure ulcer assessment tool in the United States. Available in many languages (English, French, Portuguese)[38–40] and used worldwide, this copyrighted tool was created in 1987 by Barbara Braden and Nancy Bergstrom[32] and is available at http://www.bradenscale.com/images/bradenscale.pdf.[38] The Braden Scale has six subscales: sensory perception, moisture, activity, mobility, nutrition, and friction/shear[32,34,37] (Table 13-1). The scale is based on the two primary etiologic factors of pressure ulcer development—intensity and duration of pressure and tissue tolerance for pressure. "Sensory perception, mobility, and activity address clinical situations that predispose a patient to intense and prolonged pressure, while moisture, nutrition, and friction/shear address clinical situations that alter tissue tolerance for pressure."[34]

Table 13-1 Braden Scale: Predicting Pressure Ulcer Risk

SENSORY PERCEPTION Ability to respond meaningfully to pressure-related discomfort	1. Completely limited: Unresponsive (doesn't 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 surface	2. Very limited: Responds only to painful stimuli. Can't communicate discomfort except by moaning or restlessness OR has a sensory impairment that limits the ability to feel pain or discomfort over half of body	3. Slightly limited: Responds to verbal commands but can't always communicate discomfort or need to be turned OR has some sensory impairment that limits ability to feel pain or discomfort in one or two extremities	4. No impairment: Responds to verbal commands. Has no sensory deficit that would limit ability to feel or voice pain or discomfort
MOISTURE Degree to which skin is exposed to moisture	1. Constantly moist: Skin is kept moist almost constantly by perspiration or urine. Dampness is detected every time patient is moved or turned.	2. Often moist: Skin is often but not always moist. Linen must be changed at least once per shift.	3. Occasionally moist: Skin is occasionally moist, requiring an extra linen change approximately once per day.	4. Rarely moist: Skin is usually dry; linen only requires changing at routine intervals.
ACTIVITY Degree of physical activity	1. Bedfast: Confined to bed.	2. Confined to chair: Ability to walk severely limited or nonexistent. Can't bear own weight and must be assisted into chair or wheelchair	3. Walks occasionally: Walks occasionally during day, but for very short distances, with or without assistance; spends majority of each shift in bed or chair	4. Walks frequently: Walks outside the room at least twice per day and inside room at least once every 2 h during waking hours
MOBILITY Ability to change and control body position	1. Completely immobile: Doesn't make even slight changes in body or extremity position without assistance	2. Very limited: Makes occasional slight changes in body or extremity position but unable to make frequent or significant changes independently	3. Slightly limited: Makes frequent though slight changes in body or extremity position independently	4. No limitations: Makes major and frequent changes in position without assistance
NUTRITION Usual food intake pattern NPO: Nothing by mouth IV: Intravenously TPN: Total parenteral nutrition	1. Very poor: Never eats a complete meal. Rarely eats more than one-third of any food offered. Eats two servings or less of protein (meat or dairy products) per day. Takes fluids poorly. Doesn't take a liquid dietary supplement OR is NPO or maintained on clear liquids or I.V. fluids for more than 5 d	2. Probably inadequate: Rarely eats a complete meal and generally eats only about half of any food offered. Protein intake includes only three servings of meat or dairy products per day. Occasionally will take a dietary supplement OR receives less than optimum amount of liquid diet or tube feeding	3. Adequate: Eats over half of most meals. Eats a total of four servings of protein (meat, dairy products) each day. Occasionally will refuse a meal, but will usually take a supplement if offered OR is on a tube feeding or TPN regimen that probably meets most nutritional needs	4. Excellent: Eats most of every meal and never refuses a meal. Usually eats a total of four servings of meat and dairy products. Occasionally eats between meals. Doesn't require supplementation
FRICTION AND SHEAR	1. Problem: Requires moderate to maximum assistance in moving. Complete lifting without sliding against sheets is impossible. Frequently slides down in bed or chair, requiring frequent repositioning with maximum assistance. Spasticity, contractures, or agitation leads to almost constant friction.	2. Potential problem: Moves feebly or requires minimum assistance. During a move, skin probably slides to some extent against sheets, chair, restraints, or other devices. Maintains relatively good position in chair or bed most of the time but occasionally slides down	3. No apparent problem: Moves in bed and in chair independently and has sufficient muscle strength to lift up completely during move. Maintains good position in bed or chair at all times	

Each subscale contains a numerical range of scores, with 1 being the lowest score possible.[32] The sensory perception, moisture, activity, mobility, and nutrition subscales have scores ranging from 1 to 4. Friction/shear is the only subscale in which scores range from 1 to 3. Definitions of each subscale as to what patient characteristics to evaluate are given for each numerical ranking. While in the past emphasis was on the overall Braden Scale score, which was derived from totaling the numerical ratings from each of the six subscales, the 2014 NPUAP/EPUAP/PPPIA clinical guideline now recommends that the subscale scores and other patient risk factors not captured on risk assessment scales should be included as part of an individualized pressure ulcer prevention plan of care.[1]

Both the CMS[27] and the NPUAP/EPUAP/PPPIA clinical guideline[1] stress the importance of using a systematic process for determining pressure ulcer risk that may include clinical judgment, skin assessment, and review of risk factors not part of validated pressure ulcer risk assessment tools and not just relying on interpreting the total Braden Scale score because not all risk factors are quantified on the scale (low subscale scores need to be considered). The systematic review of patient risk factors for pressure ulcer development by Coleman and colleagues[17] also underscores the importance of a comprehensive risk assessment when they state "overall there is no single factor which can explain pressure ulcer risk, rather a complex interplay of factors which increase the probability of pressure ulcer development."

Determining all factors that place an individual at risk for a pressure ulcer is helpful when deciding on appropriate prevention strategies. Once a person is determined to be at risk for a pressure ulcer, preventive protocols based on the individualized assessment need to be implemented. Recommendations for pressure ulcer prevention management by level of risk are available on the Braden Web site.[39] However, as the NPUAP/EPUAP/PPPIA has emphasized in the 2014 clinical guideline,[1] the individualized plan of care for any particular patient needs to be based on his or her specific risk factors and needs.

Questions have surfaced regarding when to assess patients for pressure ulcer risk and when to reassess risk. These two aspects of care are both very important. The NPUAP/EPUAP/PPPIA clinical practice guideline recommends that initial pressure ulcer risk be assessed as soon as possible after admission (within 8 hours).[1] Reassessment interval should be based

on the person's acuity and if there is any change in the patient's condition[1] (Box 13-4). Studies by Bergstrom and Braden[40,41] found that in skilled nursing facilities, 80% of pressure ulcers develop within 2 weeks of admission and 96% develop within 3 weeks of admission.

Box 13-4 Pressure Ulcer Risk Assessment Recommendations

General Recommendations for Structured Risk Assessment

1. Conduct a structured risk assessment as soon as possible (but within a maximum of 8 hours after admission to identify individuals at risk of developing pressure ulcers). (Strength of Evidence = C; Strength of Recommendation = )
2. Repeat the risk assessment as often as required by the individual's acuity. (Strength of Evidence = C; Strength of Recommendation = )
3. **Undertake a reassessment if there is any significant change in the individual's condition.** (Strength of Evidence = C; Strength of Recommendation = )
4. Include a comprehensive skin assessment as part of every risk assessment to evaluate any alterations to intact skin. (Strength of Evidence = C; Strength of Recommendation = )
5. Document all risk assessments. (Strength of Evidence = C; Strength of Recommendation = )
6. Develop and implement a risk-based prevention plan for individuals identified as being at risk of developing pressure ulcers. (Strength of Evidence = C; Strength of Recommendation = )

Caution: Do not rely on a total risk assessment tool score alone as a basis for risk-based prevention. Risk assessment tool

subscale scores and other risk factors should also be examined to guide risk-based planning.

Structured Risk Assessment

1. Use a structured approach to risk assessment that is refined through the use of clinical judgment and informed by knowledge of relevant risk factors. (Strength of Evidence = C; Strength of Recommendation = )

Risk Factor Assessment

1. Use a structured approach to risk assessment that includes assessment of activity/mobility and skin status. (Strength of Evidence = B; Strength of Recommendation = )

Activity and Mobility Limitations

1. **Consider bedfast and/or chairfast individuals to be at risk of pressure ulcer development.** (Strength of Evidence = B; Strength of Recommendation = )
2. Consider the impact of mobility limitations of pressure ulcer risk. (Strength of Evidence = B; Strength of Recommendation = )

Haesler E, ed. National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel and Pan Pacific Pressure Ulcer Injury Alliance. *Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline*. Perth, Australia: Cambridge Media 2014. Used with permission.

Risk Assessment Acute Care

In the United States, there is no CMS-mandated frequency of risk assessment for acute care hospitalized patients. The WOCN guidelines[5] recommend reassessment on a regularly scheduled basis or when there is a significant change in the individual's condition such as surgery or decline in health status. The Institute for Healthcare Improvement (IHI) recommends that pressure ulcer risk assessment be done every 24 hours.[42] The World Union of Wound Healing Societies[43] recommends that assessments be performed

daily in the intensive care unit and every 2nd day on general medical/surgical floors.

Risk Assessment LONG-TERM CARE

Assess initially upon admission, then reassess weekly for the first 4 weeks, monthly to quarterly after that, and whenever the resident's condition changes.[40] Pressure ulcer risk assessment is now required under MDS 3.0 section M, Skin Conditions.[27–29] Under section M0 100 of the MDS tool, the clinician can use *any* of the following criteria to determine risk:

- M0100A: Stage I or higher ulcer, scar over a bony prominence, or nonremoval dressing or device in place[27–29]
- M0100B: Formal assessment instrument or tool (i.e., Braden, Norton, or others)[27–29]
- M0100C: Comprehensive clinical assessment of the resident since this will include factors other than what is on formal tools[27–29]

Based on the determination of pressure ulcer risk by some methods in section M0100, in M0150, the clinician must check Yes or No to indicate whether the resident is at risk for developing pressure ulcers.[27–29] Further details about MDS 3.0 section M skin conditions can be found on the CMS Web site[27] or in the literature.[28,29]

Risk Assessment HOME HEALTH CARE

The plan of care needs to address the areas of risk for home care patients. A Braden Scale for the home care setting is available online[39] and in the literature.[32] Risk assessment tools complement clinical judgment, as patients with the same risk score may have differing actual risks. Section M1300, Pressure Ulcer Assessment of OASIS-C, identifies whether the home health agency providers assessed the patient's risk of developing pressure ulcers by either evaluation of clinical factors or use of a standardized tool. CMS does not require the use of standardized tools, nor does it endorse one particular tool.[25,26] Section M1302 addresses the question of whether the patient is at risk for developing pressure ulcers, with the answer being either No or Yes. Reassessment of risk should be performed with each home visit by a nurse.

Practice Point

CMS requires a pressure ulcer risk assessment in both long-term and home care settings. It *does not* mandate the use of a standardized risk tool (e.g., Braden, Norton).[25–27]

Patient Care to Prevent Pressure Ulcers

Preventing pressure ulcers can best be accomplished by using a multidisciplinary approach. Several pressure ulcer prevention protocols and guidelines exist,[1,2,4–9,44–47] most of which advocate taking a holistic approach to pressure ulcer prevention. This includes processes on both the healthcare organizational level and the healthcare provider level that outline an evidence-based plan of care.[1] The healthcare organization should have in place a systematic structure for monitoring and managing pressure ulcer care practices and incidence rates. Quality indicators should be developed and monitored to determine future improvements in care processes needed at the organizational level.[1] Any good prevention program for the practitioner begins with assessing the patient's skin. The skin should be assessed and its condition documented daily in acute- and long-term care settings and at each home care visit. Careful attention to preventing skin injury during performance of activities of daily living is paramount. The bathing schedule should be individualized based on the patient's age, skin texture, and dryness or excessive oiliness of the skin. Use of nondrying products to clean the skin is recommended. One study found that the incidence of stage I and II pressure ulcers could be reduced by educating the staff about using body wash and skin protectant products.[48] In another study by Carr and Benoit, the incidence of pressure ulcers decreased from 7.14% at baseline to 0% at the end of the study through education of nonlicensed staff about the use of protective skin barriers and implementation of a comprehensive interventional patient hygiene bathing and incontinence management program.[49] Avoid excessive friction and hot water when cleaning. Use nonalcoholic moisturizers after bathing. A daily bath may not be needed for all patients; elderly patients, for example, may benefit from "lotion" baths.

For the incontinent patient, moisture barrier products such as creams and ointments should be considered as treatment options. Soiled skin should

be cleaned immediately and products to protect the skin applied. If containment products are used, follow the correct methods of application. Reasons for incontinence should always be determined and appropriate measures to address the cause of the incontinence should be implemented.

Vulnerable skin should be protected from injury. Citing a B strength of evidence, the NPUAP/EPUAP/PPPIA recommends considering the prophylactic use of polyurethane foam dressing on the heel, sacrum, or other at risk locations to prevent pressure ulcers.[1] In a study of 93 high-risk patients in a surgical trauma intensive care unit, the use of intervention bundles that included a prophylactic soft silicone dressing product resulted in zero pressure ulcers.[50] Other authors have found similar results when various types of polyurethane foam or other foam dressings were used on critically ill patients and advocate their use in pressure ulcer prevention.[51–67]

Although a review of the literature suggests that one type of massage may be beneficial for at-risk patients,[68] most clinical guidelines recommend against massaging reddened bony prominences because this can lead to further tissue damage.[1,2,4] Keep the patient's heels off the bed[69]; use pillows, wedges, and foot elevation devices as indicated (Box 13-2). Flex the knee when elevating the leg to avoid pressure injury to the Achilles tendon.[1] Best practices for preventing heel pressure ulcers in orthopedic population are published.[70]

Practice Point

Keep the patient's heels off the mattress!

Be careful not to drag your patient during transfers or position changes. Use appropriate devices, such as a turn sheet or mechanical lifting device, to prevent friction injuries to the patient's skin. Use the 30-degree lateral position for patients in bed. Keep the head of the bed below 30 degrees to prevent shearing injuries, unless contraindicated due to the patient's clinical condition.

The role of nutrition in pressure ulcer interventions has been controversial. The NPUAP/EPUAP/PPPIA has two recommendations with the strength of evidence at the A level (Box 13-1).[1] In a large prospective

cohort study, Bergstrom and Braden[40] found that nursing home residents who developed pressure ulcers had a significantly lower intake of protein. Clinicians need to ensure that a patient's caloric, protein, vitamin, and mineral needs are met. Recommendations from the dietitian can be an important source of assistance in helping patients to get their required nutrients.

Physical and occupational therapists are important members of the pressure ulcer team and a valuable resource for maximizing patient mobility. Their expertise in selecting appropriate-size wheelchairs and evaluating seating angles and postural alignment can't be overemphasized. Patients who are confined to a chair should be repositioned every hour, with small shifts in weight made every 15 minutes. In the past, many clinicians considered turning and repositioning bedridden patients every 2 hours to be a standard of care. However, the NPUAP/EPUAP/PPPIA clinical guideline recommends that repositioning should take into account the individual's condition and type of pressure redistribution support surface.[1] The appropriate turning interval for all patients has yet to be determined by research. A 2-hour interval may be too long for some patients, whereas for others, every 2 hours may not be necessary, such as for palliative care patients in whom frequent repositioning would cause more pain and suffering than benefit. Because there is no standard time interval or frequency for repositioning all patients, a repositioning schedule needs to be individualized for each patient. The most recent NPUAP/EPUAP/PPPIA guideline[1] provides evidence that repositioning frequency may be influenced by the type of support surface being used. Contrary to the earlier AHCPR (AHRQ) recommendation for every 2-hour turning/repositioning,[3,4] evidence from Defloor et al.[71] and Vanderwee et al.[72] found that turning a patient every 4 hours on a viscoelastic mattress resulted in a significant reduction in the incidence of pressure ulcers. Bergstrom and colleagues found that for moderate- to high-risk residents in long-term care who were on high-density foam mattresses, the turning schedule could be 2, 3, or 4 hours.[73]

Practice Point

Repositioning schedules should take into consideration the condition of the patient and the support surface in use.[1]

The NPUAP/EPUAP/PPPIA clinical guideline has provided new recommendations regarding the repositioning of critically ill person that are at the C strength of evidence.[1] This includes beginning a repositioning schedule as soon as possible after the patient is admitted and revising the schedule based on the patient's tolerance to repositioning (two thumbs up).[1] They recommend considering the use of slow, gradual turns that allow the patient sufficient time to stabilize their hemodynamic and oxygenation status.[1] Also, clinicians should consider more frequent small shifts for critically ill persons that cannot tolerate frequent major shifts in position.[1] They caution that these small shifts are a temporary intervention done in conjunction with appropriate use of a redistribution support surface and should be replaced with routine repositioning when the person's hemodynamic status has stabilized.[1] If it is determined for medical reason that a patient who is hemodynamically unstable, or has spinal instability, cannot be turned, then clinicians should evaluate the need to change the patients support surface from a reactive to an active support surface.[1]

Appropriate pressure-relieving devices and surfaces need to be used. Rastinehad reported that using support surfaces can decrease the incidence of pressure ulcers in at-risk oncology patients.[74] Devices such as "donut cushions" should not be used[1]. The type and amount of linens used may make a different in pressure ulcer incidence.[1] Limit the amount of linen and incontinence pads placed on the support surface/bed.[1] "General rule of thumb is less is best."[1] Evidence suggests that silklike bed linens or cotton-blend fabrics reduce shear, friction, and pressure ulcer incidence.[75–77] The results of a Canadian study have led to a model for use in support surface selection.[78] See the NPUAP Web site (http://www.npuap.org) to review the latest definitions of physical concepts related to support surfaces as developed by the NPUAP Support Surface Standards Initiative.[79] (Also, see Chapter 11, Pressure Redistribution: Seating, Positioning, and Support Surfaces.)

Ongoing monitoring and documentation are essential. In a pilot study, Horn et al.[80] found that establishing a multidisciplinary team and redesigning documentation processes for certified nursing assistants in several long-term care facilities throughout the United States resulted in a decrease in the required facility documentation. For the seven facilities in

the study, there was a combined 33% reduction in pressure ulcers.[80]

Milne et al.[81] decreased facility-acquired pressure ulcer prevalence from 41% to 4.2% in a 108-bed long-term acute care hospital (LTACH) by developing policies that were supported by published clinical practice guidelines and incorporating them into the facility's plan of care. They established a wound care team, improved documentation methods, and educated the staff. They also reviewed the facility's wound products and revised its electronic record. All of these efforts resulted in care improvement outcome.[81]

Communication of the prevention plan to all members of the healthcare team, including patients and their families, is imperative. Supplement your verbal teaching with one of the prevention booklets designed for use by the consumer, such as pamphlets available from wound care companies as well as the AHRQ. The AHRQ[82] also has a pressure ulcer patient guide available in Spanish.

Pressure Ulcer Staging

A comprehensive wound assessment includes many parameters, one of which is staging. (See Chapter 6, Wound assessment.) Once the wound etiology is known, the correct classification system to describe the wound can be selected. For example, arterial and venous ulcers are described by their characteristics. Diabetic or neuropathic ulcers are classified by the American Diabetes Association, Wagner Grading System for Vascular Wounds, or San Antonio Diabetic Wound Classification System. The NPUAP staging system was designed specifically for pressure ulcers. An outcome of the first NPUAP consensus conference[83] was the NPUAP staging system for pressure ulcers, which was based on staging systems by Shea[84] and the International Association of Enterostomal Therapists[85] (now called WOCN). In February 2007, NPUAP once again revised its staging system.[86] The latest revision of the pressure ulcer classification system to include six categories or stages was included in the 2014 NPUAP/EPUAP/PPPIA international pressure ulcer guideline.[1]

Pressure ulcer staging is a classification system to describe the level of tissue destroyed. It provides practitioners with a common language to communicate with each other what the pressure ulcer looks like clinically. This staging system should only be used to describe pressure ulcers and not other types of skin or wound injuries. NPUAP states that mucous membrane

pressure ulcers should not be staged using its pressure ulcer staging system.[24] Mucosal pressure ulcers (MPrU) are "pressure ulcers found on mucous membranes with a history of a medical device in use at the location of the ulcer"[24] (Fig. 13-6). Staging is only part of the total assessment of an ulcer; a comprehensive assessment also includes factors such as the state of surrounding skin and presence of infection, among others. (See Chapter 6, Wound assessment.)

Figure 13-6. Mucosal pressure ulcer.

After the present on admission (POA) indicator went into effect in the acute care setting, confusion existed as to whether nurses could continue to stage pressure ulcers. An NPUAP position statement[87] as well as information in the literature from the American Nurses Association (ANA) reaffirms that pressure ulcer staging is within the scope of practice for RNs.[88,89]

NPUAP Classification of Pressure Ulcers Staging Definitions

Pressure ulcers are classified according to the amount of visible tissue loss.[1] Necrotic ulcers cannot be staged numerically because visualization of the wound bed is necessary to determine the level of tissue involvement; therefore, necrotic ulcers are classified as unstageable pressure ulcers. Numerical staging of necrotic wounds should be done after the necrotic tissue is removed. (See Chapter 8, Wound debridement.)

The NPUAP/EPUAP/PPPIA classification system[1] for staging or categories of pressure ulcers is described below.

Stage I

The original definition of a stage I pressure ulcer was "nonblanchable erythema of intact skin, the heralding lesion of skin ulceration."[83] Given the diversity of people with different skin pigmentation, detecting stage I pressure ulcers can be a challenge if clinicians only use color as an indicator. In 1997, to provide a more culturally sensitive definition, NPUAP revised the definition of a stage I ulcer to include indicators that went beyond color. NPUAP continued to refine this definition to include "intact skin with nonblanchable redness of a localized area usually over a bony prominence." In darkly pigmented skin, the area may not have visible blanching and its color may differ from surrounding skin (Box 13-1).

Persons with darkly pigmented skin have the lowest prevalence of stage I pressure ulcers.[90,91] The incidence of pressure ulcers was higher in people with darkly pigmented skin in several studies conducted by Lyder and colleagues.[92,93] Sprigle and colleagues[94] found that warmth or coolness was present in 85% of patients with stage I pressure ulcers. Rosen and colleagues used a quality improvement program for educating staff on assessing the subtle differences in skin color, texture, and warmth in individual with darkly pigmented skin. This resulted in eliminating the racial disparity differences in PU rates between darkly pigmented and lightly pigmented residents.[95] Differences in PU incidence rates may not be due to skin tone differences, but rather, cultural, social, or economic factors' further research may shed new light on this view point.[96]

Staging Concepts

Staging Competency

Accuracy in staging pressure ulcers is a challenge. Nurses have reported being less confident in identifying stage III ulcers.[97–101] Clinicians can test their ability to classify pressure ulcers by taking the ePUCLAS$_2$ staging test (available in English and other languages) on the EPUAP Web site (http://www.epuap.org)[102] or the quiz on the National Database of Nursing Quality Indicators (NDNQI) educational modules.[103]

Suspected Deep Tissue Injury

Clinicians often struggle with the concept of suspected deep tissue injury

(sDTI)[104] that presents as a purplish color, most often seen in the heel area. Typically, these wounds appear as dusky, boggy, or discolored areas of purple ecchymosis (Fig. 13-7). Sometimes, they appear a few days after surgery as a discolored area on the sacrum and may be misidentified as a burn.[105] Often, these areas deteriorate rapidly from intact skin to deep open wounds.

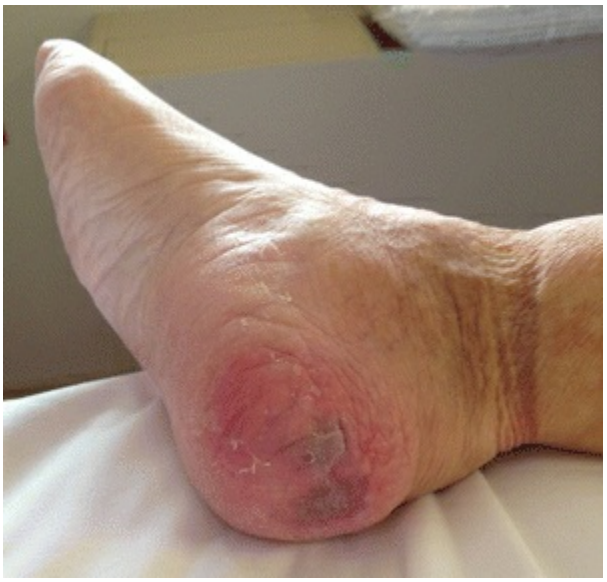

Figure 13-7. Suspected deep tissue injury. © J. M. Levine, MD

What is sDTI?[19,106] Can these pressure ulcers be prevented and, if so, how? What is the best treatment? Clinicians have sought guidance about this particular type of pressure ulcer. Black reports the finding of Baharestani that 36% of her sDTI cases resolved and 90% of these patients had anemia.[106] Initially, sDTI lesions have the appearance of a deep bruise and may herald the subsequent development of a stage III or IV pressure ulcer even with optimal management. Honaker, Brockopp, and Moe have described precipitating events to sDTI, as well as a research tool, which evaluates the severity of deep tissue injury.[107,108]

Early Detection of sDTI

The pressure ulcer staging system relies on visual inspection of the skin. Newer technologies may hold the promise of early detection for pressure ulcer injury *before* visible signs of tissue destruction can be seen. One study reported on the preulcerative changes in long-term residents using diagnostic ultrasound.[109]

Pressure Ulcer Treatment

Avoidable Pressure Ulcers

As a result of an international consensus conference in February 2010 and then again in 2014, NPUAP issued a revised definition of unavoidable pressure ulcers that was applicable across all care settings.[110,111] It is based on the CMS definition of "unavoidable" from Long-Term Care F-Tag 314.[10]

Practice Point

"Unavoidable PrU may occur even though providers have evaluated the individual's clinical condition and PrU risk factors have been evaluated and defined and interventions have been implemented that are consistent with individuals needs, goals, and recognized standards of practice. Further, an unavoidable PrU was characterized as one that occurs even though providers have monitored and evaluated the impact of preventive interventions and revised these approaches as appropriate."[111]

Although the debate continues as to whether all pressure ulcers are avoidable,[110–112] consensus was reached on 15 items at the 2014 NPUAP conference. Several guidelines are now available for clinicians to use in planning treatment.[1–10,44–47] Clinical guidelines provide the foundation for establishing evidence-based pressure ulcer management.

The wound bed preparation model has been applied to pressure ulcer prevention and treatment (Fig. 13-8).[113] A pressure ulcer won't heal unless the underlying causes are effectively managed. A general assessment should include identifying and effectively managing the patient's medical diseases, health problems (such as urinary incontinence), nutritional status, individual concerns (such as pain level),[114] and psychosocial health. Unless these major areas are effectively addressed, the probability of the pressure ulcer healing is unlikely.

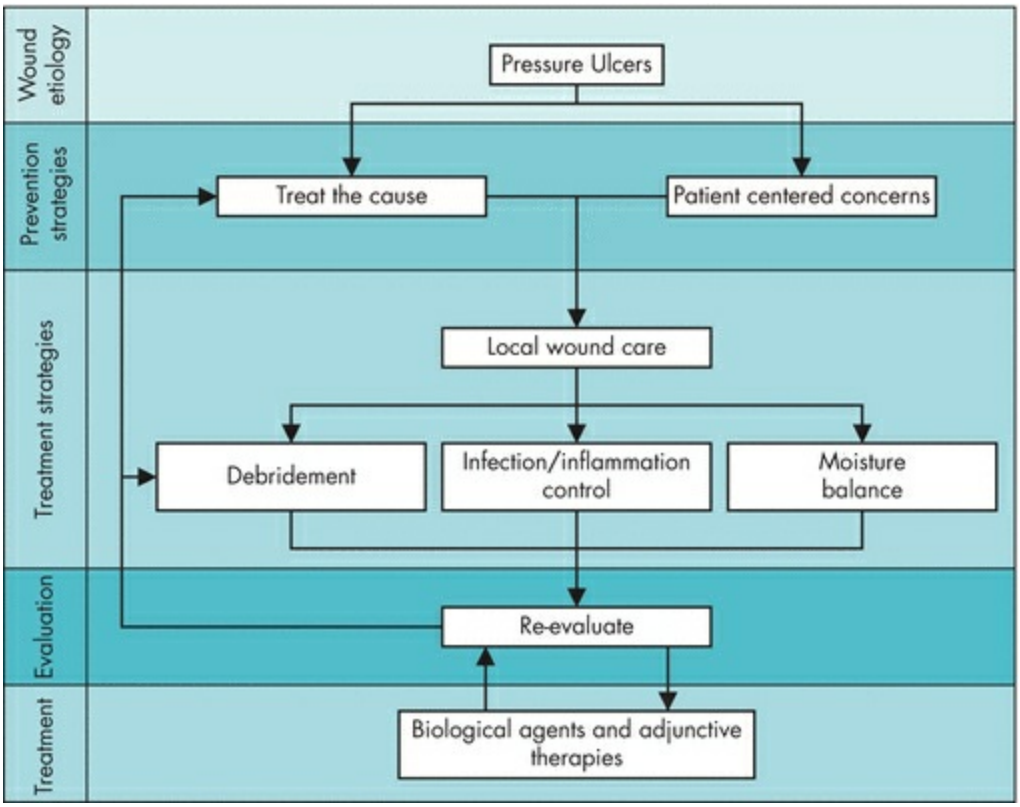

Figure 13-8. Pressure ulcer treatment and prevention using the wound bed preparation model. (From Dr. David Keast. Used with permission.)

The comprehensive local management of pressure ulcers includes cleaning, controlling infections, debridement, dressings that promote a moist wound environment (if a healable wound), nutritional support, and redistribution of pressure (repositioning and use of support surfaces). (See Chapter 11, Pressure Redistribution: Seating, Positioning, and Support Surfaces.) The use of adjunctive therapies to heal pressure ulcers should be considered for recalcitrant ulcers. Options are discussed later in the chapter.

Practice Point

Unstageable pressure ulcers are those covered with necrotic (slough and eschar) tissue. Until the necrotic tissue is removed, the ulcer can't be visualized accurately and is therefore unstageable in patients in all care settings.

Monitoring Healing

Because reverse staging of pressure ulcers to monitor healing is inappropriate, several instruments have been developed and validated to assess the healing of pressure ulcers.

Tools That Measure Healing

The two most widely used tools to measure healing in pressure ulcers are the Bates-Jensen Wound Assessment Tool (BWAT)[115] and the Pressure Ulcer Scale for Healing (PUSH).[116] The BWAT is comprised of 13 variables that provide a numerical indicator of the status of the pressure ulcer (healing or deteriorating).[115] The score ranges from 1, which indicates tissue health (or healed), to 65, which indicates wound degeneration. The variables that comprise the BWAT score include wound size (length and width), depth, edges, undermining, necrotic tissue type, necrotic tissue amount, exudate type, exudate amount, skin color of surrounding wound, peripheral tissue edema, peripheral tissue induration, granulation tissue, and epithelialization. The BWAT provides a comprehensive assessment of pressure ulcers[115] and is currently being evaluated for use with other wound types.

The PUSH tool[116,117] uses only three variables—surface area (length and width), exudate amount, and tissue appearance—to derive a numerical indicator of the status of the pressure ulcer (Box 13-5). A score of 0 indicates that the pressure ulcer has healed, while the highest score of 17 indicates wound degeneration. The PUSH tool intentionally takes a "minimalist approach." Using research databases in its development, the PUSH tool seeks to select the minimum number of assessment parameters needed to monitor ulcer healing or deterioration. Its brevity and accuracy in monitoring make the PUSH tool ideal for quality assurance monitoring of large groups of patients and identifying patients who are deteriorating and require reassessment and possibly treatment changes. The PUSH tool isn't intended to provide a comprehensive assessment of pressure ulcers and hasn't yet been validated for other types of wounds, although it is being tested in research studies and clinical use. A survey of 103 respondents found the PUSH tool reliable and easy to use.[118]

Box 13-5 NPUAP PUSH Tool

PATIENT NAME: PATIENT ID. No.

ULCER LOCATION: DATE:

Directions

Observe and measure the pressure ulcer. Categorize the ulcer with respect to surface area, exudate, and type of wound tissue. Record a subscore for each of the ulcer characteristics. Add the subscores to obtain the total score. A comparison of total scores measured over time provides an indication of the improvement or deterioration in pressure ulcer healing.

LENGTH	**0** 0 cm^2	**1** <3 cm^2	**2** 0.3–0.6 cm^2	**3** 0.7–1.0 cm^2	**4** 1.1–2.0 cm^2	**5** 2.1–3.0cm^2	**Blank**
× WIDTH		**6** 3.1–4.0 cm^2	**7** 4.1–8.0 cm^2	**8** 8.1–12.0 cm^2	**9** 12.1–24.0cm^2	**10** >24.0 cm^2	**Subscore**
EXUDATE AMOUNT	**0** None	**1** Light	**2** Moderate	**3** Heavy		**Subscore**	
TISSUE TYPE	**0** Closed	**1** Epithelial tissue	**2** Granulation tissue	**3** Slough	**4** Necrotic tissue		**Subscore**
							Total score

LENGTH × WIDTH

Measure the greatest length (head to toe) and the greatest width (side to side) using a centimeter ruler. Multiply these two measurements (length× width) to obtain an estimate of surface area in square centimeters (cm^2). Do not guess! Always use a centimeter ruler and always use the same method each time the ulcer is measured.

EXUDATE AMOUNT

Estimate the amount of exudate (drainage) present after removal of the dressing and before applying any topical agent to the ulcer. Estimate the exudate as none, light, moderate, or heavy.

TISSUE TYPE

This refers to the types of tissue that are present in the wound (ulcer) bed. Score as a "4" if any necrotic tissue is present. Score as a "3" if any amount of slough is present and necrotic tissue is absent. Score as a "2" if the wound is clean and contains granulation tissue. Score as a "1" if the wound is superficial and re-epithelializing. Score as a "0" if the wound is closed.

4–Necrotic tissue (eschar): Black, brown, or tan tissue that adheres firmly to the wound bed or ulcer edges and may be either firmer or softer than surrounding tissue.

3–Slough: Yellow or white tissue that adheres to the ulcer bed in strings or thick clumps or is mucinous.

2–Granulation tissue: Pink or beefy-red tissue with a shiny, moist, granular appearance.

1–Epithelial tissue: For superficial ulcers, new pink or shiny tissue (skin) that grows in from the edges or as islands on the ulcer surface.

0–Closed/resurfaced: The wound is completely covered with epithelium (new skin).

Although tools exist for measuring pressure ulcer healing, more evidence is needed to determine pressure ulcer healing rates.[119–122] Among chronic wounds, pressure ulcers have the slowest healing rate—0.077 mm/day, with 2.156 mm of healing expected at 4 weeks.[121] The use of high-frequency portable ultrasound to measure wound healing has been

introduced. The use of this technology, which can capture three-dimensional measurements, has been shown to be quite beneficial in objectively monitoring healing. Ultrasound is also "color blind"; that is, it can detect stage I pressure ulcers in darkly pigmented skin.[123]

Principles of Local Wound Care

After addressing the cause and patient-centered concerns, healing a pressure ulcer using the wound bed preparation model requires attention to determining the wound prognosis (healable, maintenance, or nonhealable wound)[124] and applying the principles of local wound care (debridement, infection management, moisture balance before attention to the edge).

Cleansing

Cleaning the pressure ulcer to remove devitalized tissue and decrease bacterial burden is often recommended. NPUAP/EPUAP/PPPIA also recommends cleansing the surrounding skin.[1] Pressure ulcers can exhibit delayed healing in the presence of high levels of bacteria.[125] Solutions that don't traumatize the healing ulcer should be used.[1,126,127] Normal saline solution (0.9%) is usually recommended because it isn't cytotoxic to healthy tissue.[1,4] Although the active ingredients in newer wound cleaners may be noncytotoxic (surfactants), the inert carrier may be cytotoxic to healthy granulation tissue. Review of all ingredients is warranted. Hellewell et al.[128] found that antiseptic cleaners were the most cytotoxic to granulation tissue. An in vitro study found relative toxicity indexes ranging from 0 to 100,000 with saline and Shur-Clens to be least toxic to fibroblasts, while Dial antibacterial soap and Ivory liquid gel were the most toxic.[129] Least toxic to keratinocytes were Biolex, Shur-Clens, and Techni-Care, while hydrogen peroxide, modified Dakin's solution, and povidone (10%) were most toxic.[129]

The mechanical method used to deliver the cleaning agent must provide enough pressure to remove debris without presenting trauma to the ulcer bed.[1] Optimal pressure to clean an ulcer is between 4 and 15 pounds per square inch (psi).[130] A 35-mL syringe with a 19-gauge needle creates an 8-psi irrigation pressure stream,[1] which was found to be more effective in removing bacteria than other irrigation pressures.[131] It should be noted that irrigation pressures exceeding 15 psi can cause trauma to the ulcer bed and

may drive bacteria into the tissue.[132] New technology such as battery-powered, disposable irrigation devices can provide an alternative to the syringe and needle system to loosen wound debris.

Debridement

The presence of necrotic devitalized tissue promotes the growth of pathological organisms and prevents wounds from healing.[133] Debridement is, therefore, a very important step in the local management of pressure ulcers. There's no optimal debridement method; selection should be based on the goals of the patient, absence or presence of infection, amount of necrotic tissue present, and economic considerations for the patient and the facility.

Many types of debridement, including surgical, autolytic, enzymatic, mechanical, biological, and laser, are available. (See Chapter 8, Wound debridement.) However, surgical (or sharp laser) debridement is considered by many to be the most effective form of debridement because it involves the cutting away (with a scalpel) of necrotic tissue.[134] In addition, surgical debridement is relatively quick and can be done at the bedside. Surgical debridement is essential when cellulitis or sepsis is suspected.[135] Autolytic debridement involves the use of a semiocclusive or occlusive dressing (e.g., hydrocolloids, hydrogels) and employs the body's own natural enzymes to digest necrotic tissue. Autolytic debridement takes much longer than sharp debridement. Watch closely for signs and symptoms of infection. Enzymatic debridement uses proteolytic enzymes to remove necrotic tissue. In the United States, papain, urea, and trypsin are no longer available for use; only collagenase can be used for enzymatic debridement. Mechanical debridement uses wet-to-dry gauze to adhere to the necrotic tissue, which is then removed. Upon removal of the gauze dressing, necrotic tissue and wound debris are removed. However, healthy granulation tissue is also removed, which can delay wound healing.[134]

Pressure Redistribution

The use of support surfaces is an important consideration in redistributing pressure. (See Chapter 11, Pressure Redistribution: Seating, Positioning, and Support Surfaces.) The NPUAP Support Surface Standards Initiative redefined the physical concepts related to support surfaces.[79]

Few studies demonstrate significant differences within the support

surface classifications and preventing or healing pressure ulcers. Therefore, the level and type of risk factors should guide the level and type of support surface selected. The NPUAP/EPUAP/PPPIA clinical practice guideline recommends that a patient should not be positioned directly on a pressure ulcer and that an individualized turn and reposition schedule be established based on the characteristics of the support surface, the person's response, and the clinical goals.[1]

Dressings

The use of moist wound therapy dressings is a major component in managing a healable pressure ulcer. (See Chapter 9, Wound Treatment Options.) At present, it's conservatively estimated that thousands of different dressings are available for pressure ulcer management. Dressings can be broken down into several classifications: gauze, nonadherent gauze, transparent films, hydrocolloids, foams, alginates, hydrogels, collagens, antimicrobials, composites, and combinations. Matching the dressing to the wound bed characteristics is essential. A guiding principle is to maintain a moist environment for a healable wound.

Although nongauze dressings are usually more expensive than gauze dressing, less frequent dressing changes, faster healing rates, and decreased rates of infection can make non–gauze-based dressing more cost-effective over time.[136–139] It's also important to note that wet-to-dry gauze dressings are a form of debridement and shouldn't be used on ulcers with good granulation tissue. CMS specifically states that use of wet-to-dry dressings should be limited in long-term care patients.[10] No specific dressing heals all pressure ulcers within an ulcer classification. Consequently, a careful assessment of the pressure ulcer, the patient's needs, and environmental factors (frequency of dressing changes to increase adherence) must be considered. (See Chapter 9, Wound Treatment Options.)

Nutrition

Nutrition is important to maintain the body in positive nitrogen balance,[1] thereby increasing wound healing. Patients should be assessed and screened for nutritional status, including weight, weight history (e.g., significant weight loss), and adequacy of nutrient and fluid intake.[1] It's important to provide protein (30 to 35 calories/kg body weight) for malnourished patients with pressure ulcers.[1] Always check renal status to make sure that

the patient's kidneys can handle the protein load.[1] Although dietary supplementation of vitamins and minerals in the absence of deficiency remains controversial,[140–143] the NPUAP/EPUAP/PPPIA recommends "vitamin and mineral supplements when dietary intake is poor or deficiencies are confirmed or suspected (Strength of Evidence = B strength of recommendation )."[1] The use of enteral and parental nutritional support should always be considered when the patient is unable to meet caloric needs.[1]

Control of Infections

All pressure ulcers will become colonized with both aerobic and anaerobic bacteria; therefore, pressure ulcers aren't sterile wounds. (See Chapter 7, Wound, bioburden, and infection.) Avoid swab-culturing the surface of a pressure ulcer to diagnosis infection. Clean technique is customarily used when treating pressure ulcers. If an ulcer may be infected (independent of a puncture biopsy), most experts assess for the amount of drainage and odor and examine the surrounding tissue for cellulitis. It should be noted that some infected ulcers may not demonstrate the typical signs and symptoms associated with infection; rather, they may appear as nonhealing ulcers.

A pressure ulcer will not heal until infection is controlled. The use of topical agents such as silver sulfadiazine (Silvadene) and oral antibiotics for a 1- or 2-week period may be useful. Nonhealable or infected wounds may benefit from short-term use of 1% povidone–iodine. Clearly, additional research is needed to examine the role of topical antibiotics in decreasing bacterial loads in pressure ulcers. Systemic antibiotics should only be used when a systemic infection is suspected.

Adjunctive Therapies

The use of adjunctive therapies is the fastest-growing area in pressure ulcer management. Adjunctive therapies include electrical stimulation, hyperbaric oxygen, radiant heat, growth factors, and skin equivalents. Except for electrical stimulation, published research substantiating the effectiveness of adjunctive therapies in healing pressure ulcers is scarce.

The NPUAP/EPUAP/PPPIA clinical practice guideline recommendation on electrical stimulation is at the A level of evidence.[1] Electrical stimulation is the use of electrical current to stimulate a number of cellular processes, such as increasing fibroblasts, neutrophils, macrophages,

collagen synthesis, and DNA synthesis and increasing the number of receptor sites for specific growth factors.[144] Electrical stimulation appears to be most effective on stage III and IV pressure ulcers[1] that are unresponsive to traditional methods of healing. Although there are much data to suggest that electrical stimulation is effective in healing pressure ulcers, the optimal electrical charge needed to stimulate pressure ulcer healing remains unclear. The literature suggests that an optimal electrical charge of 300 to 500 uA/second produces positive effects on the pressure ulcer.[145] However, additional research is needed to determine the optimal electrical charge based on the characteristics of pressure ulcers (e.g., stage, depth, and amount of drainage).

Hyperbaric oxygen is believed to promote wound healing by stimulating fibroblasts, collagen synthesis, epithelialization, and control of infection.[146] However, controlled clinical studies couldn't be found regarding the association of hyperbaric oxygen and the healing of pressure ulcers. The limited literature that does exist suggests that topical hyperbaric oxygen doesn't increase tissue oxygenation beyond the superficial dermis.[147] NPUAP/EPUAP concluded that there was insufficient evidence to recommend hyperbaric or topical oxygen therapy for the treatment of pressure ulcers.[1]

Growth factors and skin equivalents are emerging methods of healing pressure ulcers. The use of cytokine growth factors (e.g., recombinant human platelet-derived growth factor-BB [rhPDGF-BB]), basic fibroblast growth factor (bFGF), and skin equivalents are currently being studied. Only one multicenter, randomized, double-blind study examining the use of rhPDGF-BB was found.[148] This study enrolled 45 patients with stage III or IV pressure ulcers who were randomized to treatment group 1 (300 µg/mL of rhPDGF), treatment group 2 (100 µg/mL rhPDGF), or treatment group 3 (placebo). After 4 weeks of treatment, patients in group 1 had a 40% reduction in ulcer area, group 2 had a 71% reduction in ulcer area, and group 3 had a 17% reduction in pressure ulcer area. From these results, it is clear that the use of growth factors may have a crucial impact on the future of wound healing. However, additional research is needed to evaluate the efficacy of specific growth factors in healing pressure ulcers. The NPUAP/EPUAP/PPPIA Clinical Practice Guideline notes that PDGF-BB may improve healing of pressure ulcers, but because the available evidence is insufficient, the guideline does not recommend this treatment for routine use.[1]

Prevalence and Incidence

It has been said, "what can be measured can be managed." To improve pressure ulcer care, the number of patients with pressure ulcers must be accurately determined. Doing so will require careful attention to prevalence and incidence data. The data represent the percentage of patients with pressure ulcers among all those surveyed in a setting (prevalence) and the percentage of patients who developed pressure ulcers after admission to the setting (incidence).

In 1989, at its first consensus conference, NPUAP brought attention to the pressure ulcer problem in the United States by reporting prevalence and incidence data.[83] The group set a national goal to reduce the incidence of pressure ulcers by 50% by the year 2000.[83] During the next decade, NPUAP engaged in an active program to improve pressure ulcer practice through education, research, and public policy.

At the close of the 20th century, NPUAP assessed the progress toward this goal through its Pressure Ulcers Challenge 2000 project. This 2-year project included a Medline database search for all articles on pressure ulcer incidence and prevalence published and indexed between January 1, 1990, and December 31, 2000. More than 300 studies were found. Pressure ulcer incidence and prevalence data were analyzed across care settings and in specific populations, such as people with spinal cord injuries, elderly patients, infants and children, patients with hip fractures, people of color, and those at the end of life receiving palliative or hospice care.[149]

Study data presented in the NPUAP/EPUAP/PPPIA guideline reveal a wide variation in the range of incidence rates from 2000 to 2012 (acute care, 0% to 12%; aged care, 1.9% to 59%; critical care, 3.3% to 53.5%; operating room 5% to 53.4%).[1,149] Prevalence rates from 2000 to 2012 ranged from 0% to 46% in acute care,[1] 4.1% to 32.2% in aged care, and 13.1 to 45.5 in critical care.[1] Inconsistencies in the methodologies used and in the populations studied contribute to these differences and make comparisons and analyses of trends problematic.[149,150] However, many positive developments in the prevention and treatment of pressure ulcers have occurred over the past decade, including development of evidence-based practice guidelines, standardization of risk assessment, and improved technologies for prevention and treatment.[149,150]

Most pressure ulcers, regardless of setting, are partial-thickness (stages I and II) and are located on the sacrum or coccyx.[151] Heels are the second

most common location. The proportion of sDTI pressure ulcers has increased threefold to 9% in 2009.[151] The heels (41%), sacrum (19%), and buttocks (13%) are the locations for 73% of all sDTI ulcers. This is in a different order than the most frequent locations for all pressure ulcers (sacrum, heels, etc.).[151]

Prevalence and Incidence Definitions and Formulas

Lack of clarity and consistency in definitions and calculation formulas impedes our understanding of pressure ulcer prevalence and incidence. Standardization of definitions and formulas will enhance comparability of data among future studies. NPUAP recommends the adoption of consistent definitions and formulas for determining pressure ulcer prevalence and incidence.[149,152]

NPUAP/EPUAP/PPPIA suggests that prevalence should be defined as "the proportion/percentage of individuals in a defined population who have a pressure ulcer at a specified point in time.[1] In assessing prevalence, it doesn't matter in what setting the pressure ulcer was acquired."[149,152] Suggested standard formulas for obtaining prevalence are[153]

- Pressure ulcer *point* prevalence (%)

$$\frac{\text{Number of patients with pressure ulcer at a specific point in time} \times 100}{\text{Total number of patients in the study population at a specific point in time}}$$

- Pressure ulcer *period* prevalence is the number of individuals who have a pressure ulcer at a specified period of time (usually days or weeks).

$$\frac{\text{Number of patients with pressure ulcer at a specific point in time} \times 100}{\text{Number of patients in a population during a particular time period}}$$

NPUAP/EPUAP/PPPIA recommends using the following definition of incidence: "the proportion of pressure ulcer free individuals that develop a pressure ulcer over a specific period of time."[1,54] Several approaches to

measuring incidence have been used. NPUAP defines cumulative incidence as a percentage. It is the number of patients developing new pressure ulcer during a specific time period multiplied by 100, over the total number of patients in a study population over a specific time period[1]. The formula is as follows:

Pressure ulcer cumulative incidence

$$\frac{\text{number of patients with new pressure ulcer during a specific time period} \times 100}{\text{total number of patients in a study population over a specific time period}}$$

A problem with using this approach is that it doesn't count pressure ulcers that occur in people admitted to the setting after the study population has been defined. Therefore, it may not be indicative of the true incidence of new ulcers in that setting.

Another way to calculate prevalence is to measure the number of new cases of pressure ulcers that occur in a changing population. In this case, the people who are being studied have varying lengths of stay. Incidence is calculated as the number of people developing pressure ulcers per 1,000 patient-days and is called *incidence density*. Calculate this by using the following suggested formula[153]:

- Pressure ulcer incidence density

$$\frac{\text{Number of patients developing a new pressure ulcer} \times 1{,}000}{\text{Total patient-days free of pressure ulcers}} = \frac{\text{Number of patients developing a pressure ulcer}}{\text{1,000 patient-days}}$$

Using the NPUAP-recommended standard formulas alone may not be enough to avoid errors in prevalence and incidence calculations (Box 13-6).

Box 13-6 Pitfalls to Calculating Prevalence and Incidence

Be sure to avoid the following pitfalls when calculating pressure ulcer prevalence and incidence for your facility.

- Define the population and apply the definition consistently

throughout the study.

- Count the number of patients with pressure ulcers—not the number of pressure ulcers.
- Count only pressure ulcers, not other wounds.
- Define the stages of the pressure ulcers you count to include and assess them accurately.

Adapted with permission from Ayello, E.A., et al. "Methods for Determining Pressure Ulcer Prevalence and incidence," in J. Cuddigan, et al., eds. *Pressure Ulcers in America: Prevalence, Incidence, and Implications for the Future*. Reston, VA: National Pressure Ulcer Advisory Panel, 2001.

Operating Room

Since CMS stopped reimbursing hospitals at a higher rate for pressure ulcers that occur during hospitalization, much attention has been placed on reducing the incidence of pressure ulcers during the perioperative experience. Elements of a pressure ulcer prevention protocol are adapted for the special needs of perioperative patients including using additional support surfaces such as facial pads to off-load pressure points on the face and other parts of the body when the patient is in the prone position.[1] One hospital was able to reduce pressure ulcers in their surgical patients from 7.3% to 1.3%.[154] This was accomplished by educating the operative staff and using a special colored risk band to identify patients with longer operating times as being at risk for pressure ulcers.

Competencies and Curriculum

Accurate and current knowledge is essential for clinicians to prevent and treat pressure ulcers. Pressure ulcer knowledge varies among members of the wound care team. For example, physicians and residents in three studies were found to have low levels of pressure ulcer knowledge.[155–157] In addition, some nurses believe that their basic education is insufficient regarding wound care.[97–101] What's more, high pressure ulcer prevalence rates have been linked to poor knowledge.[158–161] Some of the first initiatives to decrease pressure ulcers by increasing the knowledge level of clinicians include the IHI[42] and the New Jersey Hospital Association's "No Ulcers" project.[161]

As knowledge about pressure ulcers has increased, their occurrence has decreased across care settings.[161] AHRQ has pilot tested a pressure ulcer tool kit to assist hospitals in decreasing the incidence of pressure ulcer.[162] Certification has made a difference in pressure ulcer knowledge. In one study, nurses who had received any wound care certification from the WOCN Society, the American Academy of Wound Management, or the National Alliance of Wound Care had higher scores on a standardized 47-item pressure ulcer test.[163]

Suggestions to improve both pressure ulcer prevention practice and the characteristics of pressure ulcer incidence reduction initiatives have been reported in the literature.[164–166] For example, after introduction of the Canadian Association of Wound Care Pressure Ulcer Awareness Program, the pressure ulcer prevalence and incidence rates in Canada decreased by 57% and 71%, respectively.[167] In long-term care, Horn and colleagues achieved a 33% reduction in pressure ulcers in seven facilities by implementing a real-time optimal care plan for nursing home quality initiative in which the number of documentation forms was reduced from 6.2 to 2.8.[80] Citing the limited literature on pressure ulcer injury in patients undergoing procedures in diagnostic and interventional ancillary units such as radiology, renal dialysis, and cardiac and vascular procedure laboratories, Messer[168] stresses the importance of focusing on prevention in these areas. The NPUAP has an updated competency-based curriculum on pressure ulcer prevention[169–171] for registered nurses on its Web site (http://www.npuap.org) (Box 13-7).

Box 13-7 NPUAP Registered Nursing Competency Base Curriculum: Pressure Ulcer Prevention

- Identify etiologic factors contributing to pressure ulcer occurrence.
- Conduct a structured risk assessment on admission, and repeat regularly and as frequently as required by patient acuity and setting.
- Ensure that a complete skin assessment is part of the risk assessment screening policy in place in all healthcare

settings.

- Develop and implement an individualized program of skin care.
- Demonstrate proper positioning/repositioning for pressure ulcer prevention/treatment.
- Choose appropriate support surface for a patient based on risk and the patient's attributes.
- Implement nutritional interventions as appropriate to prevent pressure ulcers.
- Accurately document results of risk assessment, skin assessment, and prevention strategies.
- Apply critical thinking skills to clinical decision making regarding the impact of changes in the individual's condition on pressure ulcer risk.
- Make referrals to other healthcare professionals based on client assessment.

Pieper, B. "National Pressure Ulcer Advisory Panel Registered Nurse Competency-based Curriculum: Pressure Ulcer Prevention."

Building knowledge about pressure ulcer care is vital. While many experts believe that all pressure ulcers cannot be prevented,[111,172] it's been shown that education can reduce pressure ulcer incidence and expedite treatment.[173] A variety of continuing education programs, symposia, national conferences, as well as company-sponsored online learning programs exist to facilitate this knowledge building. Interactive computer-based testing by the EPUAP-endorsed ePUCLAS$_2$ pressure ulcer staging module[102] is just one of the resources available. The John A. Hartford Institute for Geriatric Nursing also has several one-page quick references in its "Try This" series, one of which is using the Braden Scale.[174] The NDNQI offers four online modules covering several aspects of pressure ulcer care in which clinicians can acquire knowledge as well as measure their learning through interactive testing.[103] With all this information available, the challenge for clinicians is to put this knowledge into practice in order to prevent and/or treat pressure ulcers.

Summary

Pressure ulcers are a common healthcare problem throughout the world. Intensity and duration of pressure as well as tissue tolerance are the etiologic factors that lead to pressure ulcer development. Incorporation of clinical practice guidelines provides a basis for evidence-based pressure ulcer prevention and treatment practice. Results of a pressure ulcer risk assessment using a validated tool can serve as the foundation for developing a pressure ulcer prevention protocol. After determining the pressure ulcer stage and other wound characteristics, a comprehensive plan to treat the pressure ulcer that uses a combination of local wound care, debridement, moist wound healing, cleaning, and pressure relief needs to be implemented. A multidisciplinary approach to patient care that includes patient and family education as well as staff education is essential. Use of the standardized formulas as proposed by NPUAP will provide a basis for universal comparison of prevalence and incidence data. Many educational resources are available to clinicians to increase their knowledge level so as to decrease pressure ulcer incidence and enhance treatment.

Patient Scenario

Clinical Data

Mrs. GK is an 82-year-old woman who fractured her left hip. Following open reduction and internal fixation of the fracture, she was noncompliant with ambulation during her hospitalization and developed a sacral stage II pressure ulcer as well as pneumonia. She is now being cared for in her son's home with the assistance of home care services. Her prior dietary intake was inadequate as she had lost 7 pound in 4 weeks while hospitalized. She nibbles at her food and eats less than half of her meals. Mrs. GK is able to respond to commands. She is very reluctant to get up and walk as her "bottom is painful," so she sits in her bed, which has a standard mattress, most of the day. She wears adult incontinence briefs because it is too difficult to get her to the commode, so her skin in the pelvic area is moist. The skin on her extremities is pale, thin, and dry.

Case Discussion

Mrs. GK has several risk factors for developing pressure ulcers, including her age, poor dietary intake, recent weight loss, immobility and inactivity, and moist skin from incontinence. Her plan of care

included consultation with a dietician to review innovative ways of getting protein and nutrients into her diet. Protein supplements were added to the foods she likes to ingest (coffee, soups, and puddings).

She was referred to a certified wound and incontinence nurse for management of her incontinence, pressure ulcer, and skin care needs. The use of incontinence pads was discontinued and alternative strategies were taught to her daughter. Because moist skin needs less pressure to break down and Mrs. GK is immobile, a pressure redistribution mattress and chair cushion were acquired for use on her bed and chair, respectively. Mrs. GK's son was educated about the importance of frequent repositioning, including the use of pillows and why support surfaces should be used to promote healing and prevent further tissue insult. A schedule was developed to aid in implementing the home care strategies for Mrs. GK. Appropriate wound dressings were placed to support healing of the stage II pressure ulcer. Physical and occupational therapists were consulted for increased ambulation and muscle strengthening exercises.

Show What You Know

1. A pressure ulcer is a lesion caused by:

A. incontinence.
B. unrelieved pressure.
C. heat.
D. diabetes mellitus.

2. A patient is dragged across the bed when transferring to a stretcher. Which one of the following forces that contribute to pressure ulcer development has occurred?

A. Electrical stimulation
B. Shear
C. Friction
D. Maceration

3. A patient has a 2- × 3-cm sacral pressure ulcer that has some depth and extends into the subcutaneous tissue with some undermining; no bone is palpable or visible. There's a small amount of slough seen in one corner of the wound. Using the NPUAP staging classification system,

this pressure ulcer is:

A. stage I.
B. stage II.
C. stage III.
D. stage IV.

4. Which of the following Braden Scale scores for an elderly black man would indicate pressure ulcer risk?

A. 23
B. 21
C. 19
D. 17

5. Which one of the following should be included in a care plan to prevent pressure ulcers?

A. Turn and reposition every 5 hours.
B. Clean skin daily using hot water and soap.
C. Encourage the patient who's confined to a chair to relieve pressure every hour.
D. Limit fluids to 10 mL/kg of body weight daily.

6. Which one of the following parameters is *not* part of the NPUAP PUSH tool?

A. Depth
B. Exudate
C. Tissue type
D. Length × width

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Venous Disease and Lymphedema Management

14

Objectives

After completing this section of the chapter, you will be able to:

- describe the anatomy and physiology of the venous system
- describe the pathophysiology of lower-extremity venous ulcers
- explain the physiology of edema formation
- describe systems for classifying venous disease
- state the signs and symptoms that constitute a venous assessment
- discuss vascular laboratory tests performed for patients with venous disease
- describe the components of local wound care for a patient with a venous ulcer
- describe surgical treatment for patients with venous ulcers
- identify education needs for patients with venous disease.

VENOUS DISEASE

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"Peripheral vascular disease" is a term commonly used in reference to an arterial problem, even though it includes diseases and conditions of the venous and lymphatic systems as well as the arterial system. Patients with leg ulcers may present with a combination of arterial, venous, and lymphatic disease. This chapter presents information in two separate sections: (1) the pathogenesis and management of wounds resulting from venous insufficiency and (2) lymphatic disease.

Scope of the Problem

Up to 50% of individuals may have some sort of vein abnormality.[1] It is estimated that between 1% and 22% of the population over age 60 suffers from lower-extremity skin ulcers.[2–5] One study found the problem to be underestimated when a self-report survey indicated that high numbers of patients cared for their own ulcers without consulting a healthcare provider.[6] The principal leg ulcer etiology in most patients is some type of peripheral vascular disease. Chronic venous disease is the seventh most common chronic disease and is the underlying cause in 95% of leg ulcers.[1,7–9] In a US community health survey, 5% of adults had skin changes in the leg and more than 500,000 suffered from venous ulcers. Over 2,000,000 workdays are lost in the United States per year due to the associated morbidity of postphlebitic syndrome.[9] Although it's understood that chronic wounds have physical, financial, and psychological effects, it's difficult to measure these effects on a patient's quality of life.[10] It's also difficult to obtain accurate etiological information about leg ulcers, and in about one-third of medical records, no ulcer etiology might be documented.

Venous Anatomy and Physiology

Venous System

The venous system begins at the postcapillary level. Venules begin to coalesce, forming small veins, which again coalesce into larger veins from the periphery to a more central location. The venous system mimics the arterial system in many respects but has greater anatomic variability than the arterial tree. In the leg, the veins that course with the tibial and peroneal arteries are usually paired with numerous cross-linking branches, resulting in a retia appearance in some patients. These branches ascend along the respective arteries to form the popliteal vein, which is the first vein of significant size in the lower leg. The popliteal vein proceeds toward the head and becomes the femoral vein, commonly called the superficial femoral vein—a name that causes confusion because the vein in question is actually a deep vein. The superficial femoral vein joins the deep femoral vein to form the common femoral vein. The deep femoral vein is the deep drainage system of the thigh (Fig. 14-1).

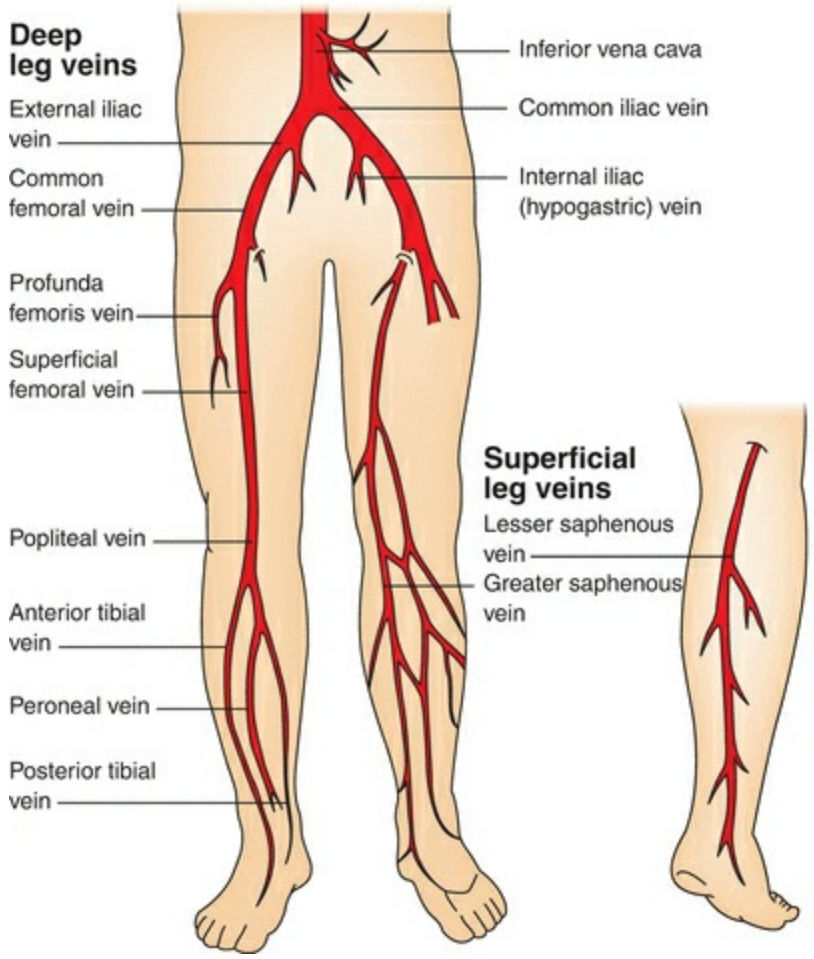


FIGURE 14-1. Deep and superficial venous systems. This illustration shows deep and superficial veins of the lower extremities.

Dual Venous System

The leg has a dual venous system—the deep system just described and the superficial system represented by the saphenous veins. The greater saphenous vein (GSV) courses along the medial aspect of the leg. The dorsal digital veins in the foot coalesce to form the GSV, which is found medial and anterior to the medial malleolus. It ascends in the leg through a variable course and may be bifurcated or even trifurcated. At knee level, its course becomes deeper in relation to the skin. As it ascends the leg, it joins the common femoral vein at the fossa ovale. The lesser saphenous vein drains the posterior aspect of the calf. It perforates into the deep compartment of the calf at the level of the popliteal fossa to join the popliteal vein. As the common femoral vein ascends behind the inguinal ligament, it becomes the external iliac vein and joins the internal iliac vein to become the common iliac vein. The common iliac veins join at the level

of the umbilicus and to the right of the aorta to become the inferior vena cava. The renal veins drain into the vena cava. More cephalad, the hepatic veins join the vena cava, which then empties into the right heart chamber.

The saphenous system is connected to the deep venous system through numerous perforator veins. Perforator veins shunt blood from the subcutaneous tissue and the greater saphenous system into the deep veins of the leg. They cross through the superficial fascia of the leg, hence their name. The location of perforator veins is somewhat variable, and some are ascribed proper names. The lowest perforator connecting the saphenous system with the deep venous system is just above the medial malleolus.

Valve Anatomy

Unidirectional valves are present in the deep and superficial venous systems and in the perforator veins. These valves are located just before bifurcation points. The GSV contains approximately six to eight valves. With rare exception, a valve is always present just below the insertion of the GSV into the common femoral vein at the fossa ovale. The orientation of the valves allows venous blood to flow from distal to proximal. Perforator veins valves are oriented to shunt blood from the lesser saphenous vein and the greater saphenous system into the deep veins of the leg.

Valve anatomy is that of a bileaflet with valve sinuses present on the lateral bases of each valve leaflet. These sinuses represent a dilation in the normal contour of the vein wall. Their function is to assist in valve closure, a passive act caused by the retrograde flow of venous blood into the sinus, thereby coapting (fitting together) the two valve leaflets. The valve leaflets are oriented parallel to the surface of the skin. It's the loss of valve function at various levels that results in varying degrees of venous insufficiency. Valve function is lost under a number of disease states. Inability of the valve to coapt can also occur with overdistention of the venous segment. This effectively stretches the valves apart so that they no longer come in direct contact, thereby allowing blood to reflux into the more dependent portion of the vein. Disease states that cause loss of valve function include:

- congenital valve absence
- deep vein thrombosis
- ectasia
- phlebitis
- valve atresia
- venous engorgement

- venous hypertension.

Venous Wall Architecture

Venous wall anatomy is similar to arterial wall anatomy except that the respective laminae are thinner. The outermost layer of a vein is the adventitia. The media varies most from the arterial media. The media within a vein contains both elastic and muscular fibers, but to a much lesser degree than the arterial media. Nonetheless, a vein can contract and adjust its size to correspond to the degree of venous blood flow. The intima layer is a delicate single layer of endothelial cells.

The relatively thin media accounts for the lack of venous compliance at increased pressures. At low pressures, the venous system is fairly compliant, but once arterial pressure is reached, the venous wall becomes distended and rigid. Venoconstriction occurs in both the superficial and deep veins; the more peripheral the vein, the more readily it contracts. This reactivity is under sympathetic adrenergic control. Peripheral veins are more sensitive than central veins to this sympathetic drive. The ability of veins to relax and dilate enables the venous system to hold 75% of the total blood volume.

Practice Point

Loss of consciousness can occur from venous pooling in motionless lower extremities—a fate not unknown to young military recruits who must stand at attention for prolonged periods of time.

The upward flow of lower-extremity venous blood, although aided by unidirectional valves and arterial pressure, is mostly dependent upon the "muscular pump." Pedal dorsal vein pressure in the supine position should approximate that of central venous pressure. Upon assuming an erect posture, this pressure can approach 100 mm Hg. With active muscle contraction, intracompartmental pressure markedly increases, thereby causing deep veins of the leg to compress and push venous blood upward. This pressure then approaches 200 mm Hg. This is possible because the muscular compartments of the leg are enclosed by relatively rigid fascial encasement. Backflush of blood is reduced when valves are competent and

reflux into the saphenous system is prevented by the unidirectional perforator valves.

Ulcer Pathophysiology

Venous Ulcers

Venous ulcers are chronic skin and subcutaneous lesions usually found on the lower extremity at the pretibial and the medial supramalleolar areas of the ankle, where the perforator veins are located. Venous ulcers were formerly known as "venous stasis" ulcers because their development was thought to be caused by blood pooled in the veins. More recent literature indicates that venous hypertension rather than venous stasis is both the cause of these ulcers and the reason they don't heal.[11,12] It's difficult to restore skin integrity in the presence of chronic venous hypertension because the underlying edema must be controlled in addition to healing the ulcer.

Venous ulceration may be precipitated by deep vein thrombosis (DVT), which can remain undiagnosed for years prior to the onset of the ulcer (Fig. 14-2). It has long been thought that the natural course of lower-extremity DVT is the eventual development of leg ulcers.[13]

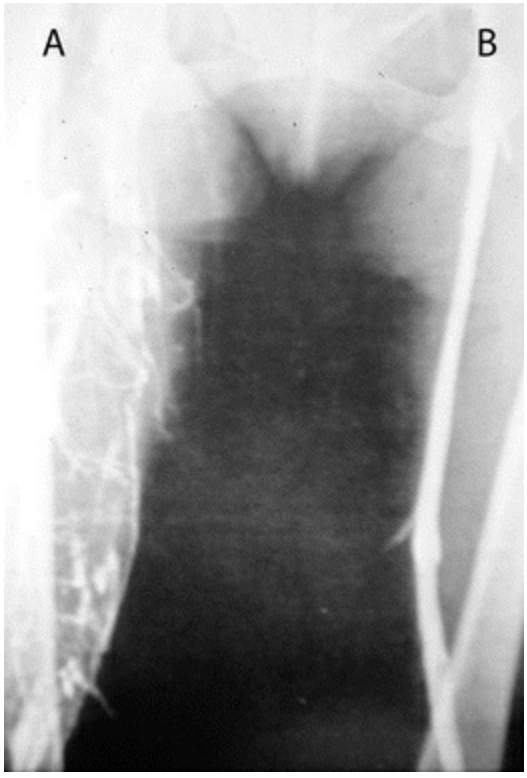


FIGURE 14-2. Venogram. In this venogram, the patient's left venous valve (**B**) is intact. On the patient's right (**A**), collateral veins are present due to venous occlusion, possibly from an undiagnosed deep vein thrombosis.

Symptomatic and asymptomatic thrombi may cause long-term complications by scarring the intima and creating valvular incompetence. When the valves are rendered incompetent, blood backs into the distal veins during diastole. With loss of perforator valve function, the high intracompartmental venous pressure, which can approach 200 mm Hg during active muscle contraction, results in distention of the saphenous system. This in turn causes a cascading effect with dilation of the GSV and worsening of already compromised valvular function. The weight of the column of blood increases the pressure inside the capillaries.

Characteristics of Venous Ulcers

Venous hypertension distends the superficial veins, resulting in vein wall damage and exudation of fluid into the interstitial space, thereby causing edema of venous insufficiency. Over time, an actual leakage of red blood cells occurs through these compromised veins. As they break down, the red blood cells deposit hemosiderin into the tissues, causing a form of "internal tattooing" of the skin; the coloration is that of a brownish hue noticeable even in black skin (Fig. 14-3). The skin loses its normal texture and becomes somewhat shiny and subsequently sclerotic, giving a taut skin appearance in these areas.

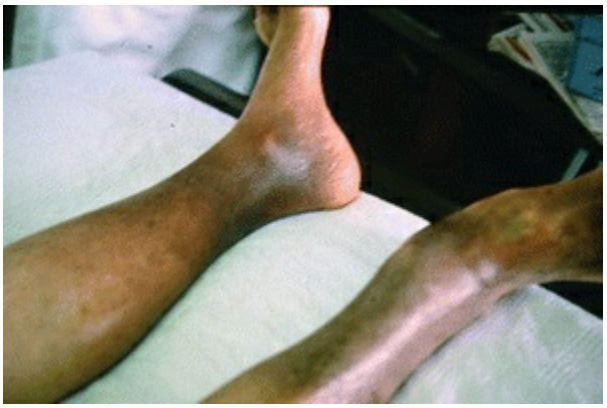


FIGURE 14-3. Hemosiderin staining. Hemosiderin staining caused the discoloration seen here in the patient's right leg from chronic venous hypertension.

Edema and loss of red cells into the subcutaneous tissue occur at the

point of greatest gravitational pressure, the ankle. This gives rise to the pathognomonic features of chronic venous stasis, hyperpigmentation, and stocking distribution induration of the subcutaneous tissues,[14,15] the characteristics of long-standing venous insufficiency called lipodermatosclerosis. These areas are prone to subsequent ulceration or infection; extreme pruritus and excoriation are usually present, potentially aggravating the injured skin. Dermatitis due to endogenous or exogenous sources and severe allergic reactions may complicate the situation. The skin may present as itchy, erythematous, and weeping or dry and scaly (Fig. 14-4). Chemical or mechanical factors may be responsible for contact dermatitis surrounding a leg ulcer.[16]

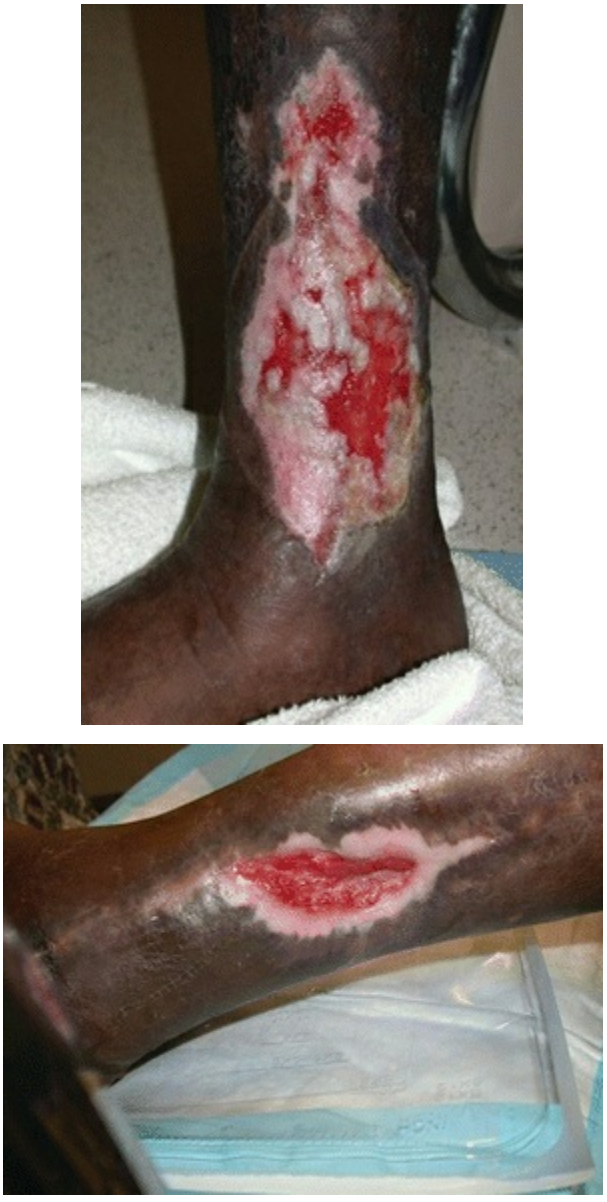


FIGURE 14-4. Venous ulcer with granulating base. Venous ulcer with granulation

tissue and epithelial islands and epithelial growth from the borders.

Another sequela of venous hypertension is irritability of the musculature. Many patients with venous insufficiency—even those in whom the condition is mild—report nocturnal leg cramps. Depolarization may occur due to fluid distention of the muscular cells, causing tetanic-like contractions of various muscle groups. Distention of veins in the subdermal plexus results in the varicosities typically seen with venous insufficiency (Fig. 14-5). The appearance of telangiectasias, more commonly called "spider veins," is the result of distention of the smaller subdermal capillary network (Fig. 14-6).

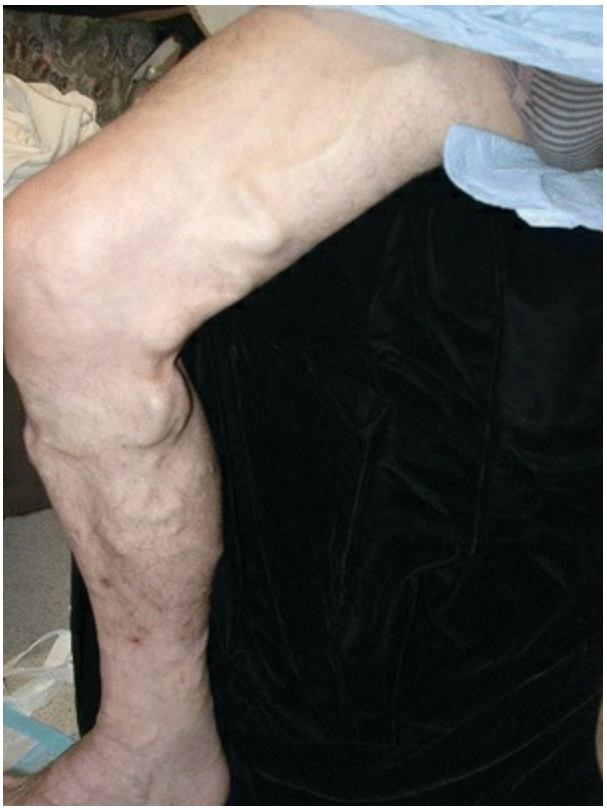


Figure 14-5. Note the presence of varicose veins in the patient's right leg.

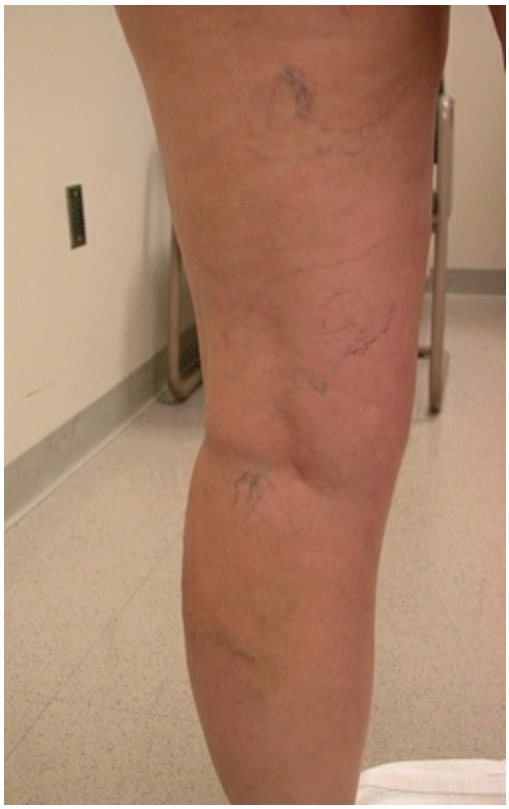


FIGURE 14-6. Telangiectasias, also known as "spider veins," are shown.

In some circumstances, venous aneurysms can occur due to massive dilation of the GSV and its tributaries. Further stagnation of flow in these areas in the presence of an abnormal vessel wall can result in thrombophlebitis, which worsens the venous outflow of the leg and aggravates an already deleterious condition. Thrombosis adheres to the wall of the vein and although recanalization occurs eventually, the valves remain incompetent. In an attempt to compensate for the reduced venous return, the surrounding collateral veins dilate. Chronic edema occurs in the ankle. Increased venous pressure impedes capillary flow, decreasing oxygen available for transport from the capillaries to the tissues, and protein and red blood cells leak into the interstitial tissues. The effect is cumulative, eventually leading to tissue damage, scar formation, and, ultimately, ulceration.

Endothelium in the normal saphenous vein facilitates contraction in response to noradrenaline. In varicose veins, the endothelial-enhanced noradrenaline vasoconstriction is decreased. Endothelial damage is thought to be a possible cause of venous dilatation and subsequent varicose veins.[17]

Venous leg ulcers are also correlated with increased ambulatory venous pressures (AVP). Nicolaides[18] obtained AVP from 220 patients admitted

with venous problems. He found that no patients with an AVP less than 30 mm Hg had leg ulcers, while 100% of those with AVP greater than 90 mm Hg had ulcers. The incidence of ulceration wasn't preferentially associated with either superficial or deep venous disease.

Evidence-Based Practice

Nicolaides' study[18] suggests that AVP should be measured in patients with nonhealing venous ulcers to determine whether they may benefit from a procedure such as a venous valve transplant, which reduces the AVP to less than 30 mm Hg.

Pathogenesis of Venous Ulcers

Several theories have been proposed to explain the mechanism of venous hypertension leading to ulceration. In 1917, Homans suggested that stasis of blood in dilated veins in the skin may cause anoxic cell death, leading to ulcers. In 1929, Blalock[19] found blood oxygen content to be higher than normal in varicose veins, suggesting that arteriovenous communications may be responsible for venous hypertension. In 1972, however, a study using radioactive macroaggregates refuted the arteriovenous shunting hypothesis.[20]

Two hypotheses—the "fibrin cuff" and "white cell trapping" theories—are more recent attempts to explain venous ulcer formation. The fibrin cuff theory states that sustained venous hypertension causes distention of dermal capillary beds, which allows plasma exudate to leak into the surrounding tissue. Fibrin precipitation in the peripapillary space forms fibrin cuffs, which impair oxygen, nutrient, and growth factor transport. The tissues undergo inflammation and fibrosis.[21] A subsequent study suggests that peripapillary fibrin is present but doesn't influence healing of lower-extremity ulcers.[22]

The white cell trapping theory states that the neutrophil aggregation in the capillaries causes lipodermatosclerosis. Increasing venous pressure is thought to reduce capillary perfusion pressure and flow rate. Low capillary flow rate initiates white blood cell adherence to the cell wall. Endothelial cells and leukocytes interact and release proteolytic enzymes, oxygen-free radicals, and lipid products. The white cells are then activated, damaging

the vessel walls, increasing capillary permeability, and allowing larger molecules such as fibrinogen to exit the capillaries.[23,24]

The trap hypothesis of venous ulceration was proposed by Falanga and Eaglstein.[25] This hypothesis proposes that fibrin and other macromolecules that leak out bind or trap growth factors and other substances necessary for maintaining normal tissues and healing. There has not been any new research to support either of these theories.

Classifying Venous Disease

Chronic venous insufficiency (CVI) has been defined as an abnormally functioning venous system caused by venous valvular incompetence with or without associated venous outflow obstruction, which may affect the superficial venous system, the deep venous system, or both.[26] CVI can result in postphlebitic syndrome, which manifests as varicose veins and venous ulcers.

In 1994, the American Venous Forum developed a descriptive system based on clinical, etiologic, anatomic, and pathophysiologic (CEAP) data to categorize the key elements in chronic venous disease.[27] The CEAP system provides an objective classification method that clarifies relationships among contributing factors and improves communication regarding venous disease (Table 14-1). The system is subdivided into seven categories based on objective signs of chronic venous disease.[28] In 2004,[29] this system was refined to add C_0 to the clinical component to represent no visible or palpable signs of venous disease, and subclasses were also added.

Table 14-1 CEAP Classification System

The CEAP classification system for chronic venous disease consists of four elements:

- **C**linical classification
- **E**tiological classification
- **A**natomical classification
- **P**athophysiological classification

Clinical Classification (C_0–C_6)

Class	Description
0	No signs of venous disease
1	Telangiectasias or reticular veins
2	Varicose veins
3	Edema
4a	Pigmentation and eczema
4b	Lipodermatosclerosis and atrophie blanche
5	Healed venous ulcer
6	Active venous ulcer

Etiological Classification (E_C, E_P, E_S)

- Congenital (E_C)
- Primary (E_P)—with undetermined cause
- Secondary (E_S)—with known cause

Anatomical Distribution Classification (A_S, A_D, and A_P)

This element consists of classifications A_S, A_D, and A_P, and segments 1 through 18. See table below for a breakdown.

- Superficial veins (A_S)
- Deep veins (A_D)
- Perforating veins (A_P)

Segment	Classification
As	Superficial veins
1	Superficial veins Telangiectasias/reticular veins Greater (long) saphenous vein
2	• Above knee
3	• Below knee
4	• Lesser (short) saphenous
5	• Nonsaphenous
Ad	Deep veins
6	Inferior vena cava Iliac
7	• Common
8	• Internal
9	• External
10	Pelvic–gonadal, broad ligament, other femoral
11	• Common
12	• Deep
13	• Superficial
14	• Popliteal
15	Crural: anterior tibial, posterior tibial, peroneal (all paired)

Adapted from: (1) Porter, J.M., Moneta, G.L. "Reporting standards in venous disease: an update. International Consensus Committee on Chronic Venous Disease," *Journal of Vascular Surgery* 21:635-45, 1995, (2) Eklof, B., Rutherford, R.B., Bergan, J.J., et al. "Revision of the CEAP Classification for Chronic Venous Disorders: Consensus Statement,". *Journal of Vascular Surgery* 40:1248-52, 2004, (3) Meissner, et al. "Primary Chronic Venous Disorders," *Journal of Vascular Surgery* 2007;46:54S-67S.

C_4 was added to better define the differing severity of venous disease:

C_{4a}: Pigmentation or eczema

C_{4b}: Lipodermatosclerosis or atrophie blanche

In 2000, The American Venous Forum developed an assessment tool called the Venous Clinical Severity Score (VCSS) to supplement the CEAP tool.[30] This assessment tool was revised and updated in 2010.[31] Designed to provide more information about severe chronic venous disease, the VCSS tool consists of six descriptors—pain, varicose veins, venous edema, skin pigmentation, inflammation, and induration—that are rated 0 (absent), 1 (mild), 2 (moderate), or 3 (severe). In addition, there are four descriptors for ulcers: number of active ulcers (0, 1, 2, or 3 or more), active ulcer duration (N/A, <3 months, >3 months but <1 year, not healed for 1 year), active ulcer size (N/A, <2 cm diameter, 2 to 6 cm diameter, >6 cm diameter), and compressive therapy (not used, intermittent use, wears most days, full compliance). This scoring system is an elaboration of the C or clinical component of the CEAP classification system. It is dynamic in that the numbers can change as the ulcers heal or the venous disease process is treated. Combining the two tools provides a method to communicate information about the ulcer in both objective and subjective terms. The VCSS and CEAP tools are expected to improve clinicians' ability to follow venous treatment outcomes.

Diagnosing Venous Ulcers

Venous disease and ulcer etiology can be determined by obtaining a thorough patient history and performing a physical examination. A focused vascular history includes a clear description of the presenting complaint, past medical history for vascular and related conditions, current and previously taken medications, and risk factors. Signs and symptoms of lower-extremity venous disease may include pain, tissue loss, or change in appearance or sensation. It is important to include assessment of the arterial

system when evaluating venous and lymphatic disease. Adequate arterial perfusion is essential when using compression therapy for venous and lymphatic ulcers. An ankle–brachial index (ABI) is a reliable test that can be performed in any office (Fig. 14-7). An ABI should be conducted before applying any compression therapy. It may be necessary to correct the arterial problem before the ulcer can be treated. Noninvasive vascular laboratory testing is used to identify the location of vascular pathology.

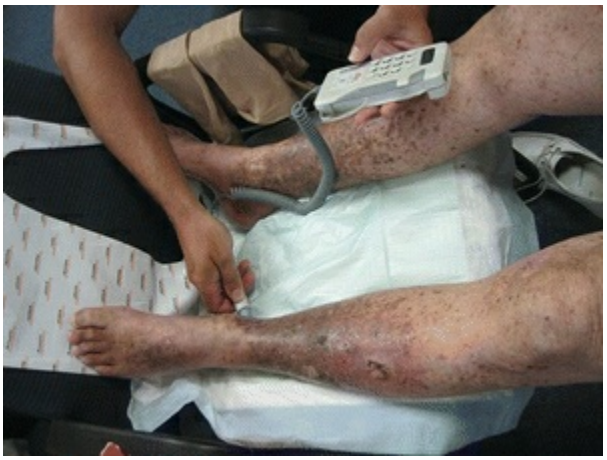


FIGURE 14-7. Ankle–brachial index (ABI) evaluation.

Physical Examination

Physical examination provides direction for intervention, starting with skin inspection. Skin changes in venous disease include hyperpigmentation, dermatitis, lipodermatosclerosis, or atrophie blanche, a characteristic white patchy scarring at the site of previous ulcers. Because skin color may indicate venous congestion, the color of each toe should be noted and compared with the color of other foot and toes. In venous insufficiency, the skin appears a dusky ruddy color. In CVI, the skin may become atrophied with scarring from a previous ulcer, or it may have weeping blisters or dry, scaly crusts.

Skin should be palpated for temperature changes. The skin over varicose veins is often warmer than the surrounding skin. Patients presenting with foot or leg ulcers should be tested for neuropathy, which is a common finding in the diabetic patient.

Edema is commonly found in lower-extremity venous disease. Early edema may be observed as a difference in calf circumference between legs and should be confirmed by measurement. After edema has been long-standing, tissue fibrosis occurs. This makes the skin and subcutaneous

tissues less resilient to palpation.

Venous Signs and Symptoms

Patients may report a gradual onset of discomfort associated with venous disease; however, often, no symptoms are present initially. Most patients describe general nondescript aching rather than specific pain. Some terms used to describe sensations in the legs include fullness, swelling, tightness, aching, or heaviness. These symptoms can be reduced with leg elevation. Venous insufficiency accompanied by acute DVT may be described as sharp, severe, deep, aching pain.[32] Varicose veins occasionally produce a pulling, prickling, or tingling discomfort localized to the area of the varicose vein.[7] In severe cases of venous insufficiency, a form of claudication can occur. The patient may complain of foot edema that makes it difficult to wear shoes.

A venous ulcer is moist and may have a yellow fibrous film covering its surface. This fibrous tissue isn't a sign of infection and doesn't interfere with healing.[33] The ulcer edges may be irregular with firm fibrotic and indurated surrounding skin. The surrounding tissue may have a brownish rust color due to erythrocyte breakdown and deposition of hemosiderin. Scar tissue may indicate the site of a previous ulcer. Because of the subcutaneous scarring, there is no allowance for the tissue expansion that occurs with edema in skin of normal elasticity. Scar tissue also prevents blood vessels from transporting oxygen to the skin, further compromising healing.[34] Lipodermatosclerosis is the term used for chronic inflammation and fibrosis of skin and subcutaneous tissues. It may be associated with diffuse inflammatory painful skin edema.

Vascular Testing

Although an experienced vascular clinician can make a vascular diagnosis based on history and physical examination alone, vascular laboratory studies contribute to the accuracy of the diagnosis. The presence, location, and severity of arterial and venous disease are confirmed by vascular laboratory procedures. Information obtained by vascular studies can predict ulcer healing when the cause is arterial insufficiency.[7] Laboratory tests differentiate among conditions that contribute to a nonhealing ulcer.

Noninvasive vascular testing is divided into direct tests that image the vessel itself and indirect tests that demonstrate changes distal to the

diseased vessel. These tests include Doppler ultrasound, venous duplex ultrasound, ABI, transcutaneous pressure of oxygen ($TCPO_2$), segmental systolic pressures, and plethysmography.

Doppler Ultrasound

In a Doppler ultrasound, a transmitting probe sends a signal that is reflected from an object to the receiving probe. If the signal strikes a moving object such as blood cells, a frequency shift is detected and reflected as sound. The audible signals of venous and arterial flow patterns can be distinguished.

Duplex Ultrasound

The venous duplex allows one to evaluate various segments of the venous tree looking for more than 0.5 seconds reflux after either a muscle contraction or manual augmentation of cephalad flow.[35] An advantage of venous duplex imaging is that it can identify sites of thrombosis with high levels of accuracy. The findings of venous reflex may influence the treatment choice. The disadvantage of this test is that it's fairly time-consuming, taking one to two hours per evaluation for a full leg imaging.

Venous Testing

It's possible to perform a crude venous assessment by physical exam using a Doppler ultrasound. By compressing the limb manually, the flow in the veins can be augmented and noted by the audible Doppler signal heard distal to the site of compression. This is a subjective test, and reliability is clinician dependent. The introduction of noninvasive vascular testing has provided much anatomical and physiological information to increase the accuracy of diagnosing venous diseases. Two tests have been commonly used to assess the severity of venous insufficiency. One is venous photoplethysmography (PPG), and the other is venous duplex imaging.

Plethysmography

Plethysmography records volume changes in the limb. Several types of plethysmography are available:

- air plethysmography, which uses a pneumatic cuff as a segmental volume sensor

- strain-gauge plethysmography, which uses a fine-bore silicone rubber tube filled with mercury wrapped around the limb to be studied
- impedance plethysmography, which measures the relative change in resistive impedance of the passage of an electrical current through a segment of the body
- PPG, which measures the degree of light attenuation, which is proportional to the quantity of blood present and not actual volume change.[7,36]

Air plethysmography and PPG are the more common tests for chronic venous disease. A photoplethysmograph consists of an infrared light-emitting diode and a photo sensor mounted on a probe. The probe is applied to the skin over the area to be tested.[7] The advantage of venous PPG is that it's quick and gives assessment of overall venous refill time. On the other hand, it only evaluates the most dependent portion of the leg in the gator area.

Treating Venous Ulcers

Treatment of venous leg ulcers necessarily involves treatment of the underlying venous disorder. Edema reduction using compression, leg elevation, and exercise are part of the ongoing intervention for venous disease.

Treatment goals for all ulcers are to:

- provide an environment conducive to new tissue growth
- protect the wound
- prevent further tissue destruction.

Topical and systemic treatments are addressed simultaneously. It's imperative to consider the cause when deciding treatment because ulcers aren't all alike and treatment for one type may be inappropriate or harmful for another type. A vascular specialist consultation is appropriate for ulcers of mixed etiology.

Wound Infection

Infected leg ulcers, soft tissue cellulitis, and osteomyelitis are treated by administering systemic IV or oral antibiotics. Topical antibiotics are not indicated for all leg ulcers.[37] Chronic wounds are colonized with normal

skin flora and should not be treated with antibiotics. Rigorous and frequent ulcer cleansing assists in removing surface bacteria. Silver-impregnated dressings have not been shown to have a significant difference in ulcer healing and routine use of these dressings is not recommended.[38]

Biopsy for a quantitative culture of the inflamed tissue surrounding nonhealing ulcers should be considered if true infection isn't responding to antibiotics. If carcinoma is suspected, biopsy of the lesion should be obtained (see Chapter 7, Wound Bioburden and Infection).

Practice Point

Bone scans are expensive and may give false-positive results in the presence of inflammation; therefore, their use is indicated only when bone infection, abscess, or fluid collection is suspected.

Skin and Wound Care

A clean wound, free from dead tissue and wound debris, is necessary for healing to occur. Wound cleaning and debridement are the initial steps in wound care. Many commercial wound cleaners and disinfectants are cytotoxic. Povidone–iodine, hydrogen peroxide, and 0.25% acetic acid have shown evidence of interfering with fibroblast formation and epithelial growth.[39–42] There may be indications for using cidal agents in a wound; however, their use should be time limited, and each caregiver should have a clear understanding of what the goals are and how to recognize when the goals have been reached.

Evidence-Based Practice

The advantage of cleaning the wound should be weighed against the risk of damaging new tissue growth.[43]

The safest wound cleanser is 0.9% saline solution. Wounds should be cleaned with a force strong enough to dislodge debris but gentle enough to prevent damage to newly growing tissue. The pressure to accomplish this

goal ranges from 4 to 15 psi.[44] A 19-gauge needle or 19-gauge angiocatheter distributes approximately 8 psi when used with a 35-mL syringe. Commercial irrigation products with caps designed to deliver adequate pressure are available. Leg ulcers treated in the home are commonly irrigated with running tap water.

Hydrotherapy or whirlpool has been used to aid in cleaning and debridement of both arterial and venous leg ulcers.[45] A clinical pilot study found that whirlpool followed by vigorous rinsing reduced the bacterial load in venous ulcers more than the whirlpool alone.[46] This may suggest that the vigorous irrigation is the significant factor in cleaning the wound. Whirlpool may be contraindicated if the water and dependent position increase edema in the leg. There are no current studies to support or refute whirlpool as a standard recommendation for cleansing venous ulcers.

Dressings chosen for specific wounds depend on the condition of the wound bed and the goal for the wound. Many new dressings are designed to support moist wound healing (see Chapter 9, Wound Treatment Options). Because the skin is fragile in patients with either arterial or venous disease and can be easily injured, tape and adhesive products should be avoided or used with extreme caution. Use other methods of securing dressings that won't injure the skin. A *Cochrane* review on leg ulcer dressings found that the type of dressing material placed under compression had no influence on healing rates.[47,48] Other studies found no advantages with the use of either silver-impregnated[49] or honey-impregnated[50] dressings.

Practice Point

The preferred attachment device for dressings on vascular leg ulcers is roller gauze or commercial devices (such as netting or tube gauze) that hold dressings in place without adhesive, which can damage fragile skin.

Compression

Venous hypertension and venous wounds are treated simultaneously. Wound care and edema management depend on whether or not the patient can be immobilized. Edema is controlled by conservative means: intermittent elevation, compression bandages, and intermittent pneumatic

compression.[51] Studies have demonstrated that moist wound healing combined with compression improves wound healing rate of venous ulcers.[52] Compression therapy is the mainstay of venous ulcer therapy.[53] Elevating the legs above the heart is recommended whenever the patient can be placed in this position. A compression dressing isn't required when the patient is immobilized with the leg elevated, such as during sleeping hours. Moist or dry (depending on the amount of exudate from the wound) gauze dressings with frequent changes can be used instead.

Compression is the application of pressure to the limb. It is measured in millimeters of mercury (mm Hg) and is applied by bandages, elastic stockings, and/or intermittent pneumatic compression pumps. The amount of compression prescribed is determined by the diagnosis, comorbid conditions, and the patient's ability or willingness to accept the treatment. The following standard pressure classification[54] is suggested: mild (<20 mm Hg), moderate (21 to 40 mm Hg), strong (41 to 60 mm Hg), or very strong (≥61 mm Hg). Compression strength of 30 to 40 mm Hg is recommended to counteract the capillary filling pressures within the leg; however, elderly or frail individuals or those who have difficulty donning the stockings may have compression ordered at 20 to 30 mm Hg. Many factors affect bandage pressure, including the bandage itself, the calf muscle and foot pump function, the shape of the leg, and the skill of the person applying the bandage.

The ambulatory venous patient is best served by semirigid dressings, such as the Unna boot or by multicomponent system compression wraps. Multicomponent compression has been shown to be more effective than single-component compression; both four-layer and short-stretch bandages have higher healing rates than paste plus an outer support (Unna boot). With the discovery of moist wound healing[55] and the advent of hydrocolloid and foam dressings, occlusive dressings may be used under compression wraps to promote growth of granulation tissue, reduce pain from the dressing rubbing against the ulcer, and promote autolytic debridement. One study found that ulcers treated with the foam dressing under the Unna boot healed twice as fast as ulcers treated without the foam.[56]

Bandages may be made of different materials, including elastic and inelastic materials or both. Stiff bandages are made of multiple layers of elastic or inelastic material. This type of bandage remains rigid and generates high pressure during exercise, which reduces venous hypertension.

Elastic bandages are considered long stretch if they are capable of

stretching to double their size. Because these dressings can be stretched too tight, they are not recommended as a primary dressing for compression.[57]

Inelastic bandages are nonstretch bandages, short-stretch bandages, and paste bandages. The resting pressure under inelastic bandages decreases over a 24-hour period, but the working pressure (with muscle movement) decreases less.

Compression wraps should be applied starting just below the toes and ending just below (two finger breadths) the popliteal fossa. A gauze roll or padded gauze dressing is typically placed over the wound area, and the dressing is covered with an elastic bandage. If the leg is misshapen, padding the leg to create symmetry facilitates a better bandage fit. This will reduce bandage slippage and avoids a tourniquet effect that increases the distal edema. Extra padding around bony prominences reduces the possibility of the compression garment creating a device-related pressure ulcer. The concave area around the lateral and medial malleoli may need extra padding to ensure sufficient pressure is applied to those areas. Training should be ongoing to avoid complications associated with the application of compression wraps, such as ineffective pressure, pressure ulcers, bandage slippage, and limb distortion[57] (Fig. 14-8).

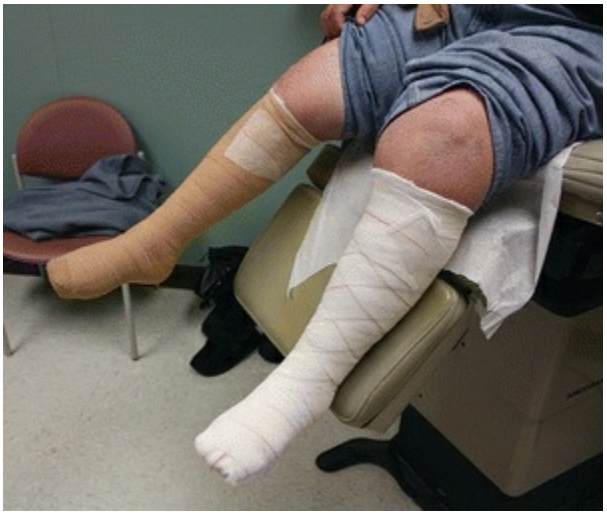


FIGURE 14-8. Compression wrapping. This photograph shows a leg being wrapped with a four-layer compression therapy dressing. (Photo courtesy of J.A. Niezgoda.)

Complications from compression include pain, damage to skin and subcutaneous tissue from pressure, reduction of calf muscle from nonuse, and skin problems. Interventions include carefully assessing to determine the cause of pain; avoiding excessive pressure under the dressing;

encouraging exercise, including range of motion; and avoiding topical products that might cause allergies or irritations.[57]

Stockings reduce AVP by decreasing venous reflux and improving calf muscle ejection capacity during use.[58] The benefit derived from stockings is in direct proportion to the fit.

In many cases, patients fail to wear the prescribed compression dressing or stocking because of difficulty donning the stockings or complaints of tightness. The importance of long-term external compression can't be overemphasized. Patients should be taught that the stockings must be replaced every 3 to 6 months. Two pairs should be purchased so that one can be worn, while the other is laundered.

Practice Point

Long-term compression therapy is an essential part of the treatment of venous leg ulcers.

A pneumatic compression pump may be used to reduce lower-extremity edema. One study found improved venous ulcer healing when compression pumps were used; however, not all third-party payers agree with the use of these pumps for treatment.[59–62] Another study[63] found 4 hours of pump compression per day improved ulcer healing when used in conjunction with compression stockings. In yet another study,[64] an intermittent pneumatic compression device (PCD) provided improved healing when used for 1 hour twice weekly in conjunction with conventional dressings. Patients who are immobile, are unable to tolerate bandages, have arterial insufficiency, or have problems with edema may benefit from intermittent pneumatic compression.[64]

Assessment of Treatment

If a venous ulcer does not show signs of healing, the patient should be assessed for other comorbidities. Diabetes, heart failure, arterial insufficiency, venous reflux or history of venous thrombosis, fibrin on the wound surface, superficial colonization of bacteria, or infection all can slow healing.[65,66]

Exercise

A graded exercise program may be used to improve the calf muscle pump in those patients with abnormalities in pump function. One author[64] determined that a structured exercise program to improve muscle function may have a significant positive outcome in patients with venous disease.[67]

Effective edema reduction from the leg reverses much of the associated comorbidity, particularly the skin changes. The achieved limb reduction must be maintained through the use of appropriately fitted compression hosiery or an alternative compression product. The application and removal of such hosiery can be problematic for individuals with limited hand function or who are unable to see or reach their toes (obese, blind, arthritic, etc.). However, application aids are available and instruction in technique does help. Other strap-type leggings and boot-like devices[40] are available to decrease the functional burden. Therapists can be very creative in combining or layering different compressive products to achieve adequate levels of compression. Compliance is crucial to prevent recurrence of edema.

Surgical Treatment for Venous Ulcers

Surgical treatment for venous ulcers is aimed at correcting the cause of the venous hypertension. Patients can have venous reflux without the symptoms of insufficiency. It is when reflux is severe enough that the insufficiency results in dermal venous hypertension and the eventual skin changes with which patients present. Procedures aimed at correcting insufficiency of the deep venous system include vein valve transplantation, direct valve repair, and venovenous bypass. Outflow obstruction of a limb is addressed with venovenous bypass, endovascular intervention, or a combination of the two. Varicose veins, the manifestation of superficial venous insufficiency, generally require ablation. Their treatment is usually by excision, ligation, injection, or the more recent method of endovenous ablation, depending on the size of the vein.

Surgical treatment for venous insufficiency remains far behind the more established treatment of arterial occlusive disease. Venous insufficiency can be grouped under two broad categories:

- venous reflux
- venous outflow obstruction.

The net result of both of these disease entities is venous hypertension and the sequelae resulting in dermal injury that ultimately ends with venous ulcerations. The mainstay for the treatment of venous insufficiency continues to be good external compression. In many patients, this is all that is required. Compression acts both as treatment for various states of venous insufficiency as well as prophylaxis for the development of the adverse sequelae. In some patients, the use of compression alone is inadequate; for these patients, surgical intervention is usually necessary.

Venous outflow obstruction is usually the result of DVT; however, other causes include obstruction from surgical excision of outflow vessels, tumor encasement of venous return, radiation-induced fibrosis, congenital atresia, injection-induced venous destruction (illicit or otherwise), obesity with immobility, and infection. When it involves isolated segments with normal segments proximal and distal, the obstruction can result in a cascading event resulting in venous insufficiency. In some patients, the outflow obstruction is the cause of the symptoms. In these patients, it may be necessary to relieve the venous outflow obstruction and the corresponding venous hypertension by bypassing the obstructed segment, by balloon angioplasty of a sclerotic or stenotic segment with or without stent placement or, if the DVT is not chronic, by either open venous thrombectomy or mechanical thrombectomy.

Endovascular treatment of iliac vein obstruction is now possible on a routine basis. The morphology of this outflow obstruction and the measures to best treat it are ideally evaluated with the use of intravascular ultrasound (IVUS).[68] Although there is an early reocclusion rate of around 35% at 3 months, subsequent ulcer recurrence-free rates of almost 60% exist out to 5 years.[69] Early thrombosis and in-stent stenosis remain the most common causes of these early failures.

For years, obstruction from DVT has been approached in Europe with open venous thrombectomy, but this technique has never caught on in the United States. Mechanical thrombectomy via percutaneous routes is a focus of current interest. Two devices currently popular are Trellis drug dispersion and thrombectomy catheter (Bacchus, Santa Clara, CA),[70,71] and the AngioJet (Possis Medical, Minneapolis, MN).[72,73] Each uses a unique method of clot destruction and aspiration combined with lytic therapy. Trellis traps the thrombus between two balloons and emulsifies it with a rheolytic agent and oscillating wire. Once the clot is emulsified, it is aspirated from between the two balloons. The AngioJet uses lytics and a high-velocity jet of liquid to fragment and then rapidly aspirate the thrombus fragments back into the catheter. Both methods are effective, with Trellis

being quicker and AngioJet gentler on the vessel's intima. Whether these devices will have good long-term results is still in debate. Posttreatment patients need to be on warfarin.

Patients who require bypass are best served using an autogenous venous conduit. The proximal and distal anastomoses of the venous bypass are dictated by the obstruction site. For example, if a patient has an isolated iliofemoral thrombosis that has failed to recannulize or has only partially recannulized and the affected leg is symptomatic, a femoral–femoral bypass from the proximal portion of the symptomatic leg to the more distal portion of the contralateral leg can be performed. The saphenous vein is usually used for this, but in contrast to arterial bypasses, the direction of the valve isn't reversed; rather the valve leaflets are oriented to prevent reflux. Similarly, a bypass from a more proximal vein within a symptomatic leg to the more cephalad iliac vein may be indicated.[74]

In patients with an outflow obstruction, but in whom insufficiency or hypertension is caused by occlusion of the GSV, the venous hypertension may be alleviated by isolated partial saphenous vein ligation and stripping. This is usually done at the knee level with stripping of the affected saphenous segment. If, however, the reflux or hypertension is the result of the deep venous system, then stripping the GSV wouldn't help and actually may be detrimental due to elimination of one of the venous outflow tracts of the extremity. This information must be known before a surgical procedure is performed to correct venous insufficiency. Three tests are available to evaluate reflux:

- ascending/descending venography
- duplex imaging
- venous PPG with sequential tourniquet placement.

These tests are all readily available through either an accredited vascular laboratory or a radiology department with experience in venous testing. Venous PPG can determine whether the deep or superficial system is involved with venous reflux. It is an excellent assessment of overall limb reflux and helps assess for perforator incompetence. Venous duplex, which interrogates specific segments of both the superficial and deep veins for reflux, gives a more detailed anatomic evaluation of the affected extremity. Both are needed to properly evaluate a patient with venous disease. Isolated valve segments of the more proximal venous system can be evaluated using descending venography looking for contrast reflux past the incompetent valve. Venography and venous duplex imaging can also determine sites of

stenoses within the venous system. One of the problems associated with accurate duplex imaging is that it's dependent upon the competence of the technician performing the test. Certain laboratories have more expertise in these areas than do others. This notwithstanding, duplex imaging has largely replaced venography in evaluating patients with venous disease. Venography is still used if surgical bypass and/or valvular surgery is planned.

When deep venous insufficiency is due to valvular incompetence, it isn't known how many competent valves are required and in what locations for the deep venous system to become competent again. Research in these areas is still ongoing as more attention is given to venous insufficiency.[75]

Three techniques are available for surgical correction of venous insufficiency due to valvular incompetence:

- artificial venous valve insertion
- autogenous vein valve transplantation using a segment of vein, usually from the upper extremities or axilla
- direct valvuloplasty.

Autogenous valve transplantation is a procedure in which a segment of vein with a competent valve, usually from the upper extremity or axilla, is identified. This section of vein is resected and an interposition graft placed at the harvest site. The vein is then transposed into the venous system of the affected extremity, maintaining the orientation of the valve to keep the leaflets open in a cephalic direction. Postoperative anticoagulation with heparin and subsequently Warfarin is commonly used.

Approximately 75% of the stasis ulcers remain healed 12 months after valve transplantation.[76] However, considerable degradation occurred over the course of the 2nd year in these patients, such that only 40% of limbs remain healed.[76] After the second year, results appear to stabilize without further deterioration although the reports on this are limited in both scope and number.[76]

Variations of this procedure using valve segments from other areas or even transposing a deep vein with a competent valve with another deep vein with an incompetent valve can be done. In Ko et al.'s study, the overall results with this method appeared to be similar to that of transposition of competent vein valve segments.[76]

Direct valvuloplasty is another technique for correcting valvular insufficiency. This is performed by suture approximation of two valve

leaflets that don't fully close. It's done either with direct suturing within the areas of the cusps to obtain good apposition or by external buttressing of an incompetent vein valve sinus. Valve leaflets themselves are brought into apposition by placing the equivalent of a "girdle" around the dilated valve to reduce dilation and allow the valves to come in apposition in a more normal fashion. This sleeve technique is usually done with prosthetic material. Similarly, a transplanted valve that may deteriorate due to dilation can be made competent again by using this technique.[77]

An external valve repair known as the *Psathakis Silastic sling procedure* has been developed.[78] This procedure involves placing a Silastic sling around the popliteal vein and then attaching it to the two heads of the biceps femoris muscle. When these muscles contract, the sling is intended to occlude the popliteal vein during ambulation. The problem with this procedure is that the sling becomes intimately adherent to the vein and surrounding tissue and over time no longer functions in this fashion.

Two publications on neovalve construction and valvular repair highlight the various techniques employed to restore venous competency of the deep system and their outcomes.[79,80] These are technically challenging operations that are not widely available. When successful, ulcer healing rates exceed 88%.

In patients in whom no suitable vein valve segment can be found or it's deemed an inadequate operation, the development and implantation of a prosthetic valve holds some promise. Currently, a prosthetic venous valve comprised of a complex titanium double-leaflet system is being developed and may hold promise.

The appropriate use of adequate compression is necessary in conjunction with all the surgical treatments. The application of compression hose and management of these patients are addressed elsewhere within this chapter.

Patients with recurrent leg ulcers due to incompetent perforators in the affected area were thought to benefit from a Linton flap. This procedure requires elevation of the skin and fascia at the site of ulceration and transection with ligation of the incompetent perforator veins feeding the area. Proper application of compression is required afterward to reduce local venous hypertension. The morbidity associated with this procedure includes tissue slough along the area of incision and the overlying tissue, resulting in a prolonged healing period. This is thought to be due to the chronic disease state of the tissue at the ankle.

A subfascial ligation of incompetent perforator veins with an endoscope

(SEPS) is a significant advancement in the Linton technique. Using equipment developed for laparoscopic cholecystectomies, the scope is passed from the medial infrageniculate area into the subfascial space. The fascia is raised, often with CO_2 insufflation. The perforator veins are ligated and transected through the endoscope. Advances in technology, such as smaller-diameter scopes (5 to 10 mm), high-resolution camera and LCD screens, and single-step electrocautery/transection, have made this procedure much easier to perform. The smaller-diameter endoscopes allow easy perimalleolar inspection for perforators. This entire procedure routinely takes less than an hour to perform and in select patients can be done on an outpatient basis. The reason for ligating incompetent perforators is to eliminate the venous hypertension associated with the reflux of venous blood.[79] In a meta-analysis by Tenbrook et al., ulcers treated by SEPS with or without additional venous ablation healed in 88% of patients.[81,82]

There has been some interest in duplex ultrasound–guided foam sclerotherapy, which scleroses the perforator veins to achieve the same effect.[83,84] While this treatment is used extensively in Europe, it's not yet approved in the United States.[85] Further studies are needed to establish the overall effectiveness of this procedure for wound healing.

The use of endovenous ablation has really become widespread in the United States, supplanting traditional vein stripping. In most places, endovenous ablation is an outpatient office-based procedure. It involves ultrasound-guided cannulation of the distal saphenous vein, either lesser or greater, with a catheter whose tip is positioned 2 cm distal to the saphenofemoral junction. The length of the vein is surrounded with tumescent anesthesia usually consisting of a dilute buffered lidocaine solution (usually 300 to 500 mL). This acts as a heat trap for the delivered energy, which destroys the vein wall and coagulates the blood within. The energy delivered is either in the form of a laser (endovenous laser therapy, EVLT) or radiofrequency (RF). Proponents of both forms claim superiority. The end result, if successful, is controlled thrombosis and destruction of the vein and thereby prevention of reflux through it. US data show 99.6% successful occlusion initially,[86] falling to 86% to 89% at 4 years.[87] Devices of both types exist that can be used to directly ablate incompetent perforators under ultrasound guidance. However, neither of these devices is as easy to use as the main units.

RF and EVLT are quick procedures, routinely taking far less than 1 hour to perform. The patients generally do very well, and the procedures are well tolerated. The patient avoids a hospital stay and the need for a major

anesthetic. Endovenous ablation has been widely accepted as a tool for the patient seeking removal of mostly asymptomatic varicosities, but when appropriately applied, the procedure can correct isolated saphenous reflux disease. While the procedure has a low complication rate, it has two major drawbacks that open vein stripping does not: It usually leaves behind a distal segment of vein, and recannulation can occur. Some centers have combined endovenous ablation with a subsequent limited distal stripping or microphlebectomy, if needed. Depending on how extensive the condition, either of these procedures could be office based.

In some patients, the application of a split-thickness skin graft to an otherwise healthy stasis ulcer may be appropriate. This technique shouldn't be used in patients whose underlying venous problems haven't been addressed. The application of a split-thickness skin graft to an ulcer with persistent venous hypertension will fail, even if the patient is compliant with the use of a compression garment.

Options other than split-thickness grafting involve the use of bioengineered human dermal substitutes. These are intended to be used as carriers of human growth stimulants and to provide a scaffold upon which the patient's own dermis can regenerate, either spontaneously or with the use of a delayed thin skin graft. Dermal substitutes are not inexpensive, however, and the underlying venous issue as well as the wound bed must still be addressed. Although skin substitutes appear to hasten wound closure, recurrence rates are high.[88]

Modern venous surgery now has in its armamentarium a wider array of treatment options for those patients with venous disease. As this area of vascular surgery matures, better paradigms for patient treatment are emerging.[89] No longer is it just an Unna boot and vein stripping.

Summary

CVI is a permanent condition. Because of this, patients are given information about the disease process and rationale for intervention. The more information they have, the more likely they are to manage the condition effectively. Activities that promote venous return are encouraged. Extremity elevation should become a daily routine, and external compression is needed for life. Patients must understand the importance of this fact. Protection from skin trauma is essential. A small lesion may progress quickly to a large ulcer because of the edema. It may take years to heal, if at all. Small cuts or bruises should receive immediate medical attention. Leg

exercises to increase muscle pump activity are taught to the patient. Patients are encouraged to use these exercises during long periods of standing or sitting. When sitting, the legs should be elevated.

Success in managing venous ulcers requires a total patient commitment. Risk factors and ulcer management are dependent upon the patient's activities; therefore, the patient must have as much information as possible to participate in therapy. An understanding of venous pathophysiology and its contribution to leg ulcers is critical in managing the ulcers. Venous reconstruction is in its infancy but shows promise to reduce the sequelae of postphlebitic syndrome. Venous ulcers always require external compression, ultimately in the form of compression stockings or layered bandaging systems.

Venous ulcers always require external compression, ultimately in the form of compression stockings or layered bandaging systems. While nurses know the importance of compression as part of the treatment of venous ulcers, in a recent study, they may not feel confident in how to apply these 3- or 4-layer bandaging systems.[90] In another recent study, nurses may overestimate their ability to apply these bandaging systems.[91] Both of these studies underscore the importance of making use that nurses can correctly apply compression.[90,91]

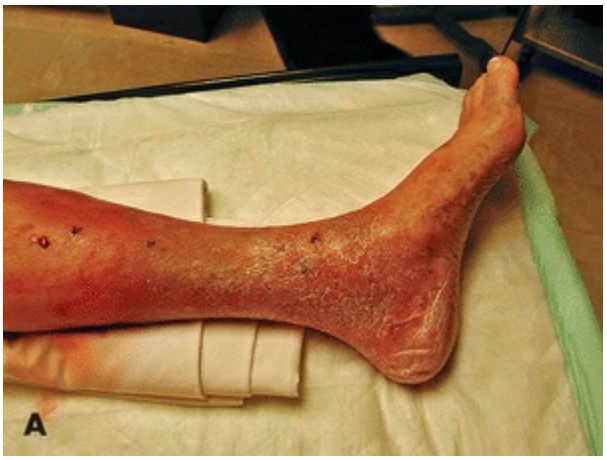
Ulcers associated with lymphedema usually respond when the edema is reduced. A variety of wound care products are available for leg ulcers, but no research exists showing one product to be more effective than another. Economic concerns make it imperative to choose the appropriate dressings and treatment, but research demonstrates little increased benefit of the newer treatments over the old. Although surgical treatment for venous ulcer lags behind that for arterial occlusive disease, there are procedures that correct the insufficiency of the deep venous system. These are vein valve transplantation, direct valve repair, and venovenous bypass.

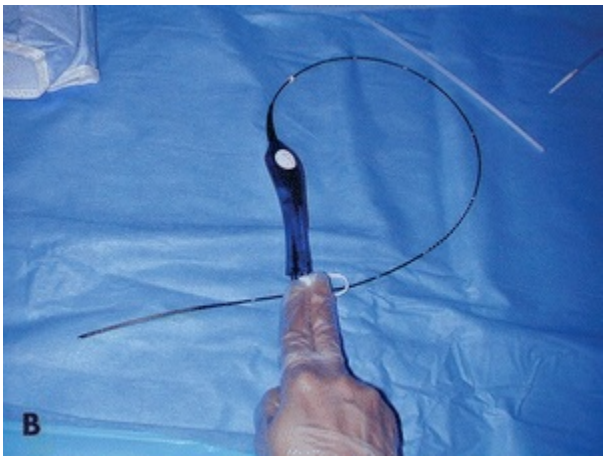
PATIENT SCENARIO

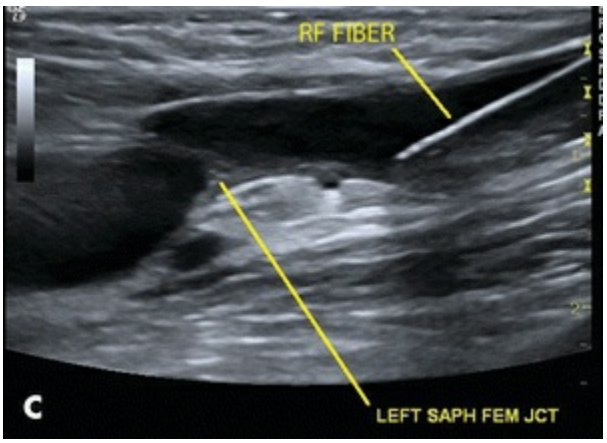
Clinical Data

Mrs. MC is an 82-year-old woman who has had multiple recurrent superficial ulcers in the medial aspect of the left ankle region, presumably resulting from venous insufficiency. Some ulcers healed spontaneously, but more recently, they required compression therapy at

a wound center. The current episode was indolent for more than 8 months and has left Mrs. MC with macerated skin that cracks and weeps serous fluid (Fig. 14-9A). She recently moved in with her daughter but is quite independent. Although Mrs. MC is thin, her nutrition is adequate and she is compliant with leg elevation. She has worn proper compression hose in the past. She was referred to the Vascular Surgery Service for evaluation.







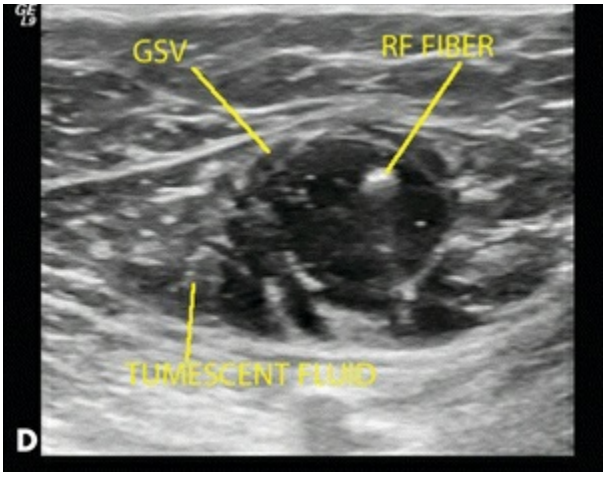


Figure 14-9. (A) Macerated skin due to multiple recurrent superficial ulcers in the medial aspect of the left ankle region. **(B)** Radiofrequency fiber. **(C)** Placement of the radiofrequency fiber approximately 2 cm distal to the junction of the common femoral vein. **(D)** Tumescent fluid infused around the GSV.

The left leg has no palpable pedal pulses, although Mrs. MC does not describe any claudication or rest pain. She has a remote history of limited smoking. Other than hypertension and the current left leg complaints, she is healthy. She underwent arterial and venous vascular lab evaluations consisting of arterial Doppler ultrasound, venous PPG, and venous duplex tests for reflux. Her ABIs were greater than 0.95 bilaterally with triphasic waveforms. Venous refill times were markedly abnormal bilaterally, 12 seconds on the left and 15 seconds on the right. Tourniquet application indicated isolated GSV incompetence in both legs. Venous duplex testing revealed no DVTs, greater than 0.5 seconds of reflux in the left proximal femoral vein and the entire left GSV. Reflux was also noted in the entire right GSV. Endovenous ablation of the left GSV was recommended.

Case Discussion

Mrs. MC's GSV incompetence on her symptomatic left side is most likely the cause of her recurrent venous ulcers and now chronic tissue and skin changes. Although there is reflux in the left proximal femoral vein, the GSV is the predominant source of her dermal venous hypertension. With ablation of this GSV and, once healed, wearing proper support hose, it is expected that her symptoms will improve and her ulcer will heal. Because of her age and the desire to avoid both

hospitalization and a major anesthetic, endovenous ablation with radiofrequency was the intervention of choice. This office-based procedure requires only local tumescent anesthesia.

The radiofrequency fiber (Fig. 14-9B) was passed up the GSV after entry at the distal calf level via ultrasound guidance. After placement approximately 2 cm distal to the junction of the common femoral vein (Fig. 14-9C), tumescent infusion around the vein was performed (Fig. 14-9D). Several cycles of heat were applied as the fiber was sequentially pulled back. Once completed, the limb was compressed using thigh-high support hose of 30 to 40 mm Hg compression; support hose were worn for an initial 72 continuous hours followed by 7 additional days of nonsleep wear. The patient then returned for a series of duplex ultrasound tests to evaluate the ablation and to rule out DVT.

Mrs. MC is now doing well with well-healed skin. This took an additional 2 months to achieve. She wears knee-high support hose of 30 to 40 mm Hg compression religiously on both limbs. The right leg remains asymptomatic.

LYMPHEDEMA

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Objectives

After completing this chapter, you will be able to:

- describe the epidemiology of lymphedema
- explain the physiology of edema formation
- describe prevention and proper management of lymphedema.

Edema (swelling) results when an increase in interstitial fluid volume occurs, representing an imbalance between capillary filtration and lymph drainage. *Lymphedema* represents a failure of lymphatic drainage.[1] This section examines the epidemiology of lymphedema as well as its mechanisms, manifestations, and management.

Epidemiology of Lymphedema

As discussed by Logan[2] and Williams et al.,[3] the epidemiology of lymphedema has been difficult to ascertain due to a variety of issues in determining prevalence and incidence rates. Perhaps more importantly, lymphedema lacks a valid and consistent definition. This is due in part to the unavailability of a good diagnostic test and challenges in quantifying the severity of edema and the associated skin and tissue changes. For example, chronic lower-extremity edema (perhaps caused by heart failure or venous insufficiency) can mimic lymphedema. In this case, excess low-viscosity, protein-poor interstitial fluid resulting from increased capillary filtration accumulates in the subcutaneous tissue because a *normal* lymphatic system cannot handle the imposed load.

True "primary lymphedema" arises from congenital or developmental defects of the lymphatic system. Usually one limb is more severely affected, and the condition was once thought to be rare. "Secondary lymphedema," by contrast, is seen more frequently and is usually triggered in Western countries by lymph node dissection and perhaps irradiation treatment for cancer and in developing countries by parasitic infection. However, traumatic injury, chronic inflammation or infection, and other surgical interventions may also precipitate the condition. More recent evidence obtained from direct visualization of the lymphatics using near-infrared imaging[4] suggests that secondary lymphedema may be a subclinical form of primary lymphedema. Patients who develop lymphedema after surgery or trauma may have had marginal lymphatic function to begin with, which became dysfunctional due to injury or insult.

Left untreated, lymphedema in some patients can progress through several stages until it manifests as elephantiasis, a most disfiguring condition. Although lymphedema can be managed successfully with lifelong adherence to a maintenance protocol (manual lymph drainage [MLD], compression garments, skin care, and exercise) and psychosocial support, cure is not possible because the damaged lymphatic system cannot be repaired.

Globally, lymphedema resulting from filariasis (infection by the nematode *Wuchereria bancrofti*, which is carried by mosquitoes) affects nearly 119 million people or 2% of the world's population,[5] primarily in tropical areas of the world such as India, Africa, Haiti, and Malaysia. However, a discussion of filariatic lymphedema is beyond the scope of this chapter. In Western countries, upper-extremity lymphedema due to

complications resulting from breast cancer treatment is by far the most common presentation and is usually associated with surgery and/or irradiation of the lymph nodes. Risk factors identified for breast cancer–related lymphedema include irradiation, the extent of axillary node dissection, combined axillary dissection and irradiation, obesity, surgical wound infection, tumor stage, and extent of surgery.[3,6–8] Although surgical improvements have been made during the past few decades, the incidence of secondary lymphedema remains at about 30%, although the data may also reflect improved detection of the condition.[3,8] Cancer-related lymphedema in the lower extremity is less prevalent. Studies have demonstrated that the incidence of lower-extremity lymphedema is dependent on the location of the cancer; for example, the incidences are 40% and 55% following groin or ilioinguinal dissection, respectively, but 6% to 11% following combined pelvic and lymph node dissection for stage III melanoma.[9–11] Risk factors for development of this type of lymphedema include tumor location and factors similar to those identified for breast cancer–related lymphedema.

Although clinicians now understand the relationship between breast cancer treatment and lymphedema, they are less likely to be aware that chronic inflammation and ulceration have the potential to damage the lymphatic system.[12–15] Chronic venous disease, trauma, recurrent infection, and arthritis may contribute to lower-extremity lymphedema, yet many clinicians may not appreciate these conditions as causative agents.[2,12–16] Increasingly, obesity is a contributing factor; although the mechanism remains unclear, it is likely due in part to increasing venous insufficiency among the morbidly obese, exacerbated by immobility and volume overload. Clinicians may not be able to differentiate between lymphedema and lipedema, a condition involving abnormal fat distribution, typically around the legs or hips. Unfortunately, if lymphatic insufficiency is not recognized, ineffective treatment and increased morbidity are likely to ensue.

A UK study recently addressed some of the aforementioned epidemiological issues by using a broader operational definition of chronic edema/lymphedema and setting specific clinical criteria to identify cases.[13–17] These criteria included persistent edema lasting longer than 3 months, little or no response to overnight elevation or diuretics, and the presence of skin changes (primarily thickened skin, hyperkeratosis, and papillomatosis). Individuals with congestive heart failure, hypoalbuminemia, or nephrotic syndrome were excluded from the study as these systemic disorders were likely the cause of the edema. The population

included only those individuals known to, or being treated by, health professionals. While the study authors acknowledged that their broad definition of chronic edema/lymphedema made it likely that cases with mixed etiologies would be included, they argued that chronic edema thus identified represented some form of lymphatic insufficiency because a functional lymphatic system ought to be able to compensate for an increase in capillary filtration by increasing lymph drainage.[13–17]

A crude prevalence of 0.13% was reported in the study, with age-related increases reaching 0.54% for those older than 65 years and 1.0% for those over age 85.[18] Although these figures translate into some 100,000 cases of chronic edema/lymphedema in the United Kingdom[18] or 395,000 in the United States based on current population estimates, it is doubtful that all patients were receiving treatment during the study period, and thus, the prevalence estimates are likely to be on the conservative side. In a Norwegian study, Petlund[19] reported a crude prevalence of 0.144% for chronic edema, which buttresses the UK figures. Other important findings from the UK study illustrated the impact of the condition on patients' lives. For example, almost one-third of the study participants suffered an acute infection in the preceding year, and a quarter of these patients required hospitalization. The condition was also responsible for sick leave in 80% of individuals and a change in employment status in 9%. Moreover, while it is often reported in the literature that lymphedema is not painful, half of the patients indicated that they experienced pain or discomfort with the edema. These issues were also reflected by low scores in many domains of the well-validated SF-36 questionnaire, suggesting a poor quality of life.[18] In summary, chronic edema/lymphedema seems to be a frequently unrecognized complaint associated with significant morbidity and appears with a frequency equal to that of leg ulcers.

Lymphatic System

Compared with the vascular systems in the leg, the lymphatic system is the least understood, and its embryologic development remains relatively unknown. Lymphatic vessels are divided into three categories: initial or terminal lymphatic capillaries, collecting vessels, and lymph nodes.

Terminal lymphatic capillaries originate in the superficial layer of the dermis and have no valves. These lymphatic capillaries drain into the deep dermal and subdermal system, which is the level at which valved lymphatic vessels can be observed (precollectors), and vessels ascend the leg into

lymph nodes at the popliteal fossa and the inguinal ligament. Generally, the lymphatic system parallels the larger veins of proximal leg above the knee, with valves operating in much the same way as venous valves. The lymphatic system then drains though the iliac lymph nodes above the inguinal ligament level, eventually coalescing into the periaortic nodes, the cisterna chyli, and thoracic duct, which ascends along the thoracic aorta on the right side of the chest and empties into the left jugular vein slightly above the jugulosubclavian junction. While the thoracic duct is considered the main terminus of the lymphatic system, some patients have an accessory right lymphatic duct that drains into the right jugular venous system.

Lymphatic vessels are much smaller than major arteries or veins—between one-seventh and one-tenth the size—because fluid flow is less. In terms of vessel anatomy, the outer adventitial layer is much thinner although the media contains some elastin fibers and smooth muscle striae, the latter being used to propel the lymphatic flow cephalad through contraction. The intimal layer consists of a single layer of endothelium.

Aspects of Lymphatic Flow

Lymphatic flow is a consequence of three factors: capillary blood pressure, osmotic pressure, and interstitial fluid pressure (hydrostatic). The intrinsic contractility of the lymphatic vessel wall, coupled with the action of muscular pumps such as the calf, which aids flow in the same way as for the venous system, creates a suction force distal to the major lymph vessels. In addition, the action of deep breathing, which creates a positive abdominal pressure and a negative thoracic pressure, also increases the cephalic lymph flow.

Because capillary cell walls are “leaky,” acellular interstitial fluid containing protein and white blood cells accumulates, and thus, the lymphatic system provides a means for drainage of this fluid as well as a mechanism for the return of white blood cells to the vasculature. A normal lymphatic system with intact functional architecture is required for unimpaired lymph circulation. Lymphedema results when disruption or injury occurs to the lymphatic system at a local level because interstitial fluid is no longer being drained adequately.

The lymphatic system can be considered a one-way transportation system that prevents the body from drowning in its own fluid. However, besides maintaining interstitial tissue fluid balance (volume and pressure), the lymphatic system also performs other key functions. Composed of a treelike hierarchical network of vessels and organs, including the spleen,

thymus, tonsils, bone marrow, and numerous lymph nodes, the lymphatic system biologically filters lymph at the nodes using macrophages and lymphocytes. The lymphatic organs and nodes also provide a means for lymphocyte maturation and transportation that is crucial to immune function; lymphocytes include natural killer cells involved in the innate immune system, whereas T-cells and B-cells are associated with the adaptive immune response.[12,20–22] In addition, the lymphatic system plays a role in certain kinds of fat absorption. Thus, when portions of the lymphatic system are injured, the local response to inflammation or infection likewise becomes disrupted by disturbing cytokine (growth factor) and cellular circulation in the affected area.[12] In other words, the lymphedematous extremities develop fatty deposits in response to chronic edema, and the swollen limb really does become "fatter." For the purpose of this chapter, only the structures and mechanisms specific to lymphatic circulation of the lower limb are discussed.

While the anatomy of the lymphatic system mirrors that of the venous system in terms of general structure and function, there are distinct differences in that the lymphatic system has lymph nodes and the vessels are thinner and have more valves than their venous counterparts. Although little is known concerning the embryology of the lymphatic system, its development proceeds on a parallel with the venous system starting with origination in lymph sacs (e.g., the jugular, iliac, retroperitoneal, and cisterna chyli sacs). Its venous origin has also been supported by recent work that demonstrates the presence of tyrosine kinase 4, a gene specific to both venous and lymphatic but not arterial endothelia early in the development process that ultimately becomes expressed solely within lymphatic endothelia.[12,23,24]

Lower Limb Lymphatic Anatomy

The lymphatic system is comprised of the small, noncontractile initial lymphatic vessels, also known as lymphatic capillaries, whose function is to absorb interstitial fluid, linked to the progressively larger contractile collector vessels. Lymphatic capillaries begin as blind-ended tubes only a single cell in thickness, but the cells are arranged in a slightly overlapping pattern similar to the shingles on a roof and are connected to surrounding tissue through anchoring filaments. The interstitial pressure forces the cells to separate periodically, which allows lymph to enter but not escape as the cell walls then close to reestablish their overlapping pattern. This process

resembles a one-way valve system.[21,25,26] Precollector vessels linked to the capillaries possess segments with capillary-like walls, as well as valves, and merge into larger contractile vessels termed collectors or trunks, which have valves and intervalve segments called lymphangions. The function of these entities is to propel lymph forward cephalically via vessel wall smooth muscle contractions initiated by pacemaker cells or modulated through sympathetic activation.[25,26]

Practice Point

Smooth muscle contraction in the lymphangions is dependent on the influx of calcium ions. Therefore, calcium channel blocking agents commonly used to treat high blood pressure can have a negative effect on lymphatic contractility that contributes to peripheral edema.[13]

Akin to the venous system, the deep fascia divides the lymphatic system into deep/subfascial and superficial/suprafascial networks connected by perforating vessels. Superficial capillaries originate in the dermis and drain into subcutaneous collectors, which are organized into progressively larger bundles. The superficial system drains both the dermis and subcutaneous tissue. While skin areas emptied of interstitial fluid by one collector form topographical strips known as *skin zones* in which the lymph vessels freely communicate, skin zones associated with all the collectors from a lymph vessel bundle form distinct territories that are separated by watersheds in which few vessels communicate.[27]

Practice Point

The communication ties or anastomoses between lymphatic capillaries in different areas provide an anatomical basis for "manual lymph drainage," an important component of the therapeutic intervention for lymphedema. MLD consists of specific manual techniques designed to increase lymph flow and remove excess tissue fluid from the congested area of one watershed via

the lymphatic capillaries to another whose drainage is intact.

Muscles, bones, and joints are drained by the deep subfascial collectors. However, although subfascial collectors share the same perivascular sheath construction as their venous and arterial counterparts, lymph is directed from the deep to the superficial lymphatic system, which is the opposite of the venous system in which blood drains from superficial veins through the perforating veins into the deep veins.[28]

Lymph Flow in the Leg

Plasma that has escaped from the capillary vasculature mixes with other interstitial materials (forming prelymph) and enters the lymphatic capillaries and precollectors in a passive process aided by rhythmic contractions of the lymphangions upstream, nearby muscular contractions and arterial pulsation, suction pressure due to breathing, and manual lymph drainage. The lymph then flows into serially larger collectors of which the more proximal are known as *trunks*. Distention of the trunk wall is the stimulus for lymphangion contraction, which provides the primary propulsion necessary for lymph flow and which occurs at a rate of 6 to 10 beats per minute. In essence, the lymphangions function like miniature hearts in linear sequence[12] that are also capable of cardiac-like inotropic and chronotropic responses.[12,20,28] Under normal conditions, lymph flow can increase by an order of magnitude when an increased filtrate volume is present with higher prelymph uptake and a faster rate of lymphangion contraction,[16] ensuring a large margin of capacity. In other words, healthy lymphatics, like the heart, can work harder when more fluid is present. Until recently, the pulsatile flow of human lymphatics had never been observed in real time. These supplemental videos of near-infrared lymphatic images depict the normal collecting lymphatics on the dorsum of the foot of a 48-year-old female (Movie S1), the ankle/calf of a 47-year-old male (Movie S2), and the knee of a 43-year-old male (Movie S3) (http://www.pnas.org/lookup/suppl/doi:10.1073/pnas.1222722110/-/DCSupplemental).[29]

Practice Point

Exercise or passive movement induces alternating changes in the interstitial fluid pressure, which translates to improved lymphatic capillary filling. This effect can partially compensate for damaged collectors whose contractility has failed. Applying a bandage or other form of external compression such as manual lymphatic drainage[30] enhances the effect of movement (muscle contraction), thus increasing lymph flow.

Lymph is propelled by afferent collectors in the leg and funnels through some 7 to 15 trunks via an anteromedial route that follows the course of the great saphenous vein where it is filtered in the nodes of the popliteal fossa and the inguinal ligament.[12,13] From the inguinal nodes, lymph is moved cephalad through several iliac nodes before coalescing into periaortic nodes and trunks, from there into the cisterna chyli, and finally into the thoracic duct. The thoracic duct is located on the right side of chest and follows the thoracic aorta before draining into the left jugular vein just above the jugulosubclavian junction. In some individuals, an accessory right lymphatic duct, which drains into the right jugular venous system, may also be present.

Cephalic (toward the head) lymph flow in the central region of the body is particularly assisted by breathing and local arterial pulsation. Although the thoracic duct is traditionally considered to be chief drainage route for lymph back into the venous system, other drainage methods exist, for example, lymphovenous communications within muscle compartments and on the periphery of the body. However, it is thought that these do not typically play a role in lymph drainage unless chronic obstruction of lymph vessels or nodes is present.[31]

Practice Point

Exercise, deep breathing, and manual lymph drainage are all considered essential components in the treatment of chronic edema/lymphedema because they all increase lymph flow.

Tissue Edema

Pathophysiology of Edema in Venous Disease

Regardless of the etiology of edema, the immediate cause is always an imbalance between capillary filtration and lymph drainage. Consequently, an understanding of the forces associated with interstitial fluid balance can be helpful. Capillary filtration is easily illustrated using the Starling equation, which describes flow across a semipermeable membrane. The net movement of fluid from the inside of a capillary to the interstitium, often referred to as the capillary filtration rate, is governed by the difference between the capillary and interstitial hydrostatic pressures and the difference between the capillary and interstitial osmotic pressures. (Osmotic pressure is the force derived from the attraction of water to large-molecular-weight entities).

Net filtration is also influenced by the permeability of the capillary wall to water, to small proteins that can exert an osmotic (or oncotic) effect, and to the surface area available for filtration. Under normal conditions, the Starling equation correctly predicts a net filtration rate, and the volume of interstitial fluid that is added over a period of time is also removed. In other words, the amount of fluid in the tissue remains stable. Otherwise, accumulation of fluid in the tissue spaces would result. For many years, it was thought that reabsorption of the capillary filtrate was achieved primarily by venous capillaries, but more recent evidence from 12 types of tissue suggests that it is the lymphatic system that takes the major role of fluid uptake.[13,32,33] In other words, the lymphatics may be responsible for the majority of fluid returning to the heart. The lymphatic system returns 2 to 4 L of fluid containing approximately 240 g of protein back to the circulation, so it can be readily appreciated that without the lymphatic system, cardiovascular collapse would quickly occur, not to mention a life-threatening shortage of protein.

Edema Due to Increased Filtration

The most common form of edema, known as *high-volume lymphatic insufficiency*, develops when the capillary filtration rate exceeds lymph drainage capacity over a period of time. This is analogous to what happens when a municipal drainage system that, although it is still working, becomes overwhelmed by heavy rain, resulting in flooding. In this example, a high volume of fluid can overwhelm the capacity of the drainage system. Typically, increases in capillary filtration are the consequence of increased capillary pressure due to venous hypertension, heart failure, or fluid

overload. However, reduced plasma osmotic pressure is another cause of increased capillary pressure. In other words, fluid leaves the vasculature for the interstitium because osmolality in the interstitium is higher than in the blood vessels. This can be due to hypoalbuminemia, perhaps from nephrotic syndrome or liver failure. In addition, an increased capillary permeability to water and small proteins can occur. Inflammation may cause this increased capillary permeability; it may also increase blood flow, which further increases capillary pressure and capillary filtration rate. Thus, venous disease is associated with at least two potential causes of high-volume lymphatic insufficiency (increased capillary pressure and interstitial inflammation), and in cases in which the lymphatic system deteriorates, lymphedema may result.[22]

Edema Due to Reduced Lymphatic Transport

The swelling present in lymphedema can also be caused by a gross mechanical failure of the lymphatic system to accommodate even a *normal* load of capillary filtrate.[27] This condition is also known as *low-volume lymphatic insufficiency*. This situation is analogous to the flooding that would occur from a small amount of rain if the municipal drains become blocked. The lymphatic failure can result from lymphatic capillaries and precollectors becoming unable to absorb capillary filtrate or from a dysfunction upstream that prevents conduction or filtering of a normal load.

Mechanisms Limiting Edema Formation

Because venous disease induces capillary hypertension and therefore increased capillary filtration, edema must result. Nevertheless, there are several mechanisms by which the edema can be limited. These mechanisms, which form the physiological basis for treatment, include:

- increased interstitial pressure due to increased stiffness of the skin and soft tissues
- a reduction in the interstitial osmotic pressure
- increased lymph flow
- postural vasoconstriction in a dependent leg via the veniarteriolar reflex
- activation of the calf muscle pump.

Increased Interstitial Fluid Pressure

The ability of a tissue to resist swelling is directly related to its compliance —an inverse measure of its stiffness. Muscle or fibrotic tissue containing collagen or other extracellular matrix fibers resists swelling because a small increase in interstitial fluid volume results in a large increase in interstitial pressure, which opposes the capillary filtration force. Conversely, because compliant tissue (such as the skin of the eyelid) has elastic properties, it can accommodate a much higher volume of fluid before the interstitial pressure rises sufficiently to oppose filtration.

The venous-arteriolar reflex that generates vasoconstriction in a dependent leg also acts to oppose filtration by decreasing capillary pressure.

Reduced Interstitial Osmotic Pressure

When the capillary filtration rate increases, the local interstitial protein concentration is reduced, particularly if the prelymph removal rate does not increase, and the plasma protein concentration is increased. The combined effects tend to decrease the interstitial osmotic pressure and increase the capillary osmotic pressure with the result that further increases in capillary filtration rate are resisted. This feedback mechanism is of major importance in protection against both pulmonary and peripheral edema.

Increased Lymph Flow

Increased capillary filtration is a consequence of the elevated venous pressure found in venous disease. In addition to the previously mentioned osmotic effects opposing filtration, the higher level of filtrate entering the lymphatic capillaries stimulates more forceful lymphangion contraction cephalad, and thus, the lymph transportation rate is increased to match the load. The ability to dramatically increase transport capacity in response to increased lymphatic load is also known as the *lymphatic safety valve function* and is an important mechanism preventing edema formation. Other factors that limit edema formation include local pressure changes in tissues and capillaries induced by movement in active or passive exercise, manual lymph drainage, arterial pulsation, and, in more central tissues, breathing. Activation of the calf muscle pump not only decreases venous pressure but also enhances lymph flow.

Practice Point

Bandaging and stockings enhance the pumping effect of exercise by providing a "shell" against which the foot and calf muscles can compress during contraction. Higher evacuation of venous blood leads to a decrease in venous capillary pressure and filtration. Intermittent changes in tissue pressure from walking and other rhythmic exercise increase lymphatic capillary filling and lymphangion contractility thereby increasing lymph flow.[33–34]

When venous disease is not attended by edema, this means that the previously mentioned mechanisms are compensating satisfactorily. On the other hand, the presence of edema with venous disease indicates that either the compensatory mechanisms are inadequate or the capillary filtration rate greatly exceeds the forces that oppose it. The foremost contributory factor in the latter case is likely to be inflammation, which may advance along the perivascular perforating sheath or spread between divisions.[12,28,34]

Lymphatic Failure in Venous Disease

Lymph flow increases during the early stages of venous disease but becomes compromised in later stages when lipodermatosclerosis and/or venous ulcers are present. Obese patients have many issues that contribute to lower-extremity lymphedema, including increased capillary filtration, decreased calf muscle pumping (due to decreased activity), and long periods of dependency (particularly in patients with sleep apnea who may be unable to sleep supine). There may be genetic factors that predispose to both obesity and lymphedema.

Many theories have been proposed to explain the generation of venous ulcers, and similarly, several ideas have been advanced to explain the development of lymphedema during chronic venous disease. One of the most intriguing concepts has been that patients who develop secondary lymphedema have already acquired subclinical manifestations of the condition from birth due to genetic defects; other patients may have fewer lymphatic vessels or vessels that have been damaged over a period of time from various causes. In one study of venous ulcers, light and electron microscopy demonstrated that superficial fibrin and inflammatory cell

layers and the intermediate blood capillary layer of the ulcer bed did not contain lymphatics.[35] In addition, only a few lymphatic capillaries were present in the transition zone from granulation tissue to the deeper collagenous scar layer of the ulcer. These observations suggest greatly lowered fluid reabsorption and lymph flow in the affected area.[35] Fluorescent microlymphography studies have also observed a greatly diminished superficial lymphatic network with accompanying dilation of other lymphatics, as well as increases in permeability.[36–39] In postthrombotic syndrome, lymphoscintigraphy showed that subfascial lymphatic drainage was substantially impaired,[40] and the same technique demonstrated that lymphatic function was reduced in legs exhibiting venous ulcers compared with legs that had no ulcers.[41,42] It is theorized that obliteration of lymphatic vessels that results in loss of function probably develops through lymphangiothrombosis or lymphangitis in a similar fashion to that which occurs in veins, although it is likely to be a much slower process. In addition, lymphangions may become dysfunctional by losing their contractile ability, although whether this might be linked to valvular incompetence, and therefore, lymph reflux is not known. One technique still under development that may help to validate these theories is near-infrared fluorescent imaging, which employs fluorophores such as indocyanine green (ICG) and is much more sensitive than radiotracers. The technique has recently been used in humans for lymph mapping in breast cancer patients, intraoperative guidance, and real-time functional imaging.[43] Patients with CVI frequently develop lymphedema by an unclear mechanism. PCDs have been shown to benefit venous ulcer healing, presumably by enhanced venous return, but their effect on the lymphatic system is unknown. In an IRB-approved trial at the University of Texas Health Science Center, Houston (sponsored by Tactile Systems), 10 patients 18 and older with a venous stasis ulcer underwent eight intradermal injections of 25 μg of ICG dye to each leg. After 3 hours of baseline lymphatic imaging, a PCD was applied to one leg for 1 hour and imaging was continued for another 30 minutes. The velocity and periodicity of propulsive ICG fluorescence packets in the lymphatic system were evaluated before and after pneumatic compression. Some CVI patients were noted to have baseline lymphatic abnormalities similar to those in postmastectomy lymphedema, and in response to PCD, more than half of the patients exhibited new lymphatic vessel recruitment to regions proximal to the wound. In some cases, lymphatic vessel activation was remarkably enhanced with PCD. While further research is warranted to understand how venous disease induces lymphedema, this is the first study

to demonstrate the effect of lymphatic function in vivo and how mechanical compression devices might benefit these patients.[44] In the later stages of venous disease, which involve tissue fibrosis, the stiffness of the tissues may mitigate edema or prevent edema from being detected easily; some controversy exists regarding whether this stage should be termed chronic lymphatic insufficiency rather than lymphedema.[45,46] However, the 2003 consensus document from the International Society of Lymphology defines lymphedema as an external (or internal) manifestation of lymphatic insufficiency and deranged lymph transport,[22] and thus, lymphatic failure in later stages of venous disease would be included under this definition.

The broadest classification of lymphedema pathology defines whether it is obstructive or nonobliterative. Obstructive pathology results from any perilymphangitis etiology, whereas nonobliterative pathology can result from endolymphangitis proliferans, primary thoracic duct pathology, lymph node obstruction, congenital defects, or lymphatic thrombosis.

In the United States, tumors are the most common cause of lymph node obstruction, but primary thoracic duct disease can be congenital or surgically acquired. Endolymphangitis is typically a result of repeated intraluminal injury from many types of noxious agents that cause repeated injury. Whereas lymphangiectasis is a true atrophy of the lymphatic channel rather than a developmental problem, congenital factors, which usually cause a nonobliterative disorder, result in either agenesis or hypoplasia. One of the most common phenotypes of the nonobliterative disorder is Meige's disease, representing approximately 3% of all cases of lymphedema. Meige's disease primarily affects females; the age of onset is variable, although it typically occurs near puberty.[47]

Practice Point

Milroy's disease is the rarest form of primary lymphedema, often present at birth. Although unilateral lymphedema may be present initially, it can be bilateral.

Lymphatic thrombosis is another form of the nonobliterative disease sometimes encountered. Although anticoagulants are ineffective, the administration of benzopyrones such as coumarin (not to be confused with coumadin) may be able to reduce the edema by stimulating macrophage

activity, which increases degradation of protein in lymph fluid.[48,49]

Classifying Lymphedema

The oldest classification of lymphedema divided the condition into three categories: congenital, lymphedema precox, and lymphedema tarda. Congenital lymphedema is diagnosed at birth or shortly thereafter, while lymphedema precox is diagnosed between birth and the age of 35 years, although the majority of cases are found around puberty. The term lymphedema tarda is simply applied to a case in which onset occurs after the age of 35. The classification system in use today is based on whether the lymphedema is primary or secondary.

Primary lymphedema can be described as either hyperplastic/hypoplastic/aplastic or obstructive/nonobstructive. Obstructive pathologies are usually described according to anatomical location and divided into distal obliterative or pelvic obliterative. For example, about 10% of patients have primary hyperplastic lymphedema, which is classified as bilateral hyperplasia, sometimes with megalymphatics (large valveless lymphatic ducts similar to varicosities); bilateral hyperplasia is characterized by capillary angiomata on the sides of the feet. An obstructive process is usually present at the level of the cisterna chyli or thoracic duct, and valves can be observed when examined. The patient may exhibit little or no leg edema, but chylous reflux is present.

Secondary lymphatic obstructions result from a variety of causes, including tumors, surgical intervention, trauma, or infection. Infections can be bacterial or filarial. In developing countries, the most common cause of lymphatic obstruction is filarial infection by *W. bancrofti*. As discussed above, our understanding of secondary lymphedema is changing with the advent of better imaging techniques.

One of the most common dilemmas facing wound care clinicians is differentiating between venous disease/other types of edema and lymphatic disorders, although it has been noted that venous disease may cause lymphedema. Differentiating between these conditions is important because treatment profiles differ considerably. Many pathological processes can mimic lymphedema and need to be excluded in order to make a diagnosis. These include arteriovenous malformations, lipedema (an abnormal accumulation of fat in the tissues of the leg), erythrocyanosis frigid (bluish discoloration of extremities secondary to cold exposure), factitious edema, and gigantism.

Differential Diagnosis of Edema

Characteristic Features of Lymphedema

It is important to differentiate lymphedema from other forms of peripheral edema so that appropriate treatment can be provided[50] (Table 14-2). Clinical history, physical examination, and simple tests often make this distinction possible, although imaging procedures may be needed to confirm diagnoses in some cases. For example, diuretics increase the excretion of water and salt, thereby reducing plasma volume, venous capillary pressure, and filtration. Thus, diuretics improve filtration edema but have no effect on lymph drainage over the long term.[12] Similarly, overnight elevation of the legs will improve 90% of a filtration-based edema because higher venous pressure is coupled with higher capillary filtration rates, but only a 10% to 20% improvement in edema will be observed, if there is any improvement at all, in lymphedema.[13] Thus, relative unresponsiveness in both these situations likely indicates lymphedema.

Table 14-2 Differential Diagnosis Between Obesity, Lipedema, Lymphedema, and Lipolymphedema

Characteristic	Obesity	Lipedema	Lymphedema	Lipolymphedema
Gender	Male or female	Almost exclusively female	Male or female	Almost exclusively female
Time of onset	Childhood onward	Typically at age 10–30 y	Childhood (primary) Adult (secondary)	Typically at age 30 y onward
Family history positive	Common	Common	Only for primary lymphedema	Occasionally
Effect of dieting on condition	Positive	None	None	None
Effect of elevation	None	Minimal	None	Helpful until fibrosis occurs
Pitting edema	Absent	Minimal	Pitting may stop as fibrosis progresses	Usually present to some degree
Bruises easily	No	Yes	No	Yes
Pain	None	Present in legs	None in early stages	Present in legs
Area affected	All parts of the body	Bilateral legs, thighs, buttocks (*feet spared*); arms sometimes (*hands spared*)	Feet affected at first, then progressive leg involvement; unilateral more common than bilateral	Feet affected eventually with positive Stemmer's sign; usually lower extremities (bilateral)
Stemmer's sign	Absent	Absent	Present	Present

Reprinted with permission from Fife, C.E., Maus, E.A., Carter, M.J. "Lipedema: A Frequently

Misdiagnosed and Misunderstood Fatty Deposition Syndrome," *Advances in Skin and Wound Care* 23(2):84, 2010.

Onset of symptoms may also provide further clues, with lymphedema having a much slower duration of onset compared with edema from other causes. The distinctive skin thickening with fissures and other soft tissue changes (such as hyperkeratosis or papillomatosis) that occur in response to chronic lymphatic system congestion also point to lymphedema. While pitting is often present in the early stages of lymphedema and then disappears as the disease progresses, it can still be made to occur with prolonged compression to accommodate for the increase in fibrosis and skin thickness. Another maneuver, the Kaposi-Stemmer sign, tests the inability to pinch and pick up a fold of skin at the base of the second toe and is predictive of lymphatic insufficiency,[27] although it is not infallible. (The test is "positive" if the clinician is *not* able to pinch a fold of skin at the base of the second toe, indicating lymphedema.) Finally, an increase in swelling of more proximal segments of the affected limb, the contralateral limb, and/or the adjacent trunk quadrant in response to compression (bandaging, garments, or intermittent pneumatic compression) is an indication of lymphatic insufficiency proximal to the limb segment undergoing compression. Regrettably, not all clinicians make this observation if it occurs. Along with continuing inappropriate compression therapy, this missed observation can lead to a situation in which a patient incurs further congestion of the more proximal segment with associated damage to the lymphatic system. This problem is not uncommon in patients with late-stage venous disease in whom the leg has been decongested, but lymphedema has developed in the region of the thigh, buttock, or genital area. Individuals with this response should be referred to a certified lymphedema therapist because appropriate treatment will require complex decongestive therapy, including manual lymph drainage, and may require treatment of the trunk and/or contralateral limb (also, in the United States, refer to http:\\www.lymphnet.org for a list of Lymphology Association of North America–certified therapists by state).

Lymphatic Failure in Venous Disease: Implications for Management

Improving lymph drainage is a goal in all edema therapy because edema is caused by either the increased capillary filtration rate overwhelming the

intact lymphatic system or primary failure of the lymphatic system to transport lymph. While similarities do exist between venous disease and lymphedema in terms of treatment, it is important to detect lymphatic insufficiency in the later stages of venous disease; otherwise, improper treatment and further damage to the lymphatics could follow. Besides treatment, measures to prevent worsening of the condition are also crucial, and these include meticulous skin and wound care, interventions to reduce capillary filtration edema, and interventions to increase lymph flow using compression therapy, exercise, and/or manual lymph drainage.[51]

Skin and Wound Care

In lymphatic insufficiency, a diminished immune response coupled with the high protein content of the interstitial fluid creates ideal conditions for bacterial growth and an increased risk for infection, particularly cellulitis. Such infections may be recurrent and can occur without skin ulcers being present.[18] In particular, fibrotic skin changes, such as fissures, together with hyperkeratosis, papillomatosis, and increased surface debris and scale, elevate the possibility of bacterial or fungal colonization or invasion. Furthermore, increased edema is associated with higher rates of wound drainage, which means clinicians need to be cognizant about the risk of skin maceration, more frequent dressing changes, selection of more absorptive dressings, and appropriate bandaging. The toes and forefoot are especially vulnerable, as compression wrapping of the toes and foot is often necessary to accomplish graduated compression all the way to the knee. The use of multiple layers of short-stretch bandages and padding is generally advisable because the altered size and shape of the leg make bandaging a complex effort; in obese patients, other techniques, such as the use of Velcro strapping devices (e.g., FarrowWrap, Farrow Medical Innovations, Bryan, TX, or CircAid T3, CircAid Medical Products, San Diego, CA), may be more suitable. Patients will also need to be educated about functional ambulation, a key component of treatment, and may require assistive devices as compression bandages often limit mobility. Adjunct treatments, such as deep breathing exercises and manual lymph drainage, will benefit venous ulcer patients with lymphedema, and the latter is also an important component of pure lymphedema therapy.

Decreasing Filtration Edema

During the day, leg elevation is frequently prescribed for patients who

depend on wheelchairs for mobility. To do any good, however, the patient must lie supine with his or her legs at least at the level of the heart, if not above; "in-between" times, resting positions with the patient's torso in an upright condition and the legs raised are only partially effective. Moreover, the effective resting posture does not encourage ambulation, which might be more effective in the long term if performed regularly. Diuretics should only be prescribed in cases of sodium and fluid retention associated with heart failure or nephrotic syndrome; while they decrease venous volume and ultimately filtration rate, they do not enhance lymph flow, and their long-term use in lymphedema is ineffective and may be harmful.

Summary

Lymphedema results when there is an imbalance between capillary filtration and lymphatic drainage. In the United States, lymphedema occurs most commonly due to injury or obstruction, such as from cancer treatment, or associated with severe venous insufficiency and/or obesity. There is no cure, but treatment with compression can effect significant improvement and control of symptoms, which may include skin changes, limb distortion, and cellulitis.

PATIENT SCENARIO

Clinical Data

Patients with CVI frequently develop lymphedema by an unclear mechanism. PCDs have been shown to benefit venous ulcer healing, presumably by enhanced venous return, but their effect on the lymphatic system is unknown. In an IRB-approved trial at the University of Texas Health Science Center, Houston (sponsored by Tactile Systems), 10 patients 18 and older with a venous stasis ulcer underwent 8 intradermal injections of 25 micrograms of ICG dye to each leg (Fig. 14-10). After 3 hours of baseline lymphatic imaging, a PCD was applied to one leg for 1 hour, and imaging was continued for another 30 minutes. The velocity and periodicity of propulsive ICG fluorescence packets in the lymphatic system were evaluated before and after pneumatic compression. Some CVI patients were noted to have baseline lymphatic abnormalities similar to those in

postmastectomy lymphedema, and in response to PCD, more than half of the patients exhibited new lymphatic vessel recruitment to regions proximal to the wound. In some cases, lymphatic vessel activation was remarkably enhanced with PCD. While further research is warranted to understand how venous disease induces lymphedema, this is the first study to demonstrate the effect of lymphatic function in vivo and how mechanical compression devices might benefit these patients.

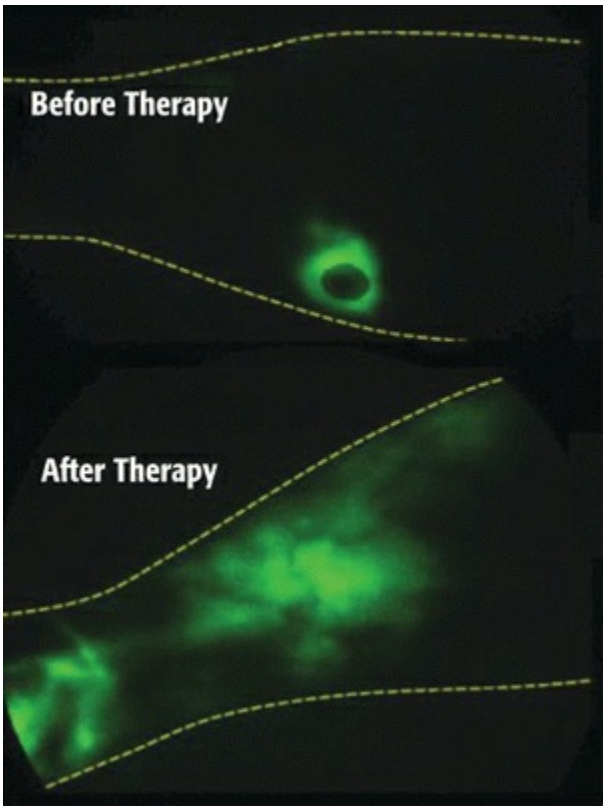

FIGURE 14-10. Appearance of a leg with a venous stasis ulcer imaged with ICG before and after 1 hour of treatment with a pneumatic compression device. The increased distribution of the ICG dye demonstrates lymphatic vessel recruitment in response to PCD as dye moves from distal (left of image at ankle) to proximal but also shows the abnormal distribution of dye due to lymphatic damage associated with venous insufficiency of long duration.

Case Discussion

A 44-year-old white female with type I diabetes presented with lymphedema. Although she had been overweight most of her life, leg swelling had begun about 10 years prior after minor trauma resulted in cellulitis. Pendulous enlargements at the back of both thighs began

slowly and continued to increase over the years until they began to affect her ability to walk. Her history was also significant for Hashimoto's disease and chronic thyroiditis. On clinical examination, she was found to have stage III lymphedema with elephantiasis and massive localized lymphedema (MLL) on both legs (Fig. 14-11). MLL is characterized by extreme localized enlargement of the limb (usually the thigh but can be many other body locations) due to lymphatic fluid and its associated inflammatory tissue changes. Because it is often mistaken for a tumor, it is sometimes referred to as a "pseudosarcoma." MLL is more common in morbidly obese patients and patients with thyroid disease.

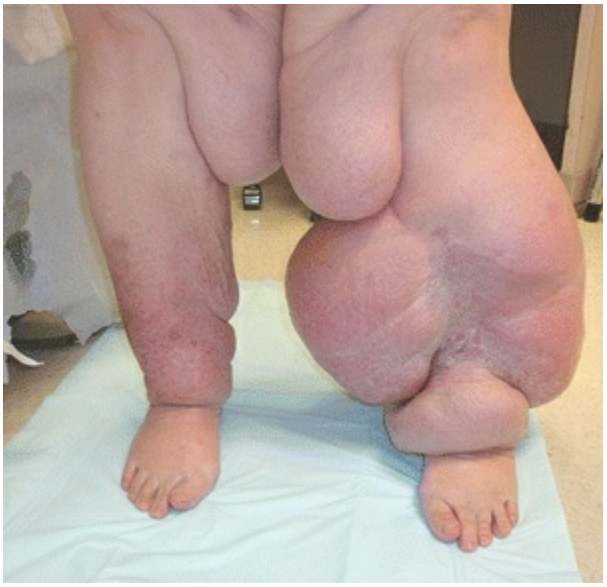

FIGURE 14-11. 44-year-old female with lymphedema and massive localized lymphedema.

The patient responded well to standard treatment for lymphedema with complete decongestive physiotherapy and compression bandaging (Fig. 14-12). She was provided with custom garments and a PCD to help her with long-term management of lymphedema.

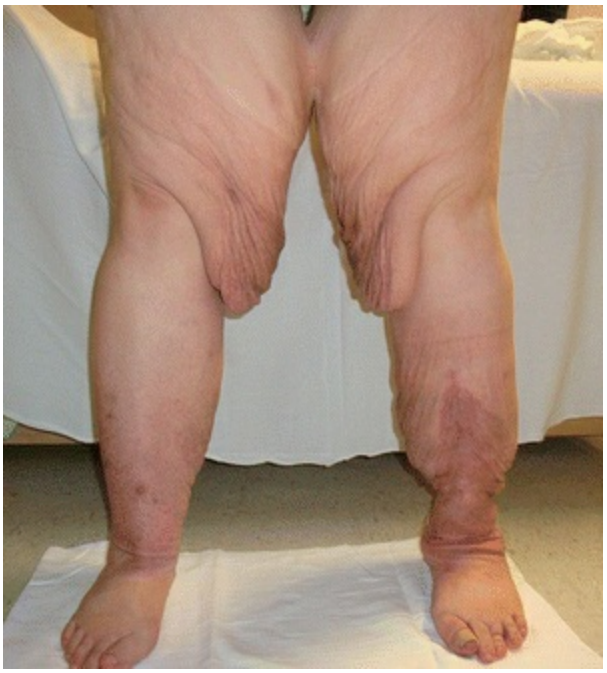

FIGURE 14-12. After complete decongestive physiotherapy.

Show What You Know: Venous Disease

1. The cause of venous ulcers is:

A. venous stasis.
B. venous hypertension.
C. embolic phenomenon.
D. varicose veins.

2. The ankle–brachial index (ABI) is an indicator of loss of perfusion in the lower extremity.

A. True
B. False

3. The most important treatment component for venous ulcers is:

A. moist wound healing.
B. antibiotics.
C. compression.
D. revascularization.

4. The most detrimental activity a patient with any vascular disease can do is:

A. walk into the pain.
B. sleep with legs dependent.
C. use nicotine.
D. fail to monitor pulses.

Show What You Know: Lymphedema

1. Which of the following statement about the epidemiology and course of lymphedema in the developed world is false?

A. It is most commonly the result of cancer treatment.
B. Overnight elevation and aggressive diuresis are likely to improve it.
C. While treatment improves symptoms, lymphedema is not curable.
D. Lymphedema may be confused with lipedema, a genetically mediated fatty deposition syndrome.

2. Which of the following scenarios is likely to contribute to edema in the lower limb?

A. A decrease in capillary filtration rate
B. A decrease in interstitial fluid pressure
C. A decrease in intravascular oncotic pressure
D. A decrease in capillary ultrafiltrate

3. Optimal care of lower-extremity lymphedema includes all of the following except:

A. Compression with short-stretch bandages and fitting of appropriate compression garments
B. Manual lymphatic drainage
C. Routine use of diuretics
D. Exercise and ambulation

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Arterial Ulcers

15

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Objectives

After completing this chapter, the reader will:

- identify the structure and explain the function of the lower-extremity arterial system
- assess the signs and symptoms of lower-extremity arterial disease and ulcers
- select appropriate vascular laboratory diagnostic testing for lower-extremity arterial disease
- evaluate medical and surgical treatment options for lower-extremity arterial disease
- design appropriate patient education for prevention and appropriate lifestyle change.

Scope of the Problem

The designation, peripheral vascular disease, commonly refers to arterial problems in the legs. The reader should be aware that some authors also include diseases of the venous and lymphatic systems in their definition of peripheral vascular disease.

Leg and foot ulcers may have several different etiologies, including arterial, venous, and lymphatic disease along with trauma, infections, inflammatory diseases, and malignancy. This chapter describes the arterial component, including anatomy and physiology, and examines the treatment of lower-extremity arterial ulcers.

Approximately 8% to 10% of patients in some series with leg and foot ulcers have pure arterial insufficiency.[1] It's estimated that between 1% and 22% of the population over age 60 suffers from lower-extremity skin ulcers.[2–5] One patient survey suggested the current estimate may be low because high numbers of patients indicated that they cared for their own ulcers without consulting a healthcare provider.[6] The principal etiology of leg ulcers is chronic venous disease, whereas foot ulcers are much more commonly caused by arterial disease.[1,7–9] Peripheral arterial ischemia is also the most common vascular consult for limited ability to walk.[7] Although chronic wounds have physical, financial, and psychological effects, it's difficult to measure this impact on a patient's activities of everyday living.[10] It's also difficult to obtain accurate etiological information about leg ulcers because no ulcer etiology documentation exists in about one-third of medical records.

Vascular Anatomy and Physiology

Vascular anatomy includes the arterial, venous, and lymphatic systems. Vascular ulcers may develop in any of these systems from a variety of causes. For the purposes of this chapter, we will confine our discussion to the arterial system.

Arterial System

Lower-extremity arterial perfusion begins with adequate cardiac performance. As blood exits the left ventricle, it begins its downward course through the descending thoracic aorta meeting several arterial branches. The **intercostal arteries**, which arise from the descending thoracic aorta, are the first important collaterals to perfusion in the legs. These become important when they are the sole collaterals in distal aortic occlusive disease. As the aorta exits the thorax and enters the true abdominal cavity, its caliber begins to decrease after every major arterial branch. Its greatest reduction in size occurs distal to the renal arteries.

Lumbar arteries usually arise as paired vessels at each vertebral level in the abdomen. The lumbar arteries become important collateral pathways to the lower extremities in distal aortic occlusions or severe aortoiliac occlusive disease. At the level of the umbilicus, the abdominal aorta bifurcates into the **common iliac arteries** that in turn branch into **internal and external iliac arteries**. The internal iliac arteries perfuse the lower

sigmoid colon and rectum. They also, by way of the **gluteal and pudendal branches,** provide another collateral pathway to perfusion of the legs. The external iliac artery becomes the common femoral artery at the level of the inguinal ligament. It's at this level that one can first appreciate the quality of the pulse wave by palpating the femoral artery.

Aspects of the Femoral Artery

The **common femoral artery** bifurcates into the **superficial femoral artery and the deep femoral artery**. The deep femoral artery is the single most important collateral pathway for perfusion of the lower portion of the leg. Its muscular perforators allow reconstitution of the popliteal artery in superficial femoral artery occlusions. The superficial femoral artery becomes the **popliteal artery** after it exits the adductor hiatus, also known as *Hunter's canal* (Fig. 15-1).

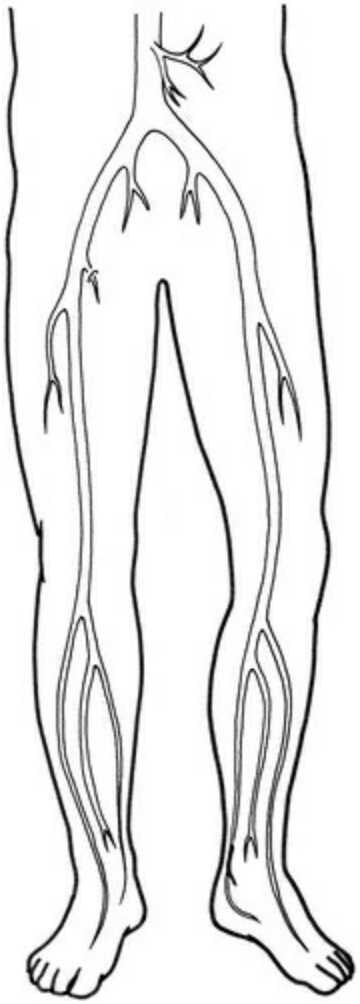


Figure 15-1. Arterial system. This illustration shows the major arteries of the

lower extremities.

The superficial femoral artery is the most commonly occluded artery in the legs of patients with peripheral vascular occlusive disease. Its occlusion infrequently results in significant ischemia to the lower leg. Below the knee, the popliteal artery bifurcates into the **tibioperoneal trunk and the anterior tibial artery**. The anterior tibial artery proceeds from the popliteal fossa through the interosseous membrane, which connects the tibia and fibula; it then courses down the anterior muscle compartment into the foot. The tibioperoneal trunk (also known as the tibiofibular trunk) at a variable distance then bifurcates into the **peroneal (also known as the fibular) artery and the posterior tibial artery.**

The peroneal artery courses down toward the ankle in the deep muscular compartment, whereas the posterior tibial artery descends into the foot in a more superficial fashion. The peroneal artery provides important muscular profusion branches. It's commonly patent even in the presence of severe lower-extremity peripheral vascular occlusive disease.

Aspects of the Tibial Arteries

The anterior and posterior tibial arteries proceed into the foot with the anterior tibial artery becoming palpable as it becomes the **dorsalis pedis artery**. The posterior tibial artery then courses behind the medial malleolus and at this level also becomes palpable. The posterior tibial artery provides both deep and superficial components to the plantar arch. Perforators from the plantar arch provide arterial perfusion to the heel, sole, and branches to the digits.

The anterior tibial artery, which becomes the dorsal pedal artery and is palpable on the dorsum of the foot, eventually communicates with the **plantar arch**, forming a complete circuit in the foot. The peroneal artery, although it stops above the level of the ankle joint, does provide medial and lateral tarsal branches that communicate with the distal-most portions of the anterior and posterior tibial arteries. This is another important collateral pathway for revascularization of the plantar arch in patients with occlusive disease. Vascular surgeons can perform bypass operations to any of these named vessels, with modern procedures successfully bypassing more distal vessels.

Arterial Wall Architecture

The arterial wall typically consists of three laminae. The outer lamina, the adventitia, is a layer of loose connective tissue that provides moderate strength to the arterial wall. The media, or middle layer, contains both elastic and muscular fibers and is responsible for arterial strength, elasticity, and contractility. The intima, the innermost layer, is the endothelial lining of an artery and a few cell layers thick. As the arterial tree descends from the heart to the periphery, muscular functions become more evident. Vessels below the common femoral artery have a greater propensity for rapid vasoconstriction or vasodilation in direct relationship to perfusion. The tibioperoneal vessels can quickly accommodate changes in perfusion by relaxation or dilation.

Arteries are capable of increasing in size (vasodilation) to maintain constant shear stress when atherosclerotic accumulation decreases luminal surface area. However, once a stenosis reaches 50% of the vessel diameter, the artery loses its ability to dilate any further, and any increase in atherosclerotic accumulation impedes arterial perfusion. Further restriction in flow through this stenotic area results in a decrease in the diameter of the artery distal to the stenosis in order to accommodate diminished blood flow. Compliance of an artery decreases as the arterial wall becomes more rigid with calcific atherosclerosis (Fig. 15-2).

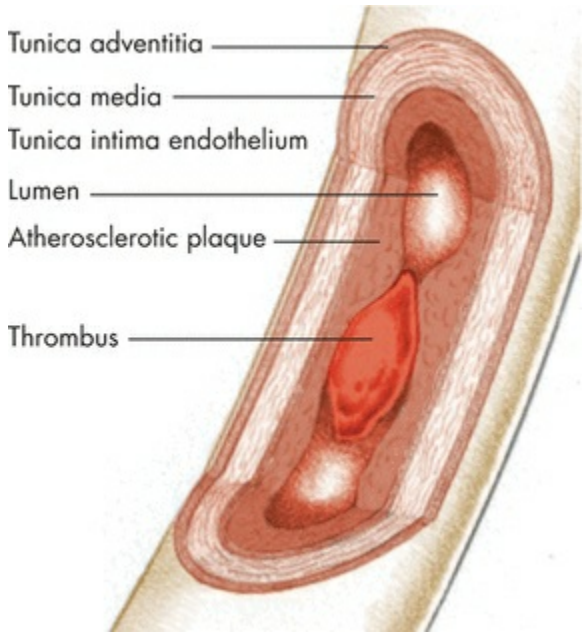


Figure 15-2. Arterial wall. In the layers of the arterial wall shown here, the plaque formation and thrombus significantly reduce blood flow through the vessel.

Arterial Perfusion

As blood descends through successively smaller arterial conduits, it eventually reaches the arteriolar level. Blood flow (rheological factors) in this precapillary bed plays an important role in perfusion. Blood is a non-Newtonian thixotropic fluid (thick or stable at rest but becoming more fluid with motion). The viscosity of blood is inversely proportional to its shear rate or the velocity of blood flow. The slower blood is propulsed, the more viscous it becomes. The primary determinant of whole blood viscosity at any given shear rate is the hematocrit. As red cell mass increases, blood viscosity markedly increases and the flow decreases.

Dehydration and polycythemia (increased number of red blood cells) are two examples of many disease states that increase whole blood viscosity and can result in blood sludging (sticking together of the red blood cells) in the precapillary bed and a resultant decrease in arterial tissue perfusion. In many elderly patients with arterial occlusive disease, even mild dehydration can result in poor extremity perfusion. Simple rehydration can reduce the red cell mass and allow for better perfusion. In other cases of increased blood viscosity, such as multiple myeloma, plasmapheresis may be necessary to remove the abnormal concentrations of proteins. Nonetheless, in the "normal" atherosclerotic patient, it's the red cell mass, measured by hematocrit, that is the primary determinant of viscosity.

As blood proceeds into the capillary bed, the diameter of the vessel approaches that of the red cell—approximately 8 μm (microns) in diameter. Red cells pass through capillaries sequentially. Red cell deformability plays a role in perfusion at this level. In conditions in which the red cell membrane is relatively rigid, tissue perfusion decreases because of increased transit time for a red cell to pass from the precapillary to postcapillary level. Although nutrient and oxygen extraction are increased by this increase in transit time, the per-unit perfusion of the tissues is decreased overall. Medications such as pentoxifylline may facilitate red cell deformability, thereby potentially increasing the per-unit perfusion of tissues.[7,8] Unfortunately, pentoxifylline has limited efficacy in arterial disease, but cilostazol (inhibiting platelet aggregation and an arterial vasodilator) improves both pain-free and maximal treadmill walking distance.[11]

In normal states, arterial tissue perfusion is well above minimal requirements, but certain tissues, such as muscle, can change their metabolic requirements. Muscle becomes more efficient under anaerobic conditions (this is called the Cori cycle)—for example, a trained long-distance runner. The process of improving efficiency is gradual, but training to increase muscle capacity is useful in patients with claudication. A regular exercise

program can increase the distance walked before claudication occurs. The skin does not have the same kind of compensatory mechanism where exercise can gradually increase blood flow.

Arterial Ulcer Pathophysiology

The pathophysiology of vascular ulcers varies according to the type of ulcer. Arterial ulcers are wounds that will not heal due to compromised or inadequate arterial blood flow or ischemia. Hypoxia due to anemia can exacerbate ischemia. Precipitating events for arterial ulcers vary. Limbs with arterial compromise may have minimal but adequate blood flow to maintain tissue viability. Ischemic lower-extremity ulcers are often precipitated by trauma or infection.[12]

The location of traumatic ulcers varies depending on the cause and site of injury, but these wounds are commonly found on the foot or on the anterior tibial area of the lower leg. Traumatic ulcers may be caused by an acute physical injury, such as blunt trauma (e.g., bumping into a piece of furniture or dropping a heavy object on the foot), or by acute or chronic pressure (such as the continual pressure from ill-fitting footwear). Several other conditions may be responsible for tissue breakdown, including thermal extremes, chemicals, or a localized clot or embolus that can also lead to decreased cellular nutrition from impaired arterial flow. *Regardless of the cause, when ischemia is present, wound healing is inhibited.* It is also postulated that the ischemic–reperfusion injury cycle may be more detrimental to ulcer healing than previously suspected and may be even more damaging than prolonged ischemia.[13] Although some wounds heal in the presence of ischemia, arterial inflow must usually be improved for healing to occur.[14,15] Injury repair requires more than baseline oxygen consumption and increased tissue nutritional need. Diminished arterial flow causes tissue hypoxia, and with injury, this will further aggravate the arterial insufficiency and can eventually lead to gangrene or tissue necrosis (Fig. 15-3).

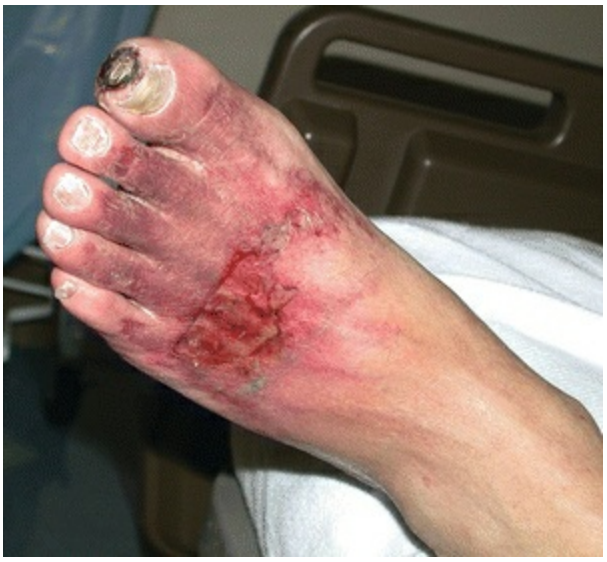


Figure 15-3. Ischemic forefoot. This photograph shows an ischemic forefoot.

Diagnosing Vascular Ulcers

Vascular disease and ulcer etiology can be determined by obtaining a thorough patient history and performing a physical examination. A focused vascular history includes a clear description of the presenting complaint, past medical history for vascular and related conditions, current and previous medications, and risk factors. Symptoms and signs of lower-extremity vascular disease may include pain or loss of protective sensation, tissue compromise, or change in appearance. Noninvasive vascular laboratory testing is required to identify the location of vascular pathology.

The first question to ask every patient is about his or her history of **allergies**. This question is important if there is a known sensitivity to medications that may be ordered or to dyes used for angiography. The next question should be about the patient's **medications** and then about his or her **occupation**.[14]

The important points to remember about a patient's history include remembering the ABCDEs,[14] all of which are increased risk factors for arterial disease. They are:

- **A_{1C}**: Hemoglobin A_{1C} detects the average blood sugar over a period of 90 days. Values below 6% are normal, those between 6.1% and 6.5% represent prediabetes, and those above 6.5% represent diabetes. The higher the HbA1C, the greater the incidence of arterial disease. There is often a personal or family history of diabetes or arterial

disease. Arterial disease often manifests at an earlier age in males and in individuals who smoke or have other risk factors.

- **B**lood pressure: Find out if it is elevated and if the patient is on medications.
- **C**holesterol: Elevated cholesterol is a risk factor, and the use of statin cholesterol-lowering agents may reduce this risk.
- **D**iet and obesity: Increased weight, especially a body mass index above 25, indicates an increased risk for heart and peripheral vascular disease as well as diabetes.
- **E**xercise: Individuals who exercise regularly have a lower risk of peripheral vascular disease and can build up a greater tolerance to overcome compromised circulation. In general, individuals with leg pain at rest or when recumbent in bed have rest pain that equates to severe ischemia, those who have pain or claudication (aching and throbbing calf muscles) with walking up a few stairs or less than 50 yards have moderate disease, and individuals with symptoms after walking one or two blocks have mild disease.
- **S**moking: One cigarette decreases circulation by 30% for 1 hour, and the more pack-years of accumulated smoking history, the greater the risk. Ask patients how many cigarettes they smoke a day and how many years they have been smoking (e.g., 30 years of smoking a half a pack a day is 15 pack-years [30 × 0.5 = 15 pack-years]).

Other risk factors include increased levels of homocysteine and hypothyroidism.[14] If peripheral vascular disease is present, it is also more common to have a history of coronary artery disease and previous stroke.[14]

Physical Examination

Skin inspection, palpation, and auscultation are all an important part of the physical examination. It includes examining the distal extremities for taut or shiny, atrophic skin that's present with arterial disease. Because skin color may indicate arterial perfusion, each toe should be noted and compared with the other foot and toes. Arterial insufficiency causes ischemic tissue to first become pale, progressing to a mottled netlike appearance (livedo reticularis) and subsequently to a dark purple hue and finally black (Fig. 15-4). Elevating the foot at a 45-degree angle causes the ischemic limb to become pale. Immediately after positioning the ischemic foot in a dependent position, it becomes dark red or ruddy (dependent rubor or redness). This finding is the reactive hyperemia of ischemic tissue. There may be a loss of

hair distally, and the nails may lose their luster and become thickened. Make sure to distinguish nail changes from changes that occur with a fungal infection (about half of all abnormal nails), psoriasis, or other nail disorders.

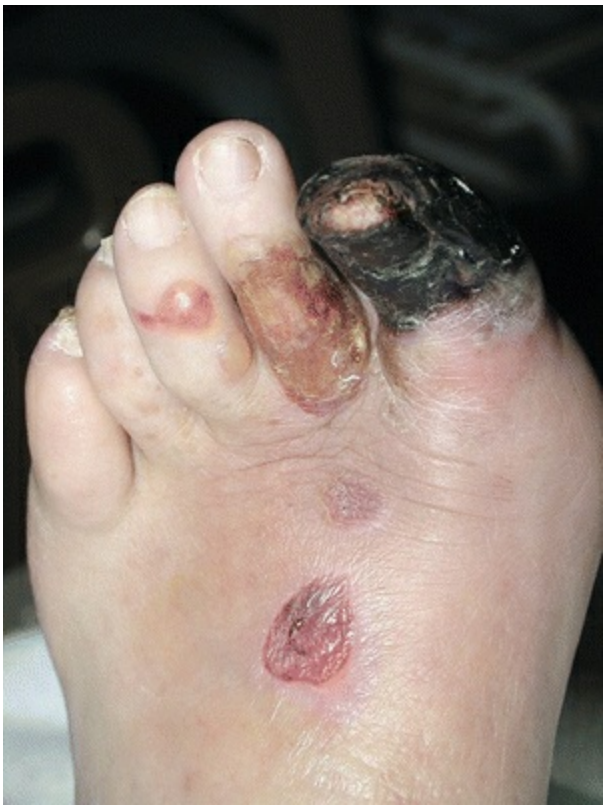


Figure 15-4. Arterial ulcer. This photograph shows a necrotic great toe with blisters on the toes and foot, representing arterial insufficiency.

Palpate the skin for temperature changes. The skin of the distal part of an ischemic limb feels cool or cold, with temperature demarcation that correlates to the diseased artery. Infrared thermometers are now available for under $100 and should be utilized by every wound care clinician.[16] These less costly thermometers have been validated to the gold standard.[16] The thermometer will also identify repetitive trauma/deep inflammation in the neuropathic foot along with deep and surrounding infection.[17]

Capillary refill time is determined by compressing the skin (dorsum of the foot or toe pad) with the thumb to remove the local profusion leading to a local blanching of color. Then, release the thumb to observe the capillary refill and return of color as a good indicator of arterial skin perfusion. Perform this test with the foot slightly elevated. Normal capillary refill time is less than 3 seconds from pallor to normal skin color.

Palpate pulses for presence, rate, regularity, strength, and equality. The most common objective physical finding is the presence or absence of pulses. Care must be taken when palpating pedal pulses. *It's common to mistake a contracting tendon for the presence of a pulse.* No universal consensus exists regarding a pulse grading system. According to the Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II),[18] pulses are graded as 0 (absent), 1 (diminished), or 2 (normal). The American Heart Association's guideline adds a grade of 3 for bounding pulses. To ensure consistency, adhere to local policy. A high degree of interobserver variability exists in determining the presence or absence of pulses. It can be confusing if clinicians report 2+ or 3+ pulse examinations. Communication is better facilitated if pulses are recorded simply as present or absent. However, even this seemingly obvious assessment parameter may not always be accurate. One study found only a 50% chance that two observers would agree with a third observer about the presence or absence of dorsalis pedis or posterior tibial pulses.[18] This same study identified the dorsalis pedis pulse congenitally absent in 4% to 12% of subjects and the posterior tibial pulse absent in 0.24% to 12.8%.[18] Additional descriptors of pulse, such as "weak" or "bounding," can be added to clarify your findings.

Practice Point

The best way to document pulses is to use descriptor terms, such as present or absent, rather than numerical ratings, such as 2+ or 3+. Use modifier words, such as weak or bounding, to further describe and clarify the pulse findings.

Although pulses in the foot may be present at rest, they may disappear with exercise. A patient who presents with claudication but has clearly discernable pulses should have an exercise test performed in the vascular laboratory. Clinicians are often tempted to skip the assessment of the elusive popliteal pulse, particularly when the dorsalis pedis and posterior tibial pulses are strong. While good pedal pulses indicate foot perfusion, finding bounding popliteal pulses may indicate a popliteal aneurysm. Popliteal aneurysms can be a source of emboli to the lower leg with resulting tissue or limb loss.

Patients with foot or leg ulcers should be tested for neuropathy. This is a

common finding in persons with diabetes, but there are several other causes associated with a loss of protective sensation. Approximately 2/3s of patients with neuropathy have diabetes, but the other 1/3 of neuropathies are from other etiologies. For example, neuropathy commonly obscures a traumatic or pressure-induced wound in an ischemic limb. Lack of protective pain sensation and injury awareness prevents the patient with diabetes from seeking appropriate care early. Evaluate neuropathy by testing light touch with monofilaments for the sensory component, examining for dry skin as part of the autonomic component, and eliciting reflexes for the motor component. You can remember to assess for neuropathy with the mnemonic SAM (**S**ensory, **A**utonomic, and **M**otor). An objective assessment of significant neuropathy is best performed by using the 5.07 Semmes-Weinstein monofilament.[19] To perform this assessment, ask the patient to close the eyes and indicate when he or she feels the monofilament. Test the areas over the plantar aspect of the first, third, and fifth toe; the first, third, and fifth metatarsal head; both sides of the plantar aspect of the midfoot; the plantar heel; and, lastly, the dorsum of the foot. Place the monofilament on the test position until it bends slightly, and then move it to the next position. Record the number of negative sites the patient reports; if there are four or more negative sites, then neuropathy is present, which indicates a loss of protective sensation (see chapter 16, Diabetic Foot Ulcers, and Figure 16-2).

Practice Point

Use the mnemonic **SAM** to assess for neuropathy:

Sensory
Autonomic
Motor

Arterial Signs and Symptoms

Arterial insufficiency is commonly associated with complaints of pain[19] resulting from atherosclerotic arterial changes interrupting blood flow to tissues.[20–22] Claudication pain is pain occurring with exercise and is relieved by rest that is usually located in the muscle group distal to the

stenosed or occluded artery. While the calf is the most common location for claudication, it can also occur in the buttocks, thighs, or feet and is predictable and reproducible. Claudication is described by patients as muscle cramping, aching, or weakness. The distance the patient is able to walk until the claudication first develops is referred to as the *initial claudication distance*. The distance the patient is able to walk before he or she has to stop is called the *absolute claudication distance* (Fig. 15-5). The period of time the patient needs for the pain to subside after he or she is forced to stop is referred to as the *recovery time*.

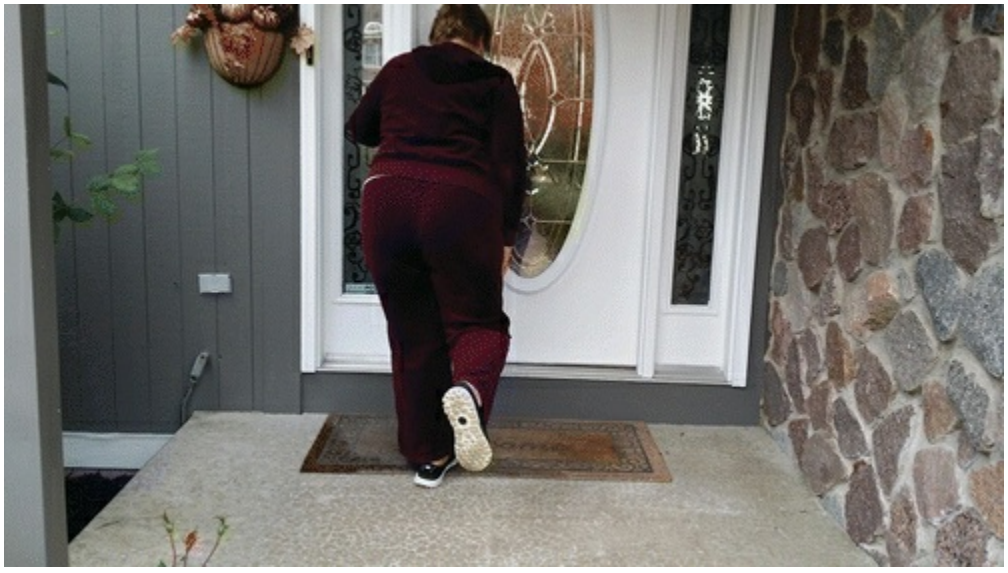


Figure 15-5. Absolute claudication distance.

Practice Point

When taking a history, it's important to find out exactly how far the patient can walk before he or she needs to stop; a shorter distance indicates more severe atherosclerosis. Reported changes in ambulatory distance may indicate progressive atherosclerotic disease.

The patient with leg ulcers and poorly perfused tissue commonly seeks care because of sharp, severe, and possibly constant pain at the ulcer site and the distal extremity. Pain that occurs at rest represents inadequate perfusion and is a sign of threatened limb viability or critical limb ischemia.

This pain is referred to as *rest pain*. The patient may describe waking at night with pain across the distal metatarsal area of the foot. In an attempt to relieve the pain, the patient will get out of bed and lower the foot to the ground that has the effect of gravity to increase blood flow and improve tissue perfusion. The patient may even ambulate. The ischemic pain may be relieved by the small contribution of blood flow from collateral vessels if the limb is placed in a dependent position. The patient with pain at rest may begin sleeping with the legs in the dependent position that will predispose the distal leg to edema accumulation. Elevation of this edematous limb will further exacerbate rest pain, distinguishing it from venous insufficiency and other causes of edema.

Practice Point

Rest pain represents end-stage arterial insufficiency and usually requires revascularization.[15,21]

Patients with extensive sensory neuropathy—for example, those with diabetes—may not experience pain even with severely ischemic ulcers. On the other hand, these patients may experience such intense hyperesthesia associated with the neuropathy that they cannot bear the light touch of stockings. Ulcers in patients with neuropathy are typically found on the plantar side of the foot and are surrounded by calluses from long-term local pressure. These patients may describe the sensations of burning, stinging, shooting, and stabbing pain (neuropathic pain) rather than the more characteristic gnawing, aching, throbbing, and tender pain (nociceptive pain) associated with an acute injury of peripheral vascular disease.

When obtaining a history, note previous arterial surgery for vascular disease, including coronary artery disease and cerebrovascular disease. Vascular disease isn't limited to any one organ but can occur in all body systems: 60% of patients with peripheral arterial disease will have coronary artery disease, and 40% will have cerebrovascular disease. Document all medications, especially vasoconstrictor drugs. Ischemic symptoms are exacerbated by nicotine. Patients with symptomatic vascular disease may aggravate their symptoms by using tobacco, nicotine gum, or nicotine patches for smoking cessation.

Another arterial finding upon examination is gangrene. In ischemic tissue, gangrene initially appears pale, then blue-gray, followed by purple, and, finally, black. Gangrenous tissue eventually becomes black, hard, and mummified. The hardened tissue isn't painful, but significant pain may be present at the line of demarcation between the gangrene and the live but ischemic tissue. Gangrene may be a small skin lesion or extend to an entire limb depending on the location of the arterial lesion. If a small patch of skin is affected, the skin will dry and fall off, producing a skin ulcer or a healed tissue deficit. Large areas of gangrene may require debridement, skin graft, or potential amputation. *Do not attempt this prior to revascularization.*[18]

Additional physical findings are local patches of color change that may appear as small black or dark purple dots, circular areas found on the distal toes, or localized infarcts around the toe nail beds (Fig. 15-6). Tissue changes found in these areas are caused by tissue ischemia from arteriosclerosis or by atheromatous debris embolizing from a proximal artery. Arterial erosions (loss of epidermis with an epidermal base) or deeper ulcers (loss of epidermis with a dermal or deeper base) may also be found between the toes, starting as a small, moist, macerated spot on the skin surface extending deep into the bony structure of the foot. This may also be caused by pressure due to ill-fitting footwear.

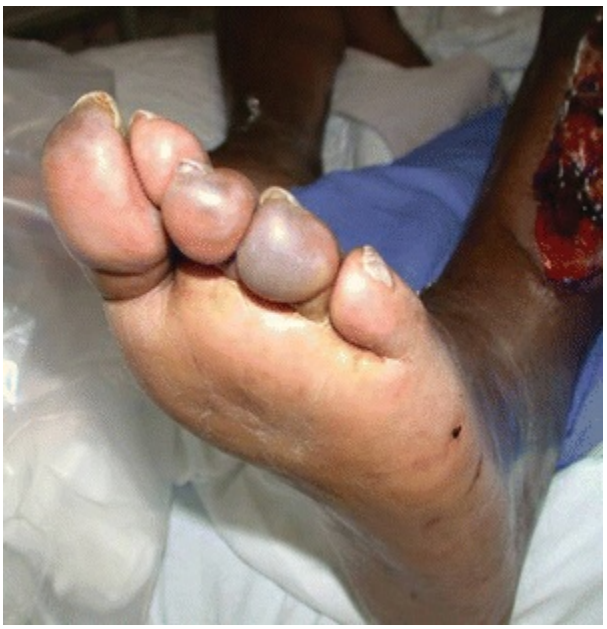


Figure 15-6. Blue toe syndrome. This photograph shows "blue toe syndrome" in the second toe caused by tissue ischemia from arteriosclerosis.

Arterial ulcers typically have distinct borders with a pale-gray or yellow-dry base. They may contain exposed tendons, fascia, fat, muscle,

bone, or joint structures in their base. In contrast, venous ulcers most often have granulation tissue in their base and seldom demonstrate other structures found in the base of an arterial ulcer. The surrounding tissue may appear pale compared with skin elsewhere on the body, or it may be reddened if the leg is dependent. Chronic ischemic skin may appear thin and shiny. Foot elevation will produce skin pallor. The red or ruddy color of a dependent ischemic limb is called *dependent rubor* or *reactive hyperemia* (Fig. 15-7). Even in a person of color, the difference in hue is discernible when the ischemic limb is compared with the contralateral well-perfused limb.

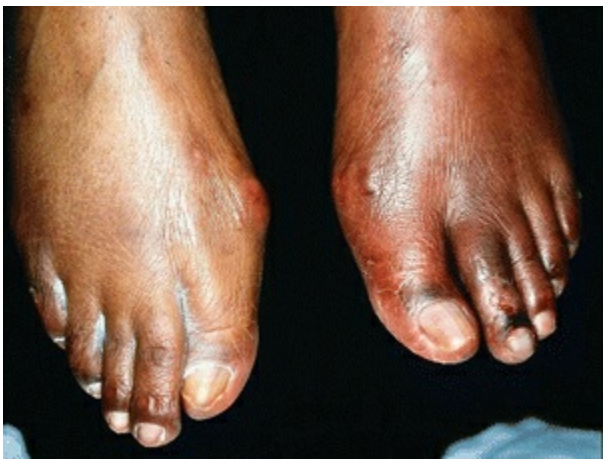


Figure 15-7. Dependent rubor. Foot elevation produces skin pallor in patients with ischemic skin (Buerger's sign or test). When dependent, the ischemic limb will have a red or ruddy color, as shown here in the patient's right leg. This is called dependent rubor or reactive hyperemia (Goldflam's sign).

Arterial pressure is one of the most reliable physical findings in peripheral arterial disease.[21,22] However, lower-extremity blood pressures aren't obtained as a part of the routine physical examination. Bilateral brachial pressures should always be obtained on the initial examination to identify whether a discrepancy exists between them. The correct pressure is always the higher of the two pressures. This pressure is used to determine the ankle–brachial index (ABI) when assessing lower-extremity perfusion.

Vascular Testing

Although an experienced vascular clinician can make a vascular diagnosis based on history and physical examination alone, vascular laboratory studies help pinpoint the diagnosis. The presence, location, and severity of

arterial disease are confirmed by vascular laboratory procedures. Information obtained by vascular studies can predict potential ulcer healing (healable ulcer) when the cause is arterial insufficiency.[7] Laboratory tests differentiate among conditions contributing to a non-healing ulcer.

Noninvasive vascular testing is divided into direct tests that image the vessel itself and indirect tests that demonstrate changes distal to the diseased vessel. These tests include segmental arterial Doppler ultrasound with pressures, arteriogram, ankle–brachial index (ABI), transcutaneous pressure of oxygen ($TcPO_2$), and toe pressures.[23]

Handheld Doppler Ultrasound

A Doppler ultrasound transmitting probe sends a signal that is reflected from an object to the receiving probe. If the signal strikes a moving object such as blood cells, a frequency shift is detected and reflected as sound (Doppler principle). The audible signals of arterial flow patterns can then be determined. The handheld Doppler is used to detect an audible signal on the dorsum of the foot or ankle (dorsalis pedis artery and posterior tibial artery). A blood pressure cuff is then placed around the lower calf and inflated until the audible signal disappears. The cuff is then slowly deflated, and when the signal returns, the systolic pressure is determined from the reading on the cuff gauge.

Arteriogram

An arteriogram is an invasive test used to identify an operative lesion in the arterial system by outlining the patent arterial lumen (Fig. 15-8). Indications for a surgical procedure include incapacitating claudication, rest pain, nonhealing ulcers, and gangrene. An arteriogram is not indicated unless a bypass or dilation procedure is required. It's also not indicated when the patient is too ill for surgery or is refusing surgical intervention.[24]

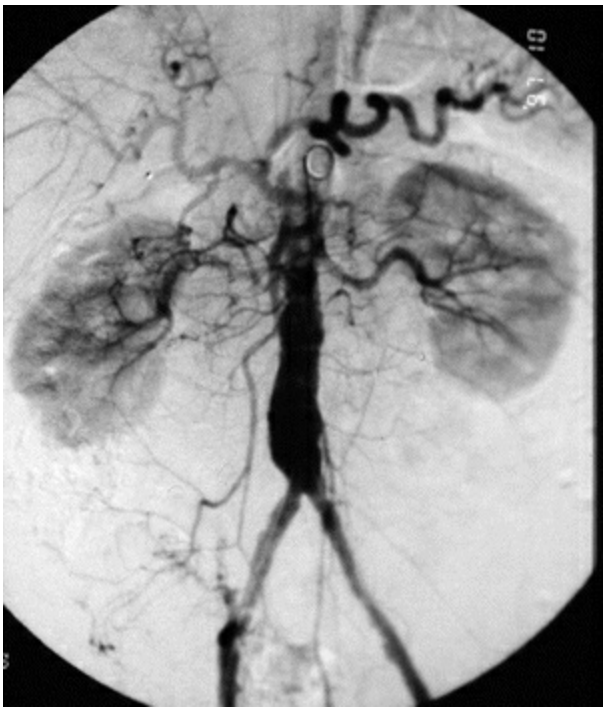


Figure 15-8. Arteriogram. The arteriogram below shows iliac stenosis.

Arterial Testing

Propagation of a pulse wave originating in the heart is easily measured by auscultation of a peripheral artery with Doppler ultrasound. Recording the Doppler shift demonstrates the normal triphasic signal representing the three phases of the pulsation in a normal peripheral artery. The first wave represents forward flow of blood and arterial distention. The second phase represents the arterial relaxation and subsequent retrograde flow of blood. The third phase or portion of the triphasic Doppler signal is believed to represent the bulging of the aortic valve, which occurs during diastole. Some authors suspect that the third phase represents the rebound of the compliant, elastic arterial wall.

The third phase of the triphasic arterial signal is first lost as an artery becomes less compliant and is followed by loss of the second phase of the triphasic Doppler signal. With worsening occlusive disease proximal to the area of auscultation, the normally sharp first wave becomes flattened and broader. In severely diseased arteries, the Doppler signal can be a monophasic, low-amplitude wave. The minimum systolic pressure that can result in forward Doppler flow is used in the calculation of the ABI, a measurement of arterial perfusion in the leg (Fig. 15-9).

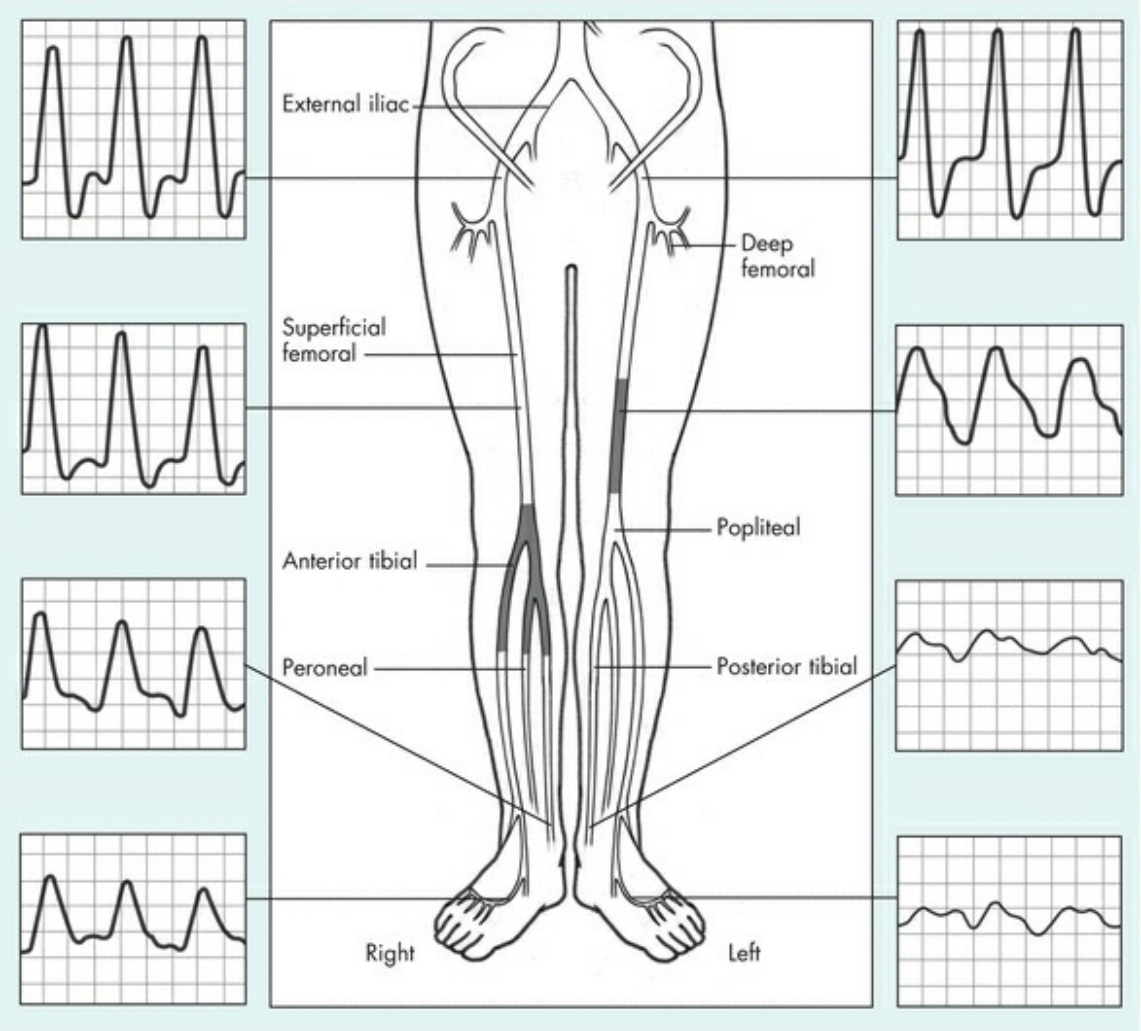


Figure 15-9. Arterial waveform changes. The arterial changes corresponding to occluded arteries are illustrated here.

Ankle–Brachial Index

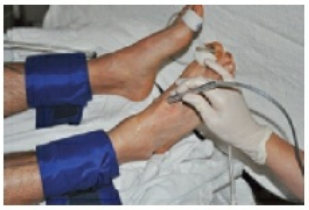
Additional tests for arterial disease include ABI, segmental pressures and waveforms, duplex ultrasound, and exercise treadmill for claudication. Perfusion is indirectly measured by the ABI—the Doppler systolic pressure of the ankle artery divided into the brachial systolic pressure. ABI ratios reflect the degree of perfusion loss in the lower extremity. If the higher leg pressure is 80 mm Hg and the higher arm pressure is 100 mm Hg, then the ankle–brachial pressure ratio (index) is 80/100 mm Hg or 0.80.

In most individuals, the resting ankle pressure in a supine position is equal to or greater than the brachial pressure, with an ABI value of 1 or more. An individual with claudication may have a normal ABI in this position and have it drop during and after exercise. Patients with pain even in the resting state will have an abnormally low ABI (<0.5). With exercise,

the ABI in the patient with rest pain usually doesn't fall because the arteries are already maximally dilated. Inadequate perfusion creates local tissue factors that result in vasodilation. Collateral pathways can't provide the additional tissue perfusion required, resulting in rest pain. A patient with ischemic tissue loss usually has a perfusion picture that is more consistent with rest pain than claudication[7] (Box 15-1).

Box 15-1 Obtaining an Ankle–Brachial Index

To obtain an ankle–brachial index (ABI), a sphygmomanometer and a Doppler device are needed. The procedure is performed as follows:



(Photo courtesy M. Sieggreen.)

- Bilateral brachial Doppler pressures are obtained while the patient is supine. The higher of the two Doppler pressures is used as the brachial pressure in the ratio.
- The blood pressure cuff is placed on the leg just above the malleoli. The Doppler probe is placed at a 45-degree angle to the dorsalis pedis or the posterior tibial artery.
- The cuff is inflated until the Doppler signal is obliterated. With the Doppler probe over the artery, the cuff is slowly deflated until the Doppler signal returns. The number is recorded as the ankle systolic pressure. The higher of the two systolic pressures in each leg is used as the ankle pressure for that leg.
- The higher of the ankle pressures is divided by the higher of the systolic brachial pressures. The ratio obtained is the ABI.

ABI Interpretation

ABI	Interpretation
1.0–1.3	Normal
0.75–0.90	Moderate disease
0.50–0.75	Severe disease
<0.5	Rest pain or gangrene
Unreliable	Diabetes

Segmental Pressures

Segmental pressures have been used since the 1950s to determine the location of arterial vascular lesions.[25] Pressures obtained at the level of the thigh, above the knee, calf, and ankle are compared with each other and with pressures in the other leg. An arterial lesion can be isolated with a 20-mm Hg gradient between cuff pressures. If no pressure gradient exists on a limb that claudicates, the patient is asked to exercise, and repeat pressures are obtained.

A palpable pulse indicates an arterial flow pressure of approximately

80 mm Hg or more in the foot. With calcification of the intima, the arterial pressures derived in the larger vessels of the leg can be falsely elevated. If an ABI is over 1.3, the results are not reliable. The vascular laboratory will then have to rely on accessory tests, such as the toe pressure procedure or $TcPO_2$ test.

Toe Pressures

In patients with severe atherosclerosis (diabetes, chronic renal failure, advanced age), the tibial vessels become circumferentially calcified, which renders them incompressible. Toe pressure tests measure the flow through the large toe where the vessel is small enough that calcium deposits don't circle the entire vessel and compressibility is usually present. A toe pressure of 55 mm Hg or higher, even in a person with diabetes, is usually adequate for healing. Toe pressures of 30 to 55 mm Hg usually indicate some vascular compromise, and the wound healing will be more difficult. Pressures below 30 mm Hg may be adequate if the skin is intact, but as soon as injury results and disrupts the cutaneous barrier, the vascular supply is often inadequate for the repair process.[26]

Evidence-Based Practice

A falsely high pressure reading is commonly seen in patients with diabetes due to incompressible artery walls caused by medial sclerosis of the arteries.[10,26] When the vessels are incompressible, toe pressures are obtained because they're reported to be more accurate.

Treating Vascular Ulcers

When treating vascular ulcers, follow the "preparing the wound bed" paradigm below[26–28]:

- Treat the cause: bypass, stents, or dilation with a consult to a vascular specialist
- Patient-centered concerns: pain, quality of life, and activities of daily living

- Local wound care:
  - Healable wound: debridement, moisture balance, and bacterial balance
  - Maintenance wound: procedures may be more conservative because of patient or system factors causing the wound to not heal
  - Nonhealable wound: requires conservative debridement, moisture reduction, and bacterial reduction

With a healable wound, debridement of slough or nonviable tissue is actively promoted to create a clean wound. This may include careful sharp surgical debridement (with bleeding after debridement) or the use of mechanical, enzymatic, or autolytic debridement methods with dressings (usually alginates, hydrogels, or hydrocolloids).

A maintenance wound—one that could heal but patient factors such as smoking, inconsistent treatment, excessive obesity, or uncontrolled diabetes may make sustained healing less likely—requires conservative, superficial debridement (with no bleeding after debridement), accompanied by local wound care for bacterial and moisture balance. If a wound doesn't have enough blood supply to heal, the surface of the wound or necrotic gangrenous tissue should be allowed to dry and demarcate. This can be facilitated by removing the soft slough around the proximal intersection of the necrotic and viable tissue but leaving the necrotic cap intact. Moisture and bacterial reduction may best be served with antiseptic agents, such as povidone–iodine or chlorhexidine, that usually reduce bacterial counts with acceptable tissue toxicity.[26,29–31] Both of these agents have a broad spectrum of action, a sustained residual effect, and acceptable tissue toxicity for this indication. Agents such as sodium hypochlorite, quaternary ammonium agents, and various aniline dyes (mercurochrome) have higher cellular toxicities and more limited antibacterial effects.[26,29–32]

Wound Infection

It's important to remember that all chronic ulcers contain bacteria (contamination). When bacteria are attached to tissue and multiply, they become colonized and can lead to damage that delays healing (such as with critical colonization, increased bacterial burden, covert infection, and superficial infection). Patients with critical colonization don't have all the classic signs and symptoms of a deep tissue infection. Infection can be diagnosed with a bacterial swab that helps identify resistant organisms or serves as a guide to antimicrobial therapy. The bacterial swab will not

identify organisms or their sensitivities in a biofilm.

The superficial compartment of the wound bed should be examined for more than one sign of bacterial damage. The key features of a wound bed can be remembered by the NERDS mnemonic[27,29] (see NERDS© and STONEES© in Chapter 7, Wound Bioburden and Infection).

Topical treatment for healable wounds that are critically colonized wounds could include the various new silver dressings, cadexomer iodine, or two nonreleasing foams-polyhexamethylenebiguanide (PHMP) and methylene blue/crystal violet. Povidone–iodine or chlorhexidine may be considered for wounds with inadequate blood supply to support healing.

Deep tissue infection requires systemic antimicrobial agents. The classic signs of warmth, tenderness, swelling, and erythema can be supplemented for persons with chronic wounds by the mnemonic STONEES[27,29] (see NERDS© and STONEES© in Chapter 7, Wound Bioburden and Infection). If exudate and odor are present, other criteria are needed to determine whether the infection is superficial or deep.

Practice Point

- The diagnosis of superficial or deep and surrounding tissue infection can be made clinically with a bacterial swab to help with treatment decisions.
- Persons with diabetes, neuropathy, and foot ulcers often have a false-negative X-ray of the foot for osteomyelitis, and other criteria, such as probing bone, should be used to make a diagnosis of osteomyelitis.
- The use of a bone scan is limited because it is expensive and may give false-positive results in the presence of inflammation. Magnetic resonance imaging (MRI) may be more helpful diagnostically.

Wound Cleansing

A clean wound, free from dead tissue and wound debris, is necessary for healing to occur. Many commercial wound cleaners have some cytotoxicity, but they have surfactant properties that are often useful. Povidone–iodine,

chlorhexidine, hydrogen peroxide, and 0.25% acetic acid have been shown to interfere with fibroblast formation and epithelial growth.[26,27] The selective use of these agents, particularly povidone–iodine and chlorhexidine, should be reserved for wounds that don't have the ability to heal or for time-limited use in wounds in which bacterial burden is more important than cellular toxicity.[26]

Evidence-Based Practice

The advantage of wound cleansing should be weighed against damaging new tissue growth.[32]

The safest wound cleanser is 0.9% saline solution or water. Wounds should be cleaned with a force strong enough to dislodge debris but gentle enough to prevent damage to newly growing tissue. The pressure to accomplish this goal ranges from 4 to 15 pounds per square inch (psi).[33] A 19-gauge needle or 19-gauge angiocatheter distributes approximately 8 psi when used with a 35-mL syringe. A commercial cap on a saline irrigation bottle is a less expensive method to distribute adequate pressure. Hydrotherapy or whirlpool debridement has been used to aid in cleaning and debridement of arterial ulcers. However, the risk of infection outweighs the benefits of whirlpool for debridement and is not currently recommended.[34] This may suggest that vigorous irrigation may be a significant factor in cleansing the wound, but the potential harm must be considered along with the benefits. (For more information about wound cleansing, see Chapter 7, Wound Bioburden and Infection.)

Dressings chosen for specific wounds depend on the wound bed condition and the goal for the wound. Many new dressings are designed to support moist wound healing (see Chapter 9, Wound Treatment Options). Because skin is fragile in patients with either arterial or venous disease and can be easily injured, tape and adhesive products should be used with extreme caution. The use of methods to secure dressings that minimize skin injury is recommended. Of the available adhesive products, soft silicones are less likely to cause local trauma during dressing changes. Products without adhesives that secure dressings are the first choice when they are available.

Practice Point

The preferred attachment device for dressings on vascular leg ulcers is a gauze roll or commercial devices (such as netting or tube gauze) that hold dressings in place without adhesive that can damage fragile skin.

Arterial Ulcer Treatment

Treatment of arterial ulcers includes increasing the blood supply to the area. Positioning the extremity in a dependent position may facilitate blood flow by gravity through collateral vessels. Use caution if devices such as a foot cradle are used for protection because an insensate foot is subject to trauma from the cradle's hardwood or metal. Debridement of nonviable tissue should not be performed in the presence of ischemia because the blood flow is insufficient to heal the new surgical wound. Ulcers without adequate arterial inflow must be kept dry—in contrast to the principle of moist wound healing for ulcers with adequate blood supply. Moisture provides a bed for bacterial growth if eschar, slough, or gangrenous tissue is present. This tissue, if kept dry, can be left in place until demarcation or debridement is indicated.

Ulcers with adequate blood supply that are expected to heal should be dressed with products that support moist wound healing principles.[26] These dressings include hydrocolloids, thin films, foams, and, if nothing else is available, moist saline gauze. The surrounding intact tissue should be protected from fluid accumulation, which can macerate the healthy skin at the ulcer border. Consider a barrier including film forming liquid acrylates, petrolatum, zinc oxide, and windowed dressings.

Arterial reconstruction is the treatment of choice to improve the circulation for most patients.[20] Treatment for arterial leg ulcers requires re-establishing arterial inflow before any other treatment is established. This is usually preceded by a noninvasive vascular test, an arteriogram (computerized tomography angiogram, magnetic resonance angiogram, digital subtraction angiogram) followed by angioplasty, and/or surgery. Simultaneously, local ulcer treatment can be determined. Usually, the arterial ulcer has a dry ulcer bed. The patient may have several punctate ulcers with regular borders as well as dry eschar or gangrene distal to the most perfused tissue—usually the tips of the toes or an entire toe. This

tissue must be kept dry until adequate arterial perfusion occurs. Moistened gangrenous tissue can provide a medium for bacterial growth (Fig. 15-10).

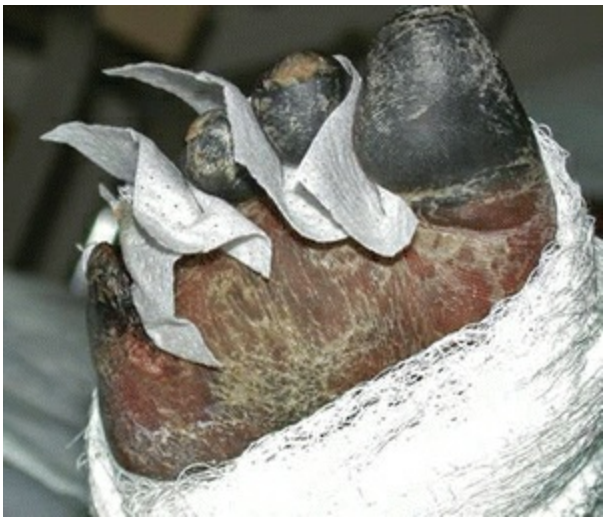


Figure 15-10. Keeping gangrene dry. Gangrenous tissue must be kept dry until adequate arterial perfusion is restored to the area. In the photograph below, the necrotic toes are left open to the air with alcohol wipes placed between them to promote drying.

Surgical Treatment for Arterial Ulcers

Surgical treatment should be considered when patients have incapacitating claudication, rest pain, nonhealing ulcers, or progressive gangrene and infection that cannot be controlled. For arterial ulcers, surgical treatment is aimed at restoring tissue perfusion. Bypass grafting may be performed using autologous veins or, when autologous veins are not available, prosthetic grafts, either reversed or in situ. Despite the fact that endovascular techniques are not superior to surgical techniques with regard to vessel patency, wound healing and limb salvage can be attained by using endovascular techniques for patients previously considered ineligible for revascularization. There are generally less sustainable long-term results from percutaneous balloon angioplasty and stent insertions, atherectomy (percutaneous endoluminal removal of atherosclerotic plaque),[35] and laser ablation of atherosclerotic lesions,[36] except in the common iliac arteries. However, these minimally invasive procedures may be very useful in the high-risk patient, and they expand treatment options.

Large arterial ulcers may require skin grafting to close the defect, but this can only be successful if combined with revascularization procedures. The BASIL (Bypass Versus Angioplasty in Severe Ischemia of the Leg)

trial,[37] which compared bypass surgery and angioplasty, clearly showed that bypass surgery was superior in achieving amputation-free survival. Also, those patients who underwent bypass surgery first fared better than those who underwent angioplasty first. However, this superiority was not significant until after 2 years. The BASIL trial[37] also showed that autologous veins were superior to prosthetic conduits for these bypasses. Unfortunately, the trial did not include the much more common practice of hybrid procedures, combined bypass surgery, and endovascular intervention. Nevertheless, it reinforces the long-held concept in limb salvage surgery that being aggressive is usually better for the patient.

The treatment of ulceration due to arterial insufficiency depends on the level that the occlusive disease occurs. Surgeries for arterial insufficiency are generally grouped into three major areas:

- Aortoiliac bypass
- Femoropopliteal bypass
- Distal bypass

Restoring Tissue Perfusion

Occlusive disease in many patients is multileveled. The rule of thumb is to improve inflow first in these patients and then, if necessary, perform an outflow procedure. Inflow usually involves the aortoiliac segments. The exact surgery is tailored to the individual patient's physiologic status and need. For example, an elderly, frail patient with severe aortoiliac occlusive disease may not be a candidate for an aortobifemoral bypass graft. In this type of patient, an extra-anatomic axillobifemoral bypass graft is considered. By avoiding intra-abdominal surgery and clamping of the abdominal aorta, the overall morbidity for these surgeries can be reduced. However, the trade-off for this is that an axillobifemoral bypass graft generally doesn't have the long-term patency rates than an aortobifemoral bypass graft does.

Percutaneous Balloon Angioplasty

The development of percutaneous balloon angioplasty, with or without stent placement, has significantly reduced the need for routine aortobifemoral bypass surgery in patients with aortoiliac occlusive disease.[38] Isolated short-segment stenoses can be treated successfully with balloon angioplasty. Short-segment stenoses are generally defined as those less than 10 cm in

length, commonly less than 5 cm. With advances in stent development, acute occlusions occurring as a result of atherosclerotic plaque rebound have decreased. The long-term patency rate for stents approaches that for arterial bypass, but only in the aortoiliac segments.[39] Good long-term outcomes with bare metal stents are still haunted by the occurrence of in-stent restenosis. The development of covered stents, with graft material (usually polytetrafluoroethylene [PTFE]) either on one side of the stent metal or enclosing it completely, may hold the answer.[40] Infrainguinal balloon angioplasty with or without stent placement is still inferior to surgical intervention. However, this procedure still holds a place in the treatment of high-risk patients.

According to the TASC II Guidelines,[18] when possible, arterial reconstruction by means of endovascular techniques should be considered before more invasive surgical techniques. Percutaneous intraluminal balloon angioplasty and stenting are considerations for arterial stenoses and occlusions classified as TASC A or B lesions. TASC C and D lesions are usually longer and more extensive and often require bypass operations. Newer hybrid procedures include a combination of open surgical and endovascular techniques.[18]

Femoropopliteal Bypass Graft

A femorol-popliteal bypass graft is the standard treatment for femoropopliteal disease. In contrast to an aortoiliac bypass, where the bypass conduit is that of a synthetic material, the femoropopliteal segment may have either a prosthetic conduit or an autogenous venous conduit. The patency rate for a bypass of the femoral popliteal segment depends upon the choice of conduit and the distal level of the bypass. In an above-knee femoropopliteal surgery, the patency rate between autogenous vein and prosthetic material has no significant difference, although long-term patency rates are better when an autogenous venous conduit is used. In a below-knee femoropopliteal bypass, prosthetic material is far inferior to that of autogenous venous conduits.[41] An autogenous venous conduit should be used in the below-knee position whenever possible (Table 15-1).

Table 15-1 Graft Patency Rates

Type of Graft	1 Year	2 Years	3 Years	4 Years
ABOVE-KNEE FEMOROPOPLITEAL GRAFTS				
Reverse saphenous vein	84%	82%	73%	69%
Polytetrafluoroethylene (PTFE)	79%	74%	66%	60%
BELOW-KNEE FEMOROPOPLITEAL GRAFTS				
Reverse saphenous vein	84%	79%	78%	77%
PTFE	68%	61%	44%	40%
Limb salvage				
Reverse saphenous vein	90%	88%	86%	75%
Reverse saphenous vein	94%	84%	83%	
INFRAPOPLITEAL GRAFTS				
Reverse saphenous vein	84%	80%	78%	76%
PTFE	46%	32%	21%	82%
Limb salvage				
Reverse saphenous vein	85%	83%	82%	48%
PTFE	68%	60%	56%	
AT OR BELOW-ANKLE GRAFTS				
Reverse bypass vein	85%	81%	76%	
In situ vein bypass	92%	82%	72%	
Foot salvage	93%	87%	84%	

This chart shows the percentage of grafts that remain patent after 1, 2, 3, and 4 years.

Below-knee femoropopliteal bypasses using veins have a higher patency rate than above-knee femoropopliteal bypasses because a certain amount of atherosclerotic disease at the level of the knee joint can be missed if only anteroposterior arteriography views are obtained. For this reason, many vascular surgeons require oblique views of the popliteal artery so as to preclude this as a source of decreased long-term patency rates.

Distal Bypass

A distal bypass, below the tibial peroneal trunk, requires an autogenous venous conduit. It's reserved for patients with tissue loss when pulsatile arterial perfusion to an ischemic area is desired. Although somewhat controversial, either a reversed venous bypass or an in situ bypass can be performed. Patency rates were equivalent in large series comparing these two techniques.[41] The in situ technique is generally reserved for patients with considerable size disparity between the proximal and distal venous conduit, such as the greater saphenous vein. An in situ bypass is technically

more demanding and requires more operative time than a reversed venous bypass. Some vascular surgeons advocate the creation of a controlled arteriovenous fistula in order to promote long-term patency rates of the prosthetic conduit, when these are used for distal bypasses. The BASIL trial would disagree with this (see Surgical treatment for arterial ulcers section).

A patient with calf claudication requires improved perfusion to the posterior calf muscles. Claudication can occur in the buttocks, thighs, or isolated compartments of the lower legs. The perfusion of the respective symptomatic musculature is what determines the level of the outflow portion of the bypass. In patients with combined aortoiliac superficial femoral popliteal disease, 90% of the claudication can be improved by merely improving the inflow to the profundal system by some form of aortoiliac bypass. It is for this reason that routine combined aortofemoral and femoropopliteal bypasses should be avoided. In patients with lifestyle-limiting claudication with isolated superficial femoral artery disease, a femoropopliteal bypass is usually all that is needed.

Patients with ischemic tissue loss usually require pulsatile arterial flow to heal their lesions. If these lesions are in the foot, then whatever bypass is necessary to restore pulsatile arterial flow to the affected area should be performed. Combined with the appropriate vascular bypass procedure, an area of ischemic tissue loss with gangrenous edges should be debrided to create viable tissue. In some patients, however, if there is dry gangrene, autoamputation can be anticipated once adequate perfusion is restored. Some clinicians allow the gangrenous eschar to autoamputate to enable normal epithelial coverage of the underlying eschar before eschar separation. If, however, the area of tissue loss involves a digit, amputation with primary closure may be recommended if no infection is present. This can be performed in conjunction with the vascular bypass procedure, or the procedures can be separated by several days if deemed appropriate.

Arterial reconstruction with an in situ graft may be used to revascularize the lower extremity well below the knee. An in situ graft is a vein left in its natural location, anastomosed to the arterial system above and below the arterial stenoses, after the valves are lysed. This procedure allows the surgeon to reconstruct the smaller distal arteries in the lower extremity near the foot. These reconstructed vessels are close to the skin surface. The surgeon must use extreme care not to cause injury to underlying vessels when using sharp debridement for the necrotic ulcers. Autolytic debridement is a safer debriding alternative.

Medical Treatment

Medical treatment of arterial disease may include antiplatelet drugs, such as aspirin or clopidogrel, which inhibit the binding of adenosine triphosphate (ATP).[42] Clopidogrel was shown to be slightly better than aspirin in a comparative study. In addition, cilostazol[43,44] has been used not only to decrease platelet aggregation but also to act as a vasodilator that may facilitate an increase in exercise capacity. However, it cannot be used in patients with heart failure. In addition, building up exercise tolerance with a conditioning program may also be important.

Measuring Healing

Calculating healing rates is problematic when no standard measurement for wound healing parameters exists. Following wounds to complete healing is one method, but this method is not satisfactory if changes in therapy are needed. Healing rates can be expressed as percent of ulcer area, measurement of change in ulcer perimeter, or percent of ulcer area healed. However, the perimeter and surface area are much greater in large ulcers. Using these measurements will give erroneously high healing rates for larger ulcers compared with smaller ulcers. For example, if the percent of ulcer healed is used as a measurement, smaller ulcers will appear to heal faster than large ulcers by comparison.

Another method traces ulcers on a celluloid screen and then measures them over time. The area and circumference of the tracing are calculated by a computer program. In general, a healing trajectory will be established if a wound is 20% to 40% smaller by week 4 and that wound should heal by week 12 provided that the same healing rate is maintained.[45]

There are some patients whose ulcers do not heal at the expected rate. If tissue damage has progressed beyond salvage, surgery is too risky, or the limitations of the ischemic limb are interfering with quality of life, amputation may be considered.

Patient Education

The patient may inadvertently neglect the ulcer or fail to use preventive measures if the nature of the condition is not understood. Patient education includes the reason for the ulcer and the treatment rationale. Patient-centered concerns should be central to the treatment process, and active patient involvement includes the recognition and reporting of changes that

indicate problems with healing. Patient and family education includes assessment of patient and family needs and level of comprehension about the arterial ulcer and its etiology. Teaching methods vary and are chosen specifically to facilitate the most appropriate method for each patient and family.

Risk Factors

Factors that increase risk for arteriosclerosis include smoking, diabetes (with elevated HbA1C), hyperlipemia, and hypertension.[44] Smoking is a risk factor in 73% to 90% of patients with atherosclerotic arterial disease.[44] Up to 30% of patients with arterial disease are reported to have diabetes,[46] and 16% to 58% of patients with diabetes have arterial disease.[47,48] Hypertension is present in 29% to 39% of patients with atherosclerosis, and 31% to 57% of patients with atherosclerosis have hyperlipidemia.[49] Risk factor modification is part of the treatment for vascular ulcers to reduce the possibility of further breakdown.

Patients can help themselves by positioning and reducing activities that impair blood flow. After a surgical or percutaneous intervention to restore arterial flow, the patient should continue behaviors that promote vascular health and reduce risk factors (see Patient Teaching: Teaching about arterial ulcers).

Patient TeachingTeaching About Arterial Ulcers

Teach the patient with an arterial ulcer to:

- monitor arterial or graft patency by palpating pulses
- recognize signs and symptoms of graft failure and what to report
- avoid nicotine in any form, including secondhand smoke
- begin or maintain a regular exercise program
- manage blood glucose, if diabetes is present
- control hyperlipidemia
- manage hypertension
- reduce weight, if indicated

- perform meticulous foot care
- manage ulcer care.

Smoking Cessation

Smoking cessation is critical for patients with arterial insufficiency. The direct relationship between tobacco use and ischemia is well known. Smokers are more likely to develop claudication than nonsmokers. However, the link between smoking and vascular disease isn't well recognized by many patients. In one study, only 37% of smokers with peripheral vascular disease understood the strong association between smoking and vascular disease.[44] Patients must be informed of the negative effects of smoking on the vascular system. They should be referred to smoking cessation specialists if needed. Teach the patient the ABCDE mnemonic to remember the risk factors of arterial disease.[10]

Summary

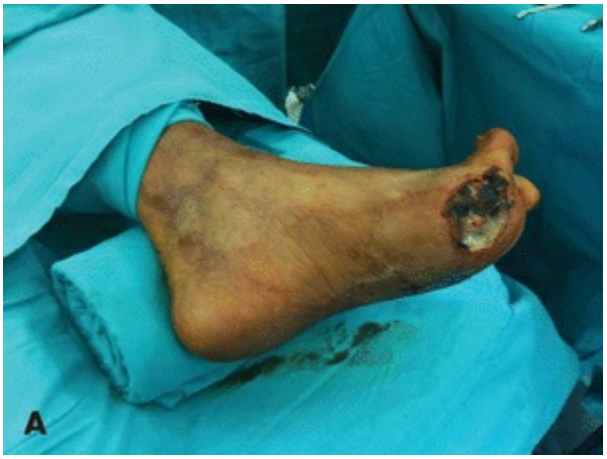
Success in managing arterial ulcers requires a total patient commitment. Risk factors and ulcer management are so dependent upon the patient's activities that the patient must have as much information as possible to actively participate in the treatment process. An understanding of the peripheral vascular blood supply and the need for adequate tissue oxygenation is critical to the management of arterial ulcers in the legs and feet.

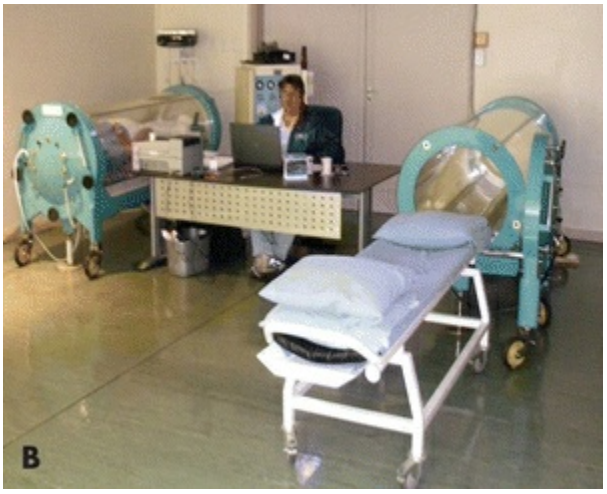
Arterial reconstruction is the hallmark of treatment for arterial disease. In general, dry arterial ulcers or those with fixed, stable, dry eschar should be kept dry until the tissue is revascularized. Economic concerns make it imperative to choose the appropriate dressings and treatment. Research demonstrates little increased healing rate benefit of the newer topical wound treatments over the old gauze dressings. Some modern wound dressings provide other cost-effective benefits along with decreased nursing time, improved quality of life, and reduced pain issues.

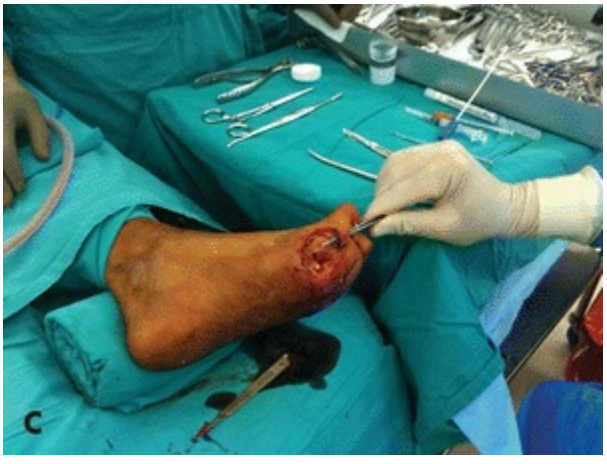
PATIENT SCENARIO

Clinical Data

Mr. W. G. was an 82-year-old Caucasian male who presented with a foot ulcer (Fig. 15-11A). His past medical history was significant for insulin-dependent diabetes, hypertension, chronic renal disease but not on dialysis, chronic obstructive airway disease, severe ischemic heart disease, and extensive peripheral arterial disease. He was a previous smoker but did not smoke at the time of the assessment.







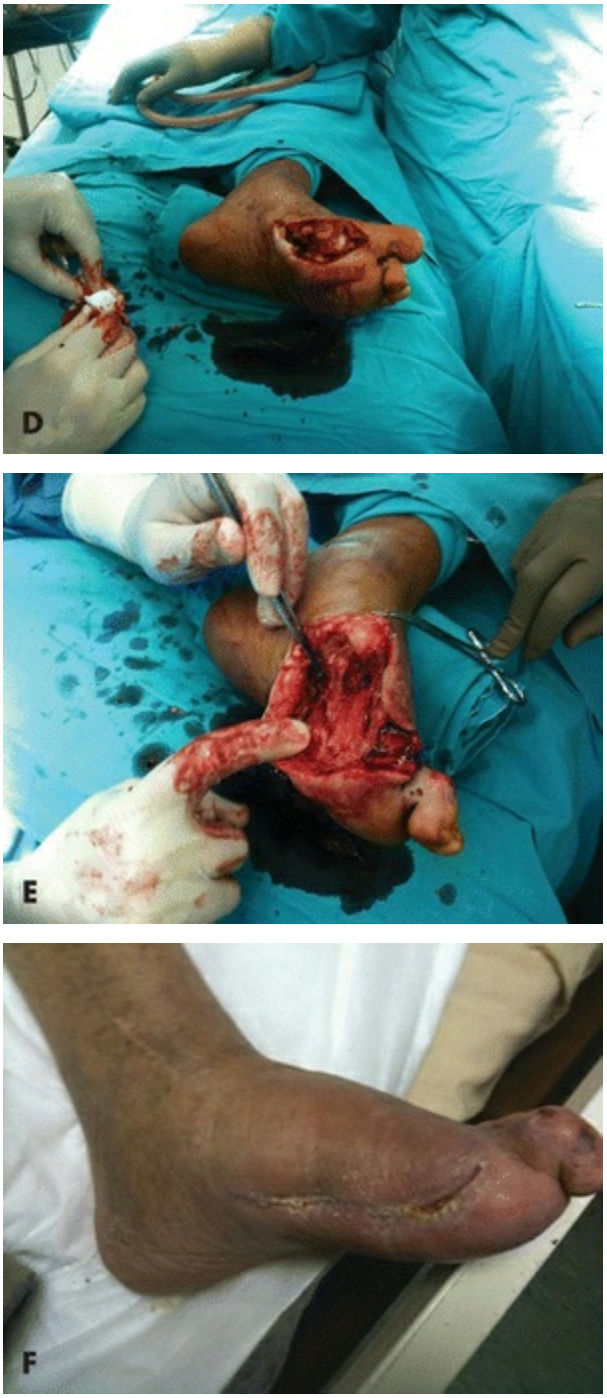


Figure 15-11. (A) Arterial foot ulcer. **(B)**Photo of monoplace hyperbaric chamber. **(C–E)**Progression of surgical debridement. **(F)** Healed arterial foot ulcer. (Photos courtesy Mary Y. Sieggreen, NP.)

His surgical history included two previous coronary bypass procedures. It was determined that he would need a femoropopliteal bypass procedure and his lesions were not amenable to endovascular intervention. He was evaluated by an anesthesiologist and a cardiologist and was determined to be too high a risk for general

anesthesia because of his cardiac status.

The option of hyperbaric oxygen therapy was considered, and he was in agreement with this plan (Fig. 15-11B). His $TcPO_2$ levels increased from resting pressures of 5 mm Hg (on room air) to 28 mm Hg after 15 minutes on a nonrebreather oxygen mask.

Debridement of his foot was done during his hyperbaric oxygen therapy (Fig. 15-11C–E). After 15 sessions of hyperbaric oxygen therapy, his $TcPO_2$ at rest increased to 23 and 45 mm Hg on a nonrebreather mask. He was able to return to his own home with intensive wound care performed by a wound care practitioner and home oxygen therapy.

On subsequent follow-up visits, he reported a remarkable improvement of his foot and quality of life (Fig. 15-11F). Unfortunately, he passed away, due to his underlying cardiac disease, within a year but avoided amputation and was able to function within his home.

Show What You Know

1. Risk factors for the development of arterial ulcers include all of the following except:

A. smoking.
B. hypercholesterolemia.
C. diabetes mellitus.
D. varicose veins.
E. hypertension.

2. Patients with arterial ulcers that do not have adequate blood supply to heal should have local wound care that includes:

A. aggressive local debridement to bleeding tissue.
B. silver dressings that promote moisture balance.
C. local antiseptics such as povidone–iodine and chlorhexidine.
D. moisture balance dressings such as a hydrogel.

3. Which of the following is most likely to be associated with an arterial ulcer?

A. Lipodermatosclerosis
B. Reduced blood flow

C. Edema
D. Systemic hypertension
E. Diabetes mellitus

4. The ankle–brachial index (ABI) is an indicator of loss of perfusion in the lower extremity.

A. True
B. False

5. Surgical treatment for arterial ulcers most commonly includes:

A. a graft.
B. valvoplasty.
C. a bypass graft.
D. phlebectomy.

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Diabetic Foot Ulcers

16

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Objectives

After completing this chapter, you'll be able to:

- state the significance of foot ulcers in patients who have diabetes mellitus
- list strategies for preventing foot ulcers in patients with diabetes
- describe wound characteristics and assessment parameters for a patient with diabetes
- list options for reducing pressure for a patient with diabetes who has a foot ulcer
- discuss the therapy options for patients with a diabetic foot ulcer.

Diabetes: A Growing Problem

Diabetes mellitus is a chronically metabolic disease with increasing prevalence worldwide.[1] The American Diabetes Association (ADA) defines diabetes as "a disease in which the body doesn't produce or properly use insulin." Diabetes mellitus is not only a major health crisis in the United States, with an increase in the incidence by 48% over the past 10 years, but also now a pandemic. However, of the 20.8 million Americans (7% of the population) who have diabetes, only 14.6 million are diagnosed,

leaving more than one-third unaware they have the disease. Blacks, Hispanics, Native Americans, and Asian-Americans have the highest prevalence of diabetes mellitus.[2]

Foot problems are one of the most common complications of diabetes that lead to hospitalization.[3,4] Admissions for foot complications account for 20% to 25% of all hospital days for patients with diabetes.[4] Between 2% and 6% of patients with diabetes will develop a foot ulcer every year.[5]

Diabetes has become the most common underlying cause of lower-extremity amputation in the United States. Recent data from the Centers for Disease Control (CDC) show an annual number of 111,000 hospitalizations for the diabetic foot in 2003, thereby surpassing the number attributed to peripheral arterial disease (PAD).[6] The annual rate of amputations in the United States has almost halved in the past decade, and most of this decrease has been in the above-ankle amputations.[7] The risk of lower-extremity amputation in people with diabetes is 15 to 46 times higher than in nondiabetic patients.[4] After the initial amputation, the risk of reamputation or amputation of the contralateral extremity is also high: 9% to 17% of patients will experience a second amputation within the same year.[4,8] The incidence of lower-extremity amputation is 1.5 times higher in Hispanics and 2.1 times higher in African Americans compared with non-Hispanic Whites[9] (Box 16-1).

Box 16-1 ADA Contact Information

The American Diabetes Association (ADA)[2] offers much information for diabetic patients and their families as well as for healthcare professionals. General information about diabetes is available, along with advice on exercise, nutrition, and daily meal planning. To contact the ADA:

1701N. Beauregard Street
Alexandria, VA 22311
1-800-DIABETES
http://www.diabetes.org

Etiology of and Risk Factors for Foot

Ulcers

A number of local and systemic risk factors for foot ulceration and amputations should be considered in the prevention and treatment of the diabetic foot. Perhaps the strongest and easiest risk factor to identify is the presence of a previous ulceration or amputation, which indicates the potential for recurrence due to scar formation or biomechanical abnormalities. The underlying pathology usually is not reversible, and most disease processes affecting the diabetic foot will continue to worsen over time. Three primary pathways or mechanisms of injury have been identified in the development of foot ulcers. These include wounds that result from ill-fitting shoes (low-pressure injuries that are associated with prolonged or constant pressure), ulcers on weight-bearing areas (repetitive moderate pressure and shear forces on the sole), and penetrating injuries from puncture wounds or other traumatic events (high-pressure injuries with a single exposure of direct pressure).[10]

Peripheral Sensory Neuropathy

Diabetes affects sensory, motor, and autonomic nerve function. In patients with sensory neuropathy, pain—the primary natural warning system that alerts the body to take action and seek medical care—is defective. Sensory neuropathy contributes to an inability to perceive injury to the foot due to what is commonly referred to as loss of protective sensation (LOPS).[11] LOPS represents a level of sensory loss where patients can injure themselves without recognizing the injury. Motor neuropathy contributes to wasting of the intrinsic muscles of the foot; muscle imbalance; structural foot deformity, such as claw toes and subluxated metatarsophalangeal joints; and limited joint mobility. Autonomic neuropathy causes shunting of blood[12] and loss of sweat and oil gland function, which leads to dry, scaly skin that can easily develop cracks and fissures. The combined effect of these neuropathies results in a foot with structural deformity and biomechanical faults; dry, poorly hydrated integument; and an inability to respond to pain and repetitive injury.

Neuropathy is one of the most common risk factors for lower-extremity complications. It is unusual to see a patient with a foot ulcer who does not have sensory neuropathy.[13] Several screening methods can be used to identify sensory neuropathy, including a systematic clinical examination, vibration perception threshold (VPT) testing with a VPT Meter, and

pressure assessment with Semmes-Weinstein monofilaments[14] (Fig. 16-1). Although these methods are noninvasive and have good sensitivity and specificity to identify patients with LOPS,[15] Semmes-Weinstein monofilaments in particular may present several problems, which should be considered before using the device. Semmes-Weinstein monofilaments should be purchased from a vendor that sells *calibrated* instruments because considerable variability exists among different brands of monofilament.[16] Booth and Young found that some brands of monofilaments buckled at 8 g of force rather than at the 10 g for which they were designated. In addition, the material properties of the monofilament wear out after repetitive testing. Young et al.[17] found that after 500 cycles of testing (or the equivalent of testing 10 sites on each foot for 25 patients), there was an average reduction of 1.2 g of testing force. A worn-out monofilament may result in patients being diagnosed as having sensory neuropathy with LOPS when they are not at risk.

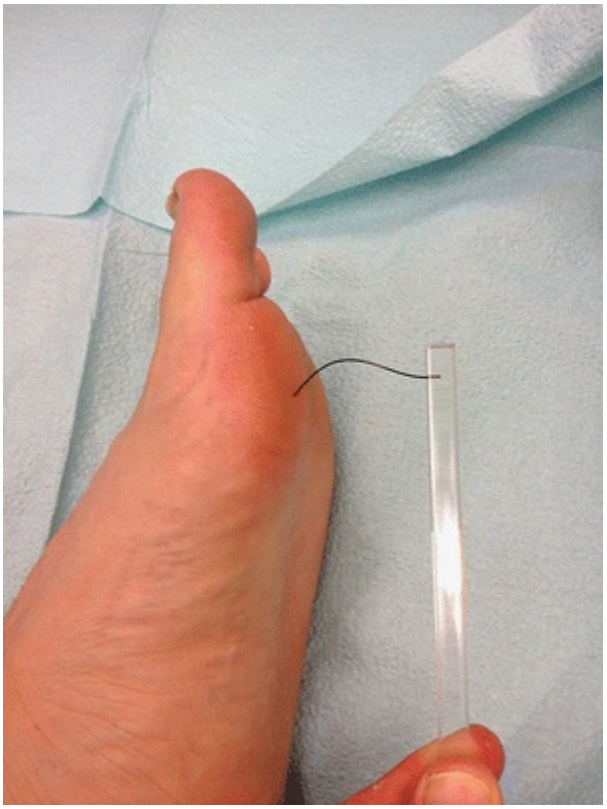


Figure 16-1. Semmes-Weinstein monofilament exam. (Photo courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

Practice Point

A Semmes-Weinstein 10-g (5.07 log) monofilament is commonly used to assess protective sensation in the feet of patients with diabetes. You can order the Semmes-Weinstein monofilament from the following companies:

- Center for Specialized Diabetic Foot Care: 1-800-543-9055
- North Coast Medical, Inc.: 408-283-1900
- Sensory Testing Systems: 1-888-289-9293
- Smith & Nephew Rehabilitation Division: 1-800-558-8633.

Use the 10-g (5.07 log) monofilament wire on each foot at the following 10 sites:

- plantar aspect of the first, third, and fifth digits
- plantar aspect of the first, third, and fifth metatarsal heads
- plantar midfoot medially and laterally
- plantar heel
- dorsal aspect of the midfoot.

Performing the Test

Place the patient in a supine or sitting position. Remove his socks and shoes and provide support for his legs. Touch the monofilament to the patient's arm or hand to demonstrate what it feels like. Then ask him to respond "yes" each time he feels the monofilament on his foot (Fig. 16-2).

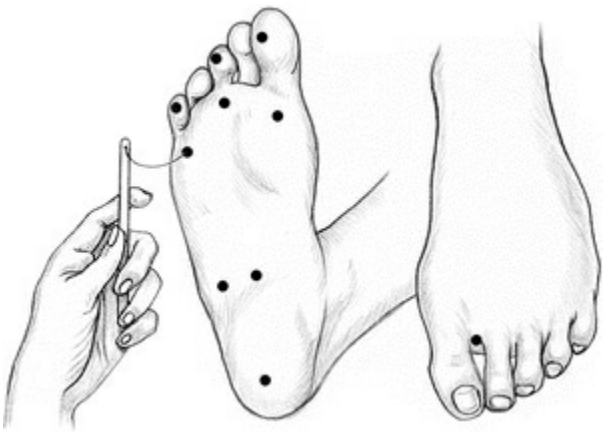


Figure 16-2 Assessing Protective Sensation with a Monofilament.

Place the patient's foot in a neutral position with his toes pointing straight up, and tell him to close his eyes. Remind him to say "yes" when he feels the monofilament on his foot. Hold the monofilament perpendicular to the patient's foot and press it against the first site, increasing the pressure until the monofilament wire bends into a C shape. Make sure it doesn't slide over the skin. Hold the monofilament in place for about 1 second. Record the patient's response on a foot-screening form. Use a "φφ" for a positive response and a "–" for a negative response. Then move to the next site.

Test all 10 sites at random and vary the time between applications so that the patient won't be able to guess the correct response. If he has a scar, callus, or necrotic tissue at a test site, apply the monofilament along the perimeter of the abnormality, not directly on it.

Loss of protective sensation is indicated if the patient can't feel the monofilament at any site on his foot. It is essential to teach a patient who has lost protective sensation to inspect and protect his feet.

Adapted from Sloan, H.L., Abel, R.J. "Getting in Touch with Impaired Foot Sensitivity," *Nursing* 28(11):50-51, November 1998; and Armstrong, D.G., et al. "Choosing a Practical Screening Instrument to Identify Patients at Risk for Diabetic Foot Ulceration," *Archives of Internal Medicine* 158(3):289-92, February 9, 1998, with permission.

Practice Point

Not all monofilaments are of the same quality or last forever; be sure to use a calibrated instrument in your patient assessments.

A systematic clinical examination can be an effective way to diagnose neuropathy and identify high-risk patients. Abbott et al. used a modification of the neuropathy disability score to evaluate a large cohort of patients (n = 9,710) with diabetes.[18] The neuropathy disability score evaluates vibration

with a 128-Hz tuning fork, pinprick, hot–cold perception, and Achilles deep tendon reflex (Fig. 16-3). Abbott showed that high neuropathy disability scores (>6) were associated with a higher ulcer incidence.

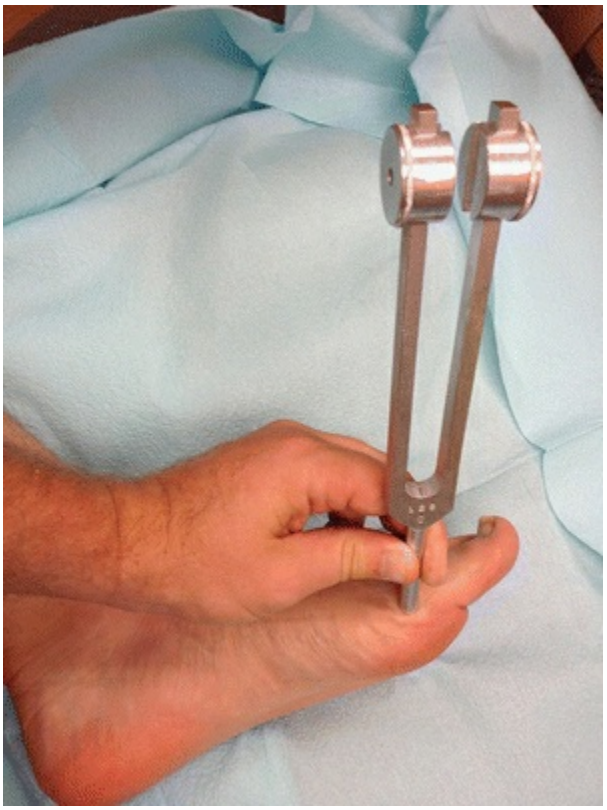


Figure 16-3. Tuning fork exam over bony prominence. (Photo courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

VPT testing is a quantitative evaluation that measures large myelinated nerve function. It is less prone to interoperator variation than are monofilaments, and it does not need to be replaced to continue providing accurate results. The VPT Meter is a handheld device with a rubber head that is applied to a bony prominence, such as the medial aspect of the first metatarsal head or the tip of the great toe. The unit contains a linear scale that displays the applied voltage, ranging from 0 to 100 V. The amplitude is then slowly increased until the patient can feel the vibration. The inability to feel greater than 25 V is indicative of LOPS and puts the patient at risk for ulceration and amputation.

Peripheral Arterial Disease

Tissue perfusion is arguably the most important aspect of the diabetic limb. PAD in patients with diabetes is characterized by multiple occlusive

plaques of small- and medium-sized arteries of the infrapopliteal vessels.[19,20] Hence, diabetes induced PAD effects both small and large vessels. This becomes an important consideration for interpreting the results of arterial diagnostic tests as well as developing a treatment plan. PAD puts the patient with diabetes at a significantly greater risk for foot ulcers, infections, and amputations.[10] Several theories attempt to explain the microvascular changes that occur in diabetes. One theory proposes that increased microvascular pressure and flow results in direct injury to the vascular endothelium, which in turn causes the release of extravascular matrix proteins. This leads to microvascular sclerosis and thickening of the capillary basement membrane. Capillary fragility also leads to microhemorrhage, which could be the reason that infection spreads through the tissue planes in patients with diabetes.[20] In addition to the direct effect on the vessels, an additional indirect effect on the microvasculature is mediated by the autonomic nervous system. LoGerfo et al.[21] believe that there is no microcirculatory occlusive process; rather, they suggest that some other indirect physiologic abnormality occurs. Altered microvascular blood flow is a complication of diabetic autonomic neuropathy that causes a shunting of blood away from the skin, making it prone to ulceration and impairing the healing process[20] (Fig. 16-4).

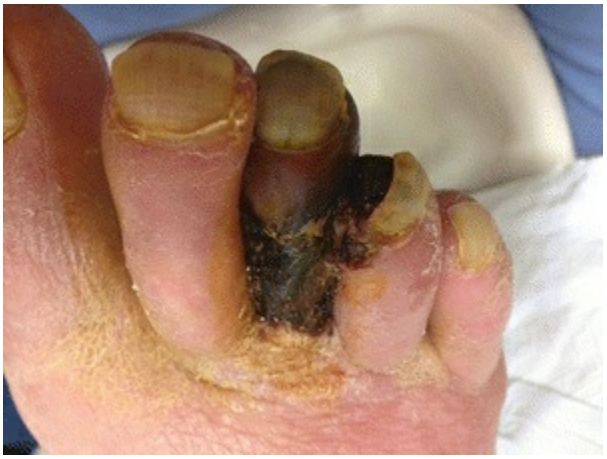


Figure 16-4. Focal necrosis of the third toe. (Photo courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

It is likely that any theory of microvascular involvement in the process of diabetic ulceration and healing must include both the direct effects of glycosylation and local inflammation and the indirect effect of alteration of microvascular hemodynamics associated with autonomic dysfunction.

Regardless of the underlying mechanism, the result is a decrease in perfusion to the tissue thereby decreasing healing potential and placing the diabetic limb at risk.

Evaluating vascular status should include a thorough history of symptoms of intermittent claudication, ischemic rest pain, and peripheral vascular surgery; clinical signs of ischemia, such as skin temperature, dependent rubor, pallor, hair loss, and shiny skin; and a clinical assessment of lower-extremity pulses.[22] According to the American College of Cardiology (ACC)/American Hospital Association (AHA) guidelines[23] for the management of patients with PAD, physicians should screen their patients for a diagnosis of PAD by determining bilateral resting ankle–brachial indices (ABIs) (Fig. 16-5). In addition, a toe–brachial index should be used when the ABI is not reliable because of excessively high systolic pressures in the ankle. When the ABI is greater than 1.3, the arteries are considered to be noncompressible due to calcifications within the endothelial walls. Segmental pressure measurements are useful to localize the site of lower-extremity PAD when planning a vascular intervention. However, in the advanced disease state of diabetes and particularly end-stage renal disease, ABIs may have limited utility due to the lack of compressibility and may require vascular surgery consultation.

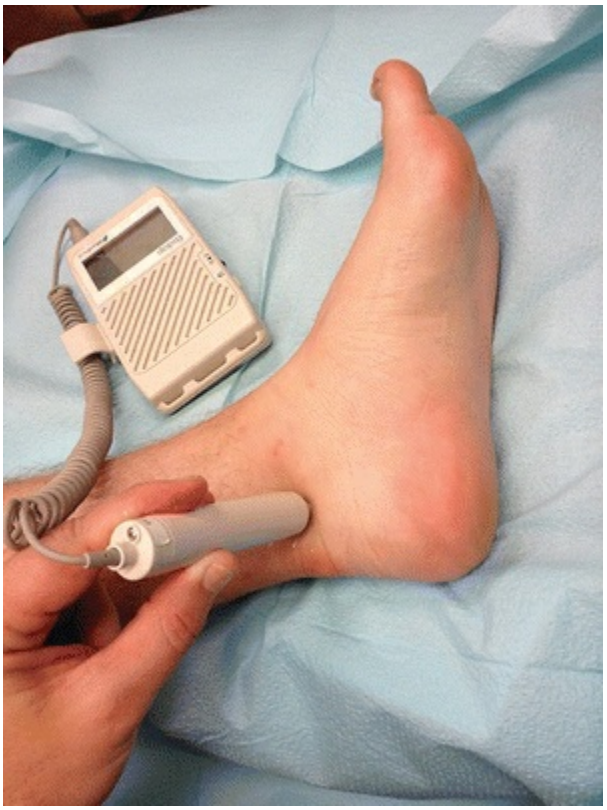


Figure 16-5. Handheld Doppler arterial exam of posterior tibial artery. (Photo courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

Skin and Nail Examination

Evaluation of the skin and nails is critical to identify the subtle signs of impending injury, including high-pressure areas, cracks, maceration, blistering, or fissures in the skin. Patient education is an important aspect of care. All patients should be instructed on how to perform skin self-examinations as a preventive measure. Discoloration of a callus or bleeding under a callus is a sign of a preulcerative lesion. Likewise, deformed and thickened nails are commonly the source of abnormal pressure on the nail bed that can cause subungual ulcerations. Common nail disorders seen in patients with diabetes mellitus include onychomycosis (tinea unguium) and onychocryptosis (ingrown toenail). While these are usually minor problems in persons without diabetes, they can result in cellulitis, osteomyelitis, neuropathy, and vascular impairment in patients with diabetes.

Musculoskeletal Examination

In patients with neuropathy, ulcerations typically develop as a result of repetitive pressure and shear on the sole of the foot or from shoe pressure on the top or sides of the foot; however, no specific level of pressure has been determined to be abnormal or pathologic.[24] Diabetes alters biomechanics in patients with preexisting structural and functional foot deformities (Fig. 16-6). Motor neuropathy is thought to contribute to atrophy and weakness of the intrinsic muscles of the foot. This leads to what has been called the "intrinsic minus foot," which describes wasting of the small (intrinsic) muscles that originate in the foot (flexor digitorum brevis, flexor hallucis brevis, extensor digitorum brevis, extensor hallucis brevis, lumbricales, interossei, and abductor hallucis).

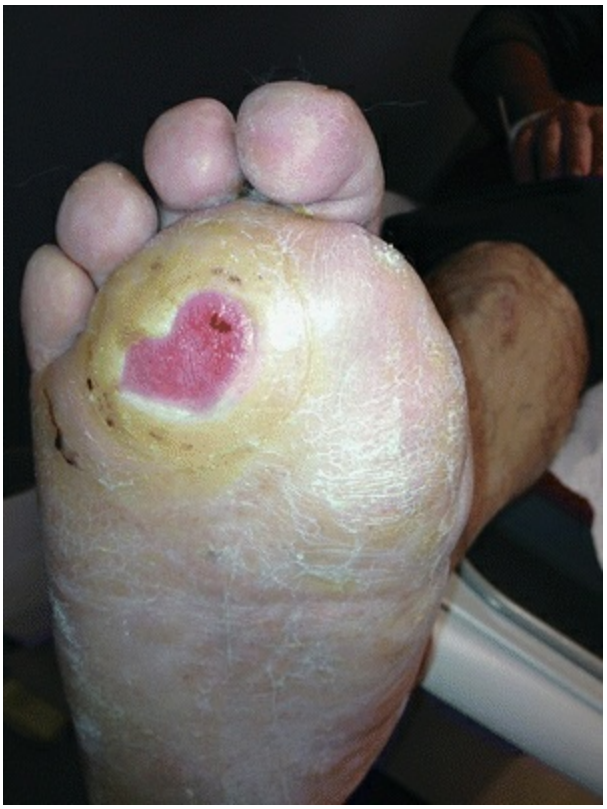


Figure 16-6. Ulcer under two to three metatarsophalangeal joints after great toe amputation. The two to three metatarsophalangeal joints are dislocated causing increased pressure on the sole of the foot. (Photo courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

Metatarsal ulcers can develop when digital deformities are irritated by the toe box of a shoe or because of loss of fat pad and increased pressure under the metatarsal head (Figs. 16-7 and 16-8). The lesser digits contract and sublux dorsally, resulting in a claw toe deformity and a strong plantar flexor force at the metatarsophalangeal joints.[25] As the toes deform and the metatarsophalangeal joints dislocate, the metatarsal heads are literally driven through the bottom of the foot. The tips and dorsal aspects of the toes and the area beneath the metatarsophalangeal heads are subjected to increased pressure and friction, which, in the presence of LOPS, can lead to ulceration.[26] Limited mobility of the ankle and metatarsophalangeal joints has been associated with soft tissue glycosylation involving the gastroc-soleus-Achilles complex and periarticular tissues.[27] Limited motion of the ankle, subtalar, and metatarsophalangeal joints has been associated with high pressures in the forefoot. Often patients with an intrinsic minus foot will appear to have a high arch; however, this is not a congenital deformity but rather is due to atrophy of the abductor hallucis muscle belly on the

medial side of the foot. A profound example of musculoskeletal abnormality of the diabetic foot is represented by Charcot neuroarthropathy, which is characterized by fracture, subluxation, and/or dislocation of joints in the foot or ankle. These structural deformities typically cause plantar bony deformities resulting in areas of high pressure leading to ulceration.

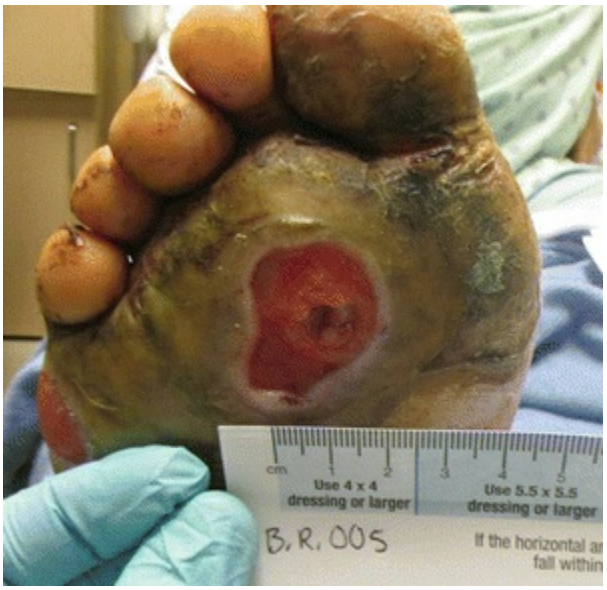


Figure 16-7. Metatarsal ulcer. (Photo courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

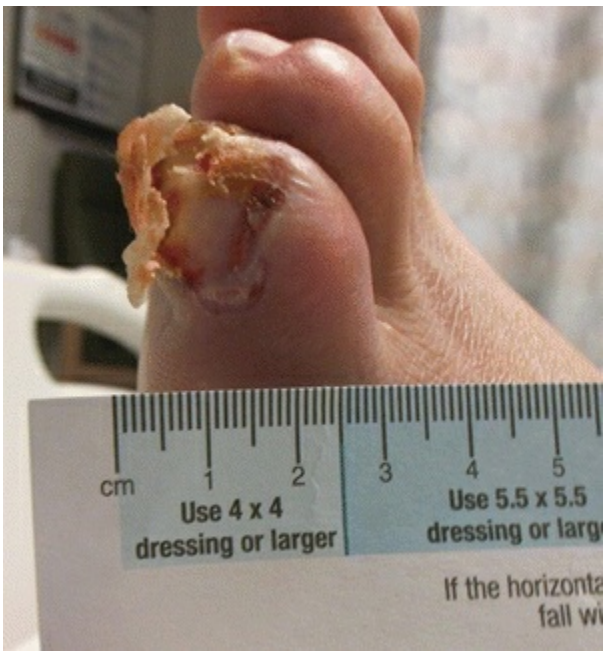


Figure 16-8. Ulcer on digital deformity. (Photo courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

Infection

Soft tissue and bone infections are very common in persons with diabetic foot ulcerations. The majority of patients with diabetic foot ulcers (56%) will be treated for soft tissue infection during the course of their ulceration. Approximately 20% of these patients will develop infection of the underlying bone.[28] Classically defined signs and symptoms of infection as outlined in the Infectious Disease Society of America (IDSA) guidelines are certainly limb threatening.[29]

Identification of foot infections in patients with diabetes requires vigilance because the normal signs of infection may be blunted or absent. Hyperglycemia impairs the humoral innate immune response by increasing the proinflammatory cytokine levels, dysregulating vasoactive cytokines, such as bradykinin and nitric oxide (NO), and decreasing complement activation. This, in turn, can lead to increased insulin resistance through several pathways, causing more hyperglycemia.[30,31] The polymorphonuclear cells (PMNs) and monocytes of the cellular innate immune system show impaired chemotaxis, adherence, phagocytosis, and intracellular killing in patients with diabetes.[32,33] The influence of hyperglycemia on the adaptive immune system (i.e., T cells and immunoglobulins) is less well understood.

The lower-extremity wounds are often colonized with microorganisms regardless of the presence of a "true infection." Routine cultures of the wounds and superficial swabs should not be done when the wound is clinically not infected.[34] When cultures are obtained, deep tissue should be obtained rather than superficial swabs. In cases of mild or moderate infection, curettages from the ulcer base after debridement should be obtained prior to the initiation of antibiotics.[35] In severe infections, empiric antibiotic therapy should be started as soon as possible. Bone biopsy, preferably under radiographic guidance, is the most reliable method to obtain bone cultures in patients suspected of having osteomyelitis.[36] (See Chapter 7, Bioburden and Infection.) Wound depth is the strongest predictor of both soft tissue and bone infections. Compared with superficial wounds, the risk of infection for wounds that extend to the bone is 23 times higher for soft tissue and 6.7 times higher for bone (Table 16-1).

Table 16-1 Risk Factors for Soft Tissue and Bone Infections

	Relative Risk	95% Confidence Intervals	*P* Value
Risk Factors for Soft Tissue Infection			
Wound penetrates to bone	23.08	8.47–62.92	0.0001
Previous history of ulceration prior to enrollment	2.15	1.07–4.32	0.03
Recurrent/multiple wounds during study period	1.92	1.20–3.06	0.007
Risk Factors for Bone Infection			
Ulcer depth to bone	6.71	2.27–19.85	0.001
Ulcer duration >30 d	4.66	1.62–13.37	0.004
Recurrent foot ulceration	2.41	1.28–4.53	0.006
Traumatic etiology to ulcer	2.36	1.12–4.98	0.02
Peripheral vascular disease	1.93	1.04–3.56	0.04

Adapted from Lavery, L.A., Armstrong, D.G., Wunderlich, R.P., et al. "Risk Factors for Foot Infections in Individuals with Diabetes," *Diabetes Care* 29(6):1288, June 2006; and Lavery, L.A., Peters, E.J., Armstrong, D.G., et al. "Risk Factors for Developing Osteomyelitis in Patients with Diabetic Foot Wounds," *Diabetes Research in Clinical Practice* 83(3):347-52, March 2009.

Biofilm is a significant growing concern that is less understood, its impact underappreciated, and not effectively treated. Biofilm is a colony of bacteria, fungus, or yeast that can populate a wound within 10 to 24 hours.[37,38] Once established, a complex and biodiverse community evolves protected by a glycocalyx shell.[39,40] Thus, conservative debridement may not be able to reach these deeper layers.

Unlike planktonic (unattached/free floating) bacteria that are metabolically active and can be generally treated with antibiotic therapy, microbes within a biofilm are relatively senescent. Thus, the mechanism of action of antibiotics, which is to interfere with protein synthesis (disruption of cell wall, cell membrane synthesis) is largely ineffectual against biofilm. Although biofilm under ideal circumstances provides a steady-state ecology of checks and balances between the microbial species, the chronic wound may be dominated by one species that develops into wound chronicity and periods of limb-threatening acute infection. Further troubling is the fact that qualitative and standard quantitative culturing techniques selectively identify specific and limited number of bacteria, fungus, and yeast. Thus, the true pathogenic microbe may not be detected and subsequently not treated.[41] Biofilm's definitive influence on wound healing and infection has yet to be determined. However, it is important to acknowledge that infection and biofilm are different entities that require treatment strategies.

Diagnostic Imaging

Whenever there's an open wound on the foot, it's always wise to order an x-ray initially to check for the presence of osteomyelitis. If the wound has been open for several weeks or if bone can be palpated with a sterile probe, further investigation is warranted even if standard x-rays are negative.

Several studies showed that the magnetic resonance imaging (MRI) is the diagnostic modality of choice to detect the presence of osteomyelitis.[42,43] However, MRI is not always available and requires a well-trained and experienced reader. If MRI is unavailable and plain x-ray reveals changes suggestive of osteomyelitis (cortical erosion, active periosteal reaction, mixed lucency, and sclerosis), a bone biopsy should be obtained prior to treatment with an antibiotic regimen or surgical resection. If the x-ray shows no evidence of osteomyelitis, the patient should be treated with an empiric antibiotic regimen, and radiographs of the foot should be repeated.

Multidisciplinary Framework for Prevention

Evaluation of risk factors and risk stratification is important to prioritize the patient's treatment according to his or her individual needs.[44] Many healthcare providers either never evaluate the feet or generally consider everyone with diabetes to be "at-risk" for foot problems. This usually leads to no preventive care, but it can also contribute to unnecessary services for low-risk patients. To help evaluate individual risk factors, a risk classification system endorsed by the International Working Group on the Diabetic Foot[13] provides a validated scheme to stratify subjects based on their risk of ulceration and amputation. Key elements of the lower-extremity examination should help to risk-stratify subjects in order to identify the frequency and level of preventive care required (Table 16-2).

Table 16-2 Lavery-Peters Diabetic Foot Risk Classification

Category	Risk Factors	Ulcer Incidence	Amputation Incidence	Prevention and Treatment
1	No neuropathy No peripheral arterial disease (PAD) No history of ulcer	2%	0.04%	• Reevaluation once a year
2	Neuropathy ± deformity No PAD No history of ulcer or amputation	3.4%	0.05%	• Podiatry every 6 mo • Over-the-counter shoes and insoles; evaluate appropriate fit
3	PAD No history of ulcer or amputation	13.8%	3.7%	• Podiatry every 2–3 mo • Professionally fit therapeutic shoes and insoles • Patient education
4	Previous ulcer or amputation	31.5%	8.1%	• Podiatry every 1–2 mo • Professionally fit therapeutic shoes and insoles • Patient education

Adapted from Lavery, L.A., Peters, E.J., Williams, J.R., et al. "Reevaluating the Way We Classify the Diabetic Foot: Restructuring the Diabetic Foot Risk Classification System of the International Working Group on the Diabetic Foot," *Diabetes Care* 31(1):154-6, January 2008.

Many strategies can be used to help prevent foot complications in diabetic patients. Several clinical studies have reported a 48% to 78%[45] reduction in amputations and a 47% to 49% reduction in lower-extremity–related hospitalizations[46] when high-risk diabetic patients are treated in specialty clinics. These clinics often include multiple specialties that focus on both prevention and care of acute complications in patients with diabetes. Further, consensus documents for prevention measures related to the "diabetic foot" have been developed by the American Orthopaedic Foot and Ankle Society,[47] the American College of Foot and Ankle Surgery,[48] the Registered Nurses' Association of Ontario, and the International Working Group on the Diabetic Foot.

While multidisciplinary clinics is an important principle in the comprehensive care of these patients, the practice of multidisciplinary care falls short. In most cases, these multidisciplinary care clinics employ specialty providers that spend a few half days each week in clinic and work in their own specialty offices for the remainder of the week. Thus, continuity of care and continuous communication between specialists are limited. Ideally, all specialists are colocated within the same clinic/institution. However, this may not be practical in many situations. Thus, a new "spoke and hub" model of wound care may make more sense. This model utilizes community-based wound care centers to screen, assess, diagnose, and treat the majority of wounds while referring the most difficult cases to tertiary

referral centers within a regional area where a more resource intensive approach can be utilized. Thus, a distinction between a “wound care center” (community based) and a “limb salvage center” (tertiary referral center) should be implemented. [49]

Another evolution of wound care thought may include a period of intensive inpatient care. Currently, weekly or biweekly clinic-based treatment is the norm that may result in little demonstrable progress. An inpatient course that includes rigid glucose control, nutrition optimization, perfusion maximization, intravenous antibiotic therapy, and serial operating room wound debridement with a plan for definitive wound closure or coverage may be of benefit.

Further complicating the treatment of chronic wounds is the diversity of wound care providers. Wound care is a discipline that spans multiple disciplines. Thus, wound treatment is driven by the scope of training, licensing, and differing philosophical approaches. This is another reason for colocated wound care where the strengths of each discipline can be combined to a single, comprehensive treatment approach.

Foot Care

Regular foot evaluation is essential to identify new risk factors and prevent impending complications. Podiatric physicians provide for debridement of callus and nails as well as regular evaluation of shoes and insoles. These routine encounters offer an additional opportunity to reinforce key educational elements, such as the need to avoid going barefoot, hydrate the skin, and inspect the feet daily. Protective footwear and insoles can be prescribed for the patient and then evaluated and monitored for their effectiveness.

Protective Footwear and Pressure Redistribution

The primary role of therapeutic footwear is to protect the foot from repetitive injuries and eliminate the shoe as a source of pathology. Extra-depth shoes have a high toe box with enough depth throughout the shoe to accommodate a total contact molded insole or orthotic. These are often recommended for patients with structural foot deformities, such as claw toes or dislocated metatarsophalangeal joints. These types of shoes usually allow for up to a ⅜”-thick accommodative insole to fit without irritating the

top or sides of the foot (Fig. 16-9).

Figure 16-9. Hemisection of diabetic extra-depth shoe with insole.

The combination of a correctly sized shoe and a protective insole can reduce pressure on the sole, top, and sides of the foot by as much as 20%.[50,51]

Several studies[52,53] have shown a significant reduction in foot ulcers in patients who receive therapeutic shoes, compared to shoes, patients would normal select themselves. There is little clinical evidence to help us understand the effectiveness of the types of therapeutic shoes and insole that are commonly used to prevent foot complications. There are a variety of insole materials and material combinations as well as different accommodations that can be built into the insole. Likewise, the type of shoe and outer sole accommodations are numerous. Most of the decisions for protective shoes and insoles are left to technicians that have little working knowledge of the medical literature. When patients reulcerate, they do not return to the pedorthist or shoemaker for care, so these providers have no follow-up to determine if their approach is effective.

There are four randomized clinical trials and two prospective cohort studies that describe the benefit of various types of shoes and insoles for high-risk patients with diabetes.[54] Most studies include patients with a previous foot ulcer and use a control group of patients with self-selected footwear. In some studies, this is because they cannot afford therapeutic shoes,[55] they refuse recommended shoes,[56] or their insurance does not pay for shoes and insoles.[57] Reulceration is much higher among patients who do not use therapeutic shoes and insoles. About 60% of patients reulcerate with self-selected shoes. Among patients who receive therapeutic shoes and insoles, there is a two- to fourfold reduction in reulceration compared to

patients that use shoes they have selected. Even with standard preventative care such as therapeutic shoes and insoles, education, and regular foot care, about 24% to 50% of patients will develop another foot ulcer within the next year.[58]

The four randomized clinical studies that evaluate therapeutic shoes and insoles provide a glimpse into the complexity of evaluating this intervention to prevent ulcers. Reiber et al. compared two insole constructs with off-the-shelf footwear compared to patient-selected shoes.[59] Uccioli et al.[60] compared custom-made shoes and insoles to patient-selected shoes. Lavery et al.[53] randomized patients to receive a shear-reducing insole compared to a standard insole and off-the-shelf shoe, as well as education and regular foot care. Rizzo et al. [61]assessed the impact of a structured follow-up program on the incidence of diabetic foot ulceration in high-risk diabetic patients.

Custom-molded shoes (Figs. 16-10 and 16-11) are individually made from a mold of the patient's foot. Custom-molded shoes can be expensive and require several weeks or months to make. However, they are only necessary in a small percentage of high-risk patients with severe foot deformities that cannot be accommodated by off-the-shelf shoes. For most patients with less severe deformities, there are a number of more affordable athletic, comfort, and therapeutic shoes with multiple sizes and extra depth to accommodate a wide variety of foot deformities. Even with the appropriate shoes, recurrence of ulcers is a problem. In a study by Waaijman et al., 171 neuropathic diabetic patients with a recently healed plantar foot ulcer and custom-made footwear were followed for 18 months or until ulceration. A total of 71 patients had a recurrent ulcer[62]. Significant independent predictors were presence of minor lesions, day-to-day variation in stride count, and cumulative duration of past foot ulcers. They concluded that the presence of a minor lesion was clearly the strongest predictor, and the use of adequately off-loading footwear was a strong protector against ulcer recurrence from unrecognized repetitive trauma.[62]

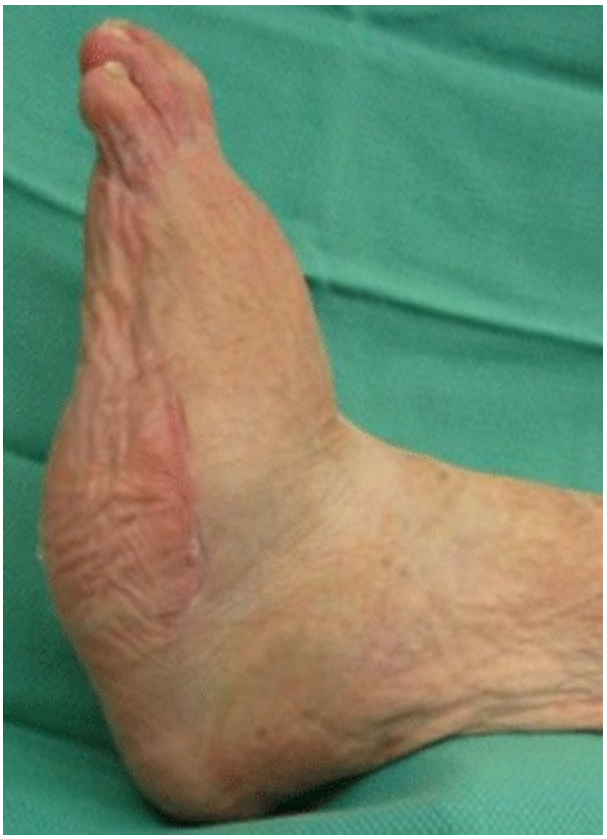


Figure 16-10. Charcot deformity. (Photo courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

Figure 16-11. Custom-molded high top boot designed to fit a patient with Charcot deformity.

The Diabetic Shoebill

Patients with diabetes who are "at risk" for foot disease and who have Medicare Part B are eligible for Medicare's Therapeutic Shoe Bill.[63] In

order to qualify, a patient must have diabetes and one or more of the following: previous amputation of part or all of either foot; a history of previous foot ulceration or preulcerative calluses; peripheral neuropathy with evidence of callus formation; foot deformity; or poor circulation.

The bill covers one of the following annually: one pair of off-the-shelf extra-depth shoes and three additional pairs of multidensity inserts or custom-molded orthoses; one pair of off-the-shelf extra-depth shoes, including a modification and two additional pairs of multidensity inserts; or one pair of custom-molded shoes and two additional pairs of multidensity inserts (Fig. 16-12).

Figure 16-12. Wide-toe shoe with insert. Shoes with a deep toe box and that are "extra depth" throughout the shoe are the mainstay of diabetic wound preventive care. The shoes typically come with laces, as shown here, or with Velcro closures. The combination of a correctly sized shoe and an accommodative insole can reduce pressure on the sole, top, and sides of the foot. The insert is customized to relieve pressure, according to the patient's needs, and then placed inside the shoe. (Photographs courtesy of Royce Medical Co.)

Preventive Education

Education has long been assumed to be an essential component of any program designed to reduce the incidence of diabetic foot ulcers. Preventive education usually takes the form of an intensive introduction to the disease and includes practical steps to cope with the manifestations of diabetes over time. In a 2004 *Cochrane Review*[64] of nine randomized, controlled trials to determine the effectiveness of educational programs in preventing diabetic foot ulceration, the authors concluded that there was only weak evidence to suggest that education reduces foot ulceration and amputations in high-risk patients. On the other hand, the studies reinforced the idea that increased knowledge of foot care had a positive effect on patient behavior in the short term. Continual education and reinforcement may be helpful, especially among high-risk patients, to improve patient outcomes over the long term.

To complicate matters, many patients with diabetes have severe limitations to classical education methods. A large proportion of patients both with and without foot ulcers lacked the visual acuity, manual dexterity, or joint flexibility to perform the self-examination necessary to care for their feet.[4,11] Forty-nine percent of patients with ulcers could not position or see their feet, and 15% were legally blind in at least one eye. When patients are obese or have limited joint mobility or impaired vision,

education and self-assessment skills should be directed to both the patient and his or her spouse or caregiver.[4,11] Repetition and regular reinforcement should be practiced by every member of the healthcare team to help the patient and family maintain an understanding of the disease process and continue to practice protective behaviors to avoid some of the serious complications of diabetes.

Temperature Monitoring

At-home temperature monitoring (Fig. 16-13) is a new concept for high-risk patients to identify early warning signs of tissue injury before a foot ulcer actually develops. Because neuropathy inhibits the natural warning system, local inflammation and pain as a result of tissue trauma go unnoticed. Several studies have used temperature assessment as a surrogate to identify tissue injury in patients at risk for diabetic foot ulcers and pressure ulcers. Indeed, two randomized clinical trials demonstrated a 3- to 10-fold reduction in foot complications among high-risk patients using at-home temperature assessment as compared with standard prevention therapy.[65,66]

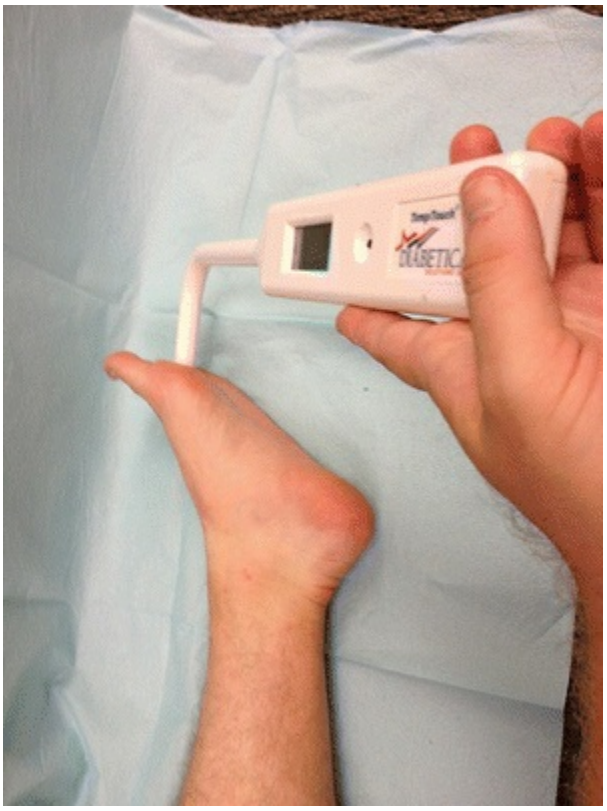


Figure 16-13. Plantar foot temperature exam. (Photo courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

Practice Point

At-home temperature monitoring can provide objective feedback to warn patients with neuropathy that their feet are injured before an ulcer develops.

Wound Characteristics and Assessment

Several classification systems can be used to classify diabetic ulcers. The University of Texas ulcer classification system (Table 16-3) is a validated system that includes a mechanism to document wound depth as well as the presence of infection and vascular impairment—two pivotal factors in predicting clinical outcomes. Using this system, the risk of amputation has been shown to be predictive of amputation as wounds increase in depth (grade 0 to III) and progress from no infection (class A), to infection (class B), to PAD (class C), and to infection and PAD (class D) (Fig. 16-14).

Table 16-3 University of Texas Diabetic Wound Classification System

	Grade			
Class	**0**	**I**	**II**	**III**
A	Preulcerative or postulcerative lesion, completely epithelialized	Superficial wound, not involving tendon, capsule, or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	Preulcerative or postulcerative lesion, completely epithelialized with infection	Superficial wound, not involving tendon, capsule, or bone, with infection	Wound penetrating to tendon or capsule with infection	Wound penetrating to bone or joint with infection
C	Preulcerative or postulcerative lesion, completely epithelialized with ischemia	Superficial wound, not involving tendon, capsule, or bone, with ischemia	Wound penetrating to tendon or capsule with ischemia	Wound penetrating to bone or joint with ischemia
D	Preulcerative or postulcerative lesion, completely epithelialized with infection and ischemia	Superficial wound, not involving tendon, capsule, or bone, with infection and ischemia	Wound penetrating to tendon or capsule with infection and ischemia	Wound penetrating to bone or joint with infection and ischemia

Reprinted from Armstrong, D.G., et al. "Validation of a Diabetic Wound Classification System: The Contribution of Depth, Infection, and Ischemia to Risk of Amputation," *Diabetes Care* 21(5):855-69, May 1998, with permission.

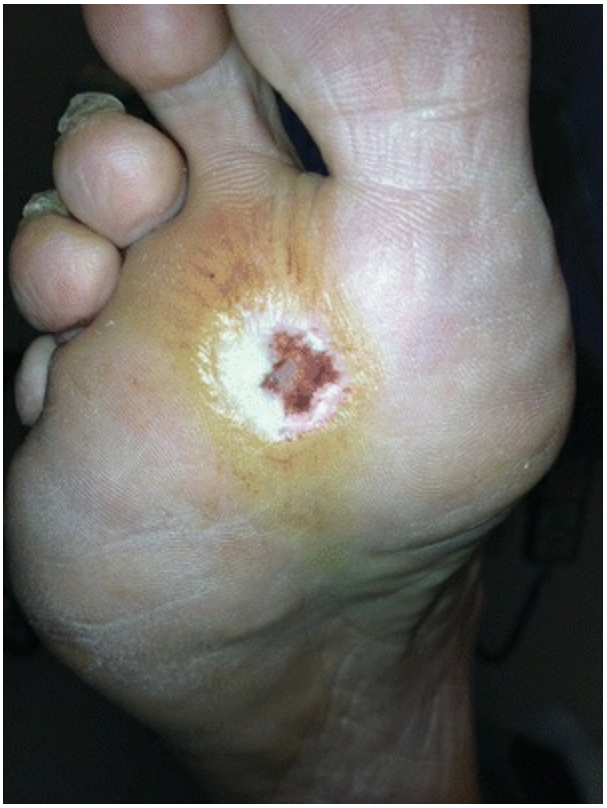


Figure 16-14. UT1A full-thickness ulcer without involvement of deep structures with no PAD or infection. (Photo courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

A classification scheme first described by Meggitt and popularized by Wagner has also been used extensively but has the disadvantage of not consistently including wound depth or the presence of infection (Table 16-4). Osteomyelitis is the only type of infection included, and end-stage disease events of gangrene are the only vascular parameters included. Furthermore, the system is difficult to use for more subtle disease processes that are critical for clinical decision making.

Table 16-4 Meggitt-Wagner Ulcer Classification

Grade	Wound Characteristics
0	Preulceration lesions, healed ulcers, presence of bony deformity
1	Superficial ulcer without subcutaneous tissue involvement
2	Penetration through the subcutaneous tissue; may expose bone, tendon, ligament, or joint capsule
3	Osteitis, abscess, or osteomyelitis
4	Gangrene of digit
5	Gangrene of foot

Reprinted from Wagner, F.W. "The Dysvascular Foot: A System for Diagnosis and Treatment," *Foot & Ankle* 2(2):64-122, September 1981; and Meggitt, B. "Surgical Management of the Diabetic Foot," *British Journal of Hospital Medicine* 16:227-32, 1976, with permission.

The ADA Consensus report[2] recommends that a systematic wound assessment include the following questions in the evaluation:

- Has the patient experienced trauma? Is the ulcer a result of penetrating trauma, blunt trauma, or burn?
- What is the duration of the wound? Is the ulcer acute or chronic?
- What is the progression of local or systemic signs and symptoms? Is the wound getting better, is it stable, or is it deteriorating?
- Has the patient had any prior treatment of the wound or previous wounds? What treatments worked? What failed?

In addition, blood glucose control and comorbidities should be evaluated. Clinical assessment should identify:

- signs of ischemia—adequate blood flow to heal the wound
- signs of soft tissue or bone infection—cellulitis, abscess, or osteomyelitis
- wound depth—undermining or exposed tendon, joint capsule, or bone
- appearance—surrounding callus, devitalized tissue, granulation tissue, drainage, eschar, or necrosis.

Practice Point

Six essentials of the ADA treatment algorithm

- debridement, early and often
- reducing pressure
- moist wound healing
- treating infection
- correcting ischemia (below the knee disease)
- preventing amputation.

Treatment Plan

Debridement (Surgical, Enzymatic, Maggot, Ultrasound)

Sharp debridement of the ulcer removes devitalized tissue, reduces the bacterial load of the wound, eliminates proteases from the wound bed, and provides a bleeding wound bed. A diabetic ulcer typically has a thick rim of keratinized tissue surrounding it. Debridement must remove all of the callus and devitalized tissue, so that a clean wound edge is created and all edge pressure from the callus is removed. Enzymatic or autolytic debridement may be an option if sharp debridement is not possible or if the patient has PAD.[22] Ongoing debridement may be needed throughout the healing process.[67] Indeed, higher healing rates have been observed in patients who have had more frequent debridement. In addition, in a post hoc evaluation from the becaplermin gel pivotal trial, Steed reported a higher proportion of healed wounds in both the treatment and placebo study groups when wound debridement was performed more frequently.[68]

It is important to acknowledge that not all forms of sharp debridement are the same. Sharp debridement performed in the clinic has significant limitations that may result in insufficient removal of devitalized tissue and pathogens (Fig. 16-15A and B). The inability to control significant bleeding, pain control, and the nonsterility of the environment in which it is performed limit aggressive debridement. Operating room debridement eliminates these

barriers. An evolution in thought for the treatment of chronic wounds should include "excisional debridement."[69] This technique describes the excision of the entirety of the wound and its perimeter much like the approach to a soft tissue tumor. Further, in the operating room setting, after this type of debridement is performed, a definitive closure or graft can be placed.

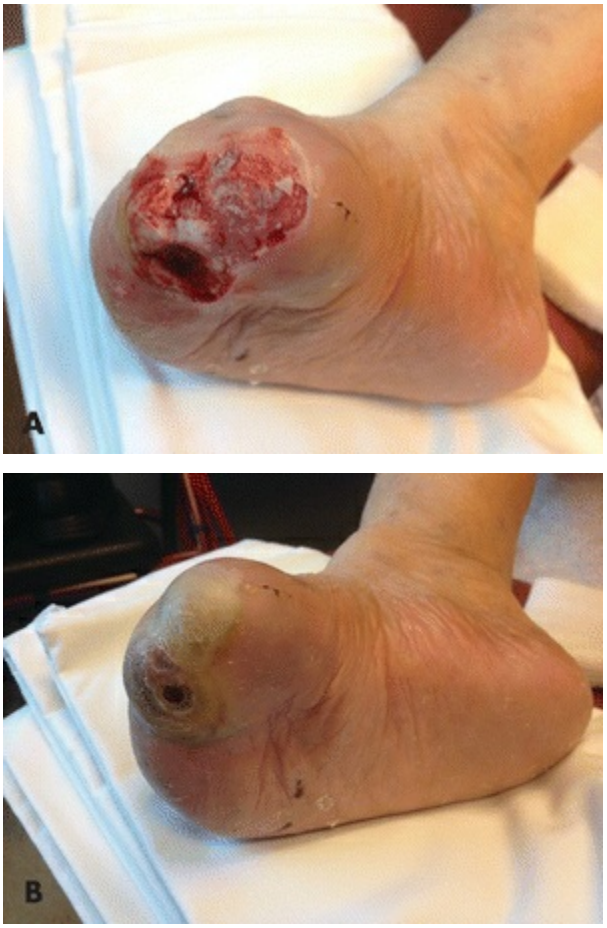


Figure 16-15. (A) Full-thickness ulcer with undermining predebridement. **(B)**Ulcer postdebridement (Note: increased in actual size). (Photos courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

Maggot therapy consists of the topical application of medical-grade, sterile, growth-arrested fly larvae. This type of debridement is reserved for the debridement of nonviable tissue in patients that may not be a candidate for surgical debridement.[70] Sherman reported a significant reduction in necrotic tissue with maggot therapy as compared with conventional wound therapy with complete removal of all necrotic tissue after 4 weeks.[71] Maggots are applied onto the wound surface and are housed in a breathable mesh cage about the wound site for 2 to 3 days at a time. The maggots digest and degrade infected and necrotic tissue continuously without effecting

healthy tissue.

Ultrasound debridement imparts sound energy to the wound site. The two types of ultrasound are contact and noncontact. As the name indicates, the contact ultrasound is in direct contact with the wound surface while the noncontact ultrasound transmits sound energy through a saline medium. Beyond removal of nonviable tissue, ultrasound energy can penetrate to deeper layers of the wound surface thereby potentially disrupting biofilm. Seth et al. reported in a rabbit ear wound biofilm model that ultrasound significantly reduced biofilm inflammatory cytokine production.[72] Robust human studies are still needed to fully understand the impact of this technology.

Off-loading

Reduction of pressure and shear forces on the foot may be the single most important yet most often neglected aspect of neuropathic ulcer treatment. Off-loading therapy is a key part of the treatment plan for diabetic foot ulcers. The goal is to reduce the pressure at the ulcer site and keep the patient ambulatory.[73,74] Several methods are available to protect the foot from abnormal pressures (Table 16-5). Off-loading strategies must be tailored to the age, strength, activity, and home environment of the patient. In general, however, more restrictive off-loading approaches will result in less activity and better wound healing. Education is critical to improve compliance with off-loading. The patient must understand that the wound is a result of repetitive pressure and that every unprotected step is literally tearing the wound apart.

Table 16-5 Off-Loading Modalities and Wound Healing

Off-Loading Modality	Mean Healing Time	Percent Healed	Source
Total contact cast	• Forefoot ulcers: 30 d • Midfoot and hindfoot ulcers: 63 d	90%	Myerson, M., et al.[75]
Total contact cast	• 38 d	73%	Helm, P.A., et al.[74]
Total contact cast	• 44 d	82%	Sinacore, D.R., et al.[76]
Total contact cast	• Forefoot ulcers: 31 d • Nonforefoot ulcers: 42.1 d	Not reported	Walker, S.C., et al.[72]
Total contact cast	• Midfoot ulcers: 28 d	100%	Lavery, L.A., et al.[77]
Total contact cast Cast boot Half-shoe	• 34 d • 50 d • 61 d	90% 65% 58%	Armstrong, D.G., et al.[78]
Total contact cast Shoe insole	• 42 d • 65 d	90% 32%	Mueller, M.J., et al.[79]
Scotch cast boot	• 112 d • 181 d	80%	Knowles, E.A., et al.[80]
Half-shoe	• 70 d	96%	Chantelau, E., et al.[81]
Custom splint	• 300 d	Not reported	Boninger, M.L., and Leonard, J.A.[82]

Practice Point

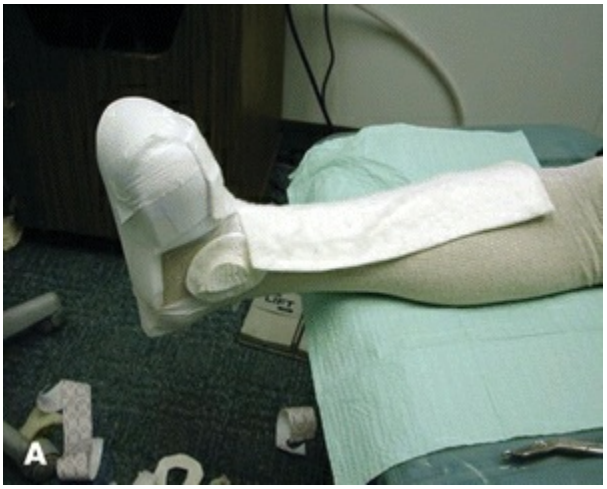
Methods to off-load the diabetic foot include:

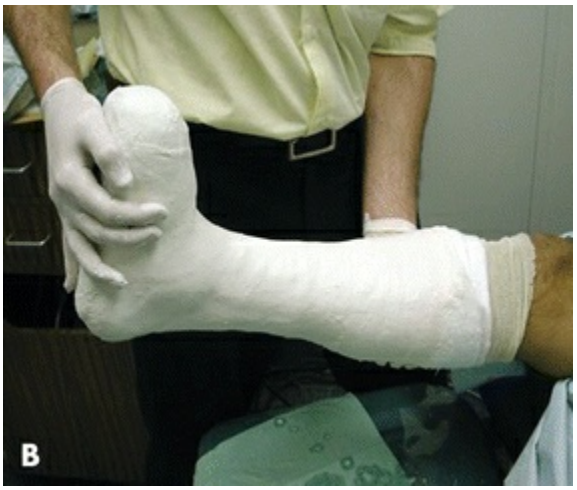
- bed rest
- wheelchair
- ambulatory aids (crutches, walker)
- felted foam padding
- half-shoes
- therapeutic shoes
- custom shoes
- custom total contact foot orthoses
- custom splints or braces
- prefabricated cast walkers.

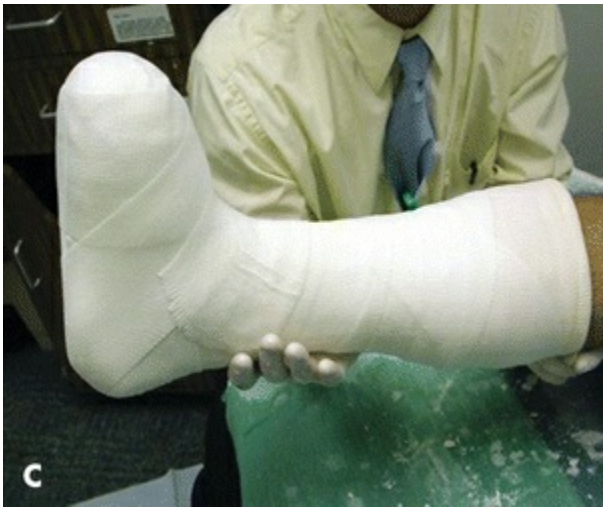
Total Contact Cast

Use of a total contact cast (TCC) is considered the gold standard for off-loading the foot. TCCs reduce pressure at the ulcer site while still allowing the patient to be ambulatory.[52] A skilled clinician or technician is required

to apply the molded plaster cast to ensure a proper fit. A TCC is a modification of a traditional fracture cast that uses minimal cast padding and includes a covering to protect the toes (Fig. 16-16). The cast is molded to the contour of the foot and leg so that no movement is possible within the cast. TCCs are generally changed every 1 to 2 weeks but may need to be replaced more frequently in patients with edema or other concerns.







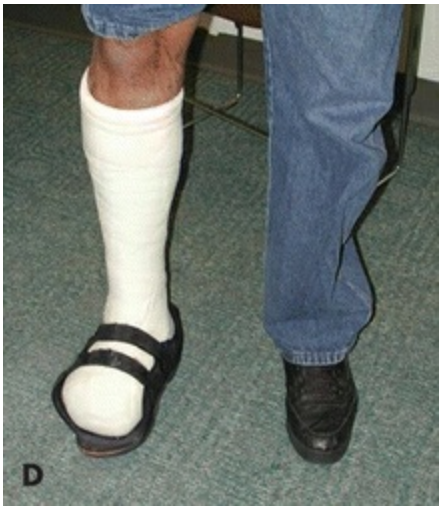


Figure 16-16. Applying a total contact cast (TCC). **(A)**A foam layer covers the toes for protection, and padding is applied over bony prominences before the first layer of casting material is applied. **(B)** Application of the TCC. **(C)** Completed TCC. **(D)** A cast boot covers the TCC. (Photo courtesy of J. McGuire, DPM. Used with permission.)

A TCC is one of the most effective ways of treating plantar neuropathic foot ulcers.[76] Numerous studies[77–79,83] have shown that TCCs can heal ulcers in 6 to 8 weeks. In descriptive and randomized clinical trials, the proportion of wounds that heal with TCCs is consistently much higher than those using topical growth factors, bioengineered tissue, or special dressings.[84,85]

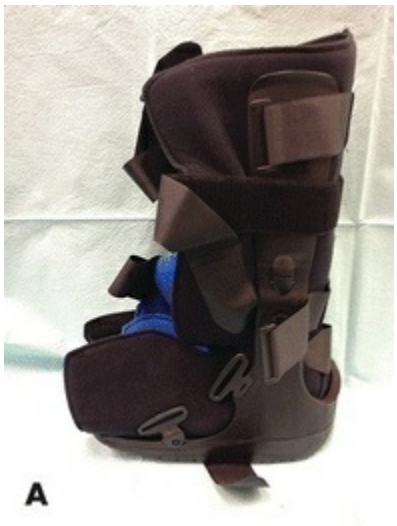
One of the main advantages of using a TCC is that it forces patient compliance with off-loading. The ulcer is protected with every step the patient takes. Using a TCC to facilitate wound healing is analogous to using a cast to heal a fracture—in both cases, healing is facilitated by rest and immobilization. The TCC reduces the patient's activity level, decreases stride length and cadence, and significantly reduces pressure at the ulcer site.[76,78] The main disadvantages for patients are the same as their complaints with a fracture cast—a cast is heavy and hot and makes bathing, walking, and sleeping difficult.

Practice Point

TCCs *should not* be used if wound infection is suspected or present.

Removable Cast Walkers

The effectiveness of removable cast walkers (Fig. 16-17) to reduce pressure at ulcer sites has been shown in several studies to be comparable to that of TCCs.[76,78] Many practitioners consider removable cast walkers to be their preferred off-loading device because they are less time consuming and easier to apply than TCCs, and they are more readily accepted by patients. In addition, the TCC has several precautions and contraindications that aren't issues with removable walkers. Edema can be overcome with constant adjustments to the fit of the device, and compression dressings can be applied in conjunction with the removable cast boot (Fig. 16-18). Wounds can be inspected regularly and treated with advanced wound care products such as growth factors, electrical stimulation, and other biologically active dressings. Because the wound and limb can be inspected frequently, the vascular concerns inherent in the occlusive irremovable TCC aren't an issue.



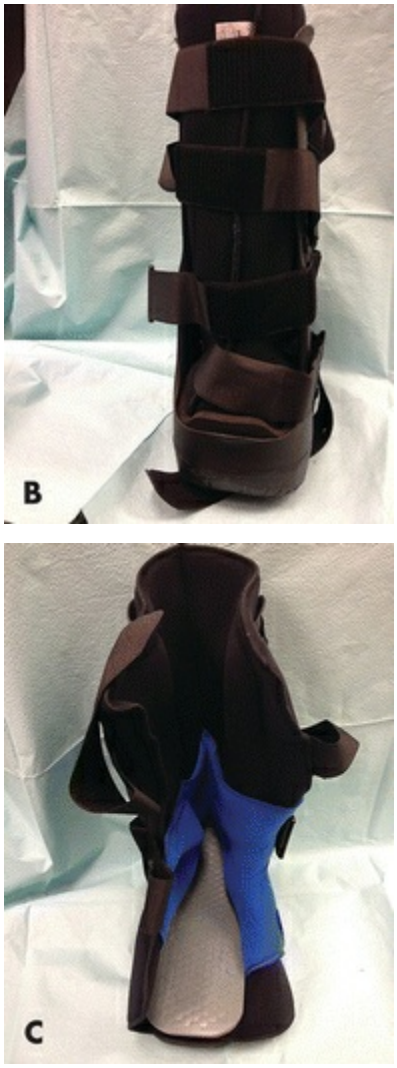


Figure 16-17. (A) Off-loading walker, lateral view. **(B)** Off-loading walker, posterior view. **(C)** Off-loading walker, anterior view with honey comb off-loading insole visible. (Photos courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

Figure 16-18. Removable cast boot with a removable cushioned foot bed. (Photo

courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

Additional advantages of removable walking boots (as compared with TCCs) are that they are relatively inexpensive, the protective insole can be easily replaced if it shows signs of wear, no special training is required for correct and safe application, and they can be easily removed to assess and debride the wound as appropriate.[86,87] It's also possible to modify removable walkers into nonremovable devices by securing the walker with cast material or a nonremovable cable tie; this is known as an instant TCC. If patients can't remove the walker, the element of forced compliance that makes the TCC attractive is maintained and the outcomes for healing improve to the levels seen with the TCC.[80,88]

No one off-loading device is appropriate for every patient. McGuire[81] has suggested a transitional approach to healing and maturing the diabetic foot ulcer that uses the instant TCC for initial pressure management and transitioning to removable devices and shoe-based platforms before the patient is ready for definitive footwear.

In a randomized controlled trial, Armstrong et al.[82] compared the effectiveness of TCCs, removable cast walkers, and half-shoes in healing neuropathic foot ulcerations in individuals with diabetes. The percentage of healing at 12 weeks was 89.5% for the TCC, 65.0% for the cast walker, and 58.3% for the half-shoe. When the cast walker is made nonremovable (ITCC), the difference between the TCC and cast walker effectively disappears.[89]

Healing Sandals and Half-Shoes

A number of healing sandals and half-shoes or wedged shoes are available to reduce pressure on the forefoot (Fig. 16-19). These sandals and shoes are useful for patients who can't tolerate a TCC or for those who need a transitional device after removal of a TCC while they're awaiting custom-made therapeutic shoes and insoles. A modification of the Carville healing sandal can be made from a standard surgical shoe with a total contact direct-molded Plastazote insole.[90]

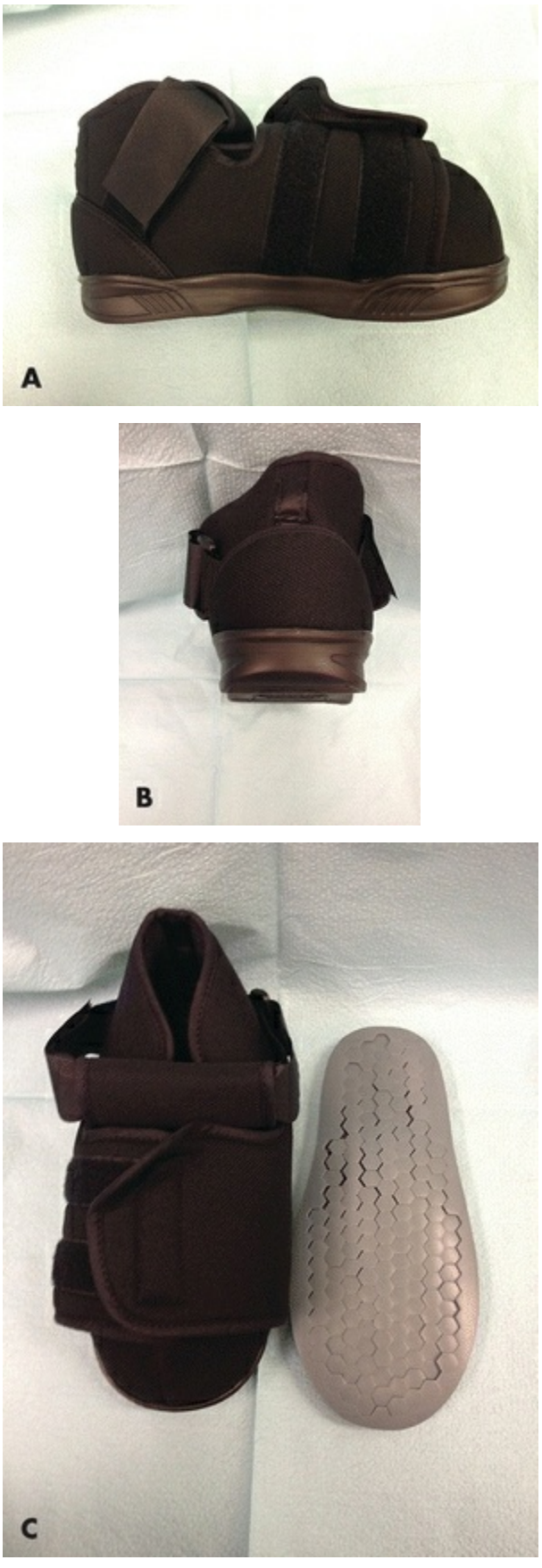


Figure 16-19. (A) Lateral view of healing sandal. **(B)** Rearview of healing sandal.

(C) Top view with off-loading honey comb insole removal (individual pieces can be removed for additional off-loading). (Photos courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

Surgical shoes with a rocker sole design are preferable to the flat design for postoperative use. Royce Medical has a healing sandal that utilizes the Active Hex insole, described above, and can be used as a transitional device after closing the wound.

The Wedge shoe (Fig. 16-20) by Darco products was originally designed to protect the forefoot after elective surgery. This shoe has a sole that's wedged at a 10-degree dorsiflexion angle, effectively removing pressure from the forefoot area. A study by Lair et al.[91] provides support for its role in postoperative patients following surgery on the forefoot. However, these types of shoes aren't well accepted by patients because they're difficult to walk in, they typically cause pain of the contralateral extremity, and they are not safe for use in patients with postural instability. Also, many people with diabetes have equinus and cannot tolerate the negative heel position created by the shoe. Further, suspension of the heel during ambulation increases pressure on the forefoot and stresses the midfoot, a common site for collapse in the diabetic Charcot foot. In a randomized clinical trial that compared TCCs with healing sandals and removable cast boots, patients in the healing sandal group were less compliant and used the device during walking significantly less than did subjects in the TCC group.[92]

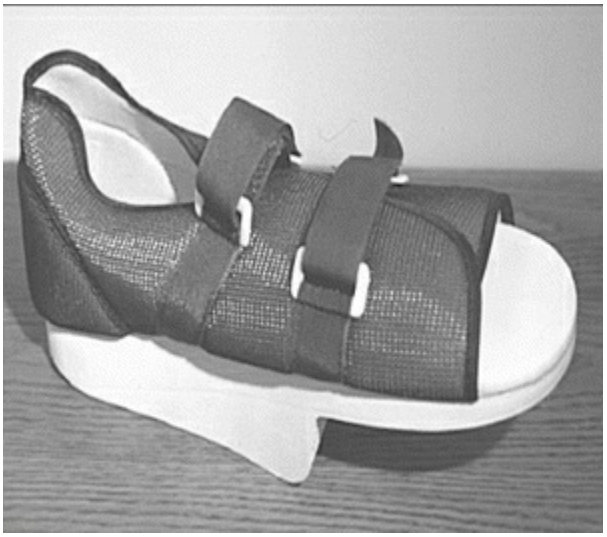


Figure 16-20. Wedged forefront-relief shoe.

Ankle-Foot Orthoses

Custom-made ankle-foot orthoses can be used for lower-extremity pathology, including Charcot fractures, tendon injuries, and neuropathic ulcers. The Charcot Restraint Orthotic Walker (Fig. 16-21), for example, initially was used to treat patients with neuropathic fractures. It provides protection to the neuropathic foot and aids in controlling lower-extremity edema. This device looks like a ski boot; it has a rigid polypropylene shell with a rocker bottom sole.

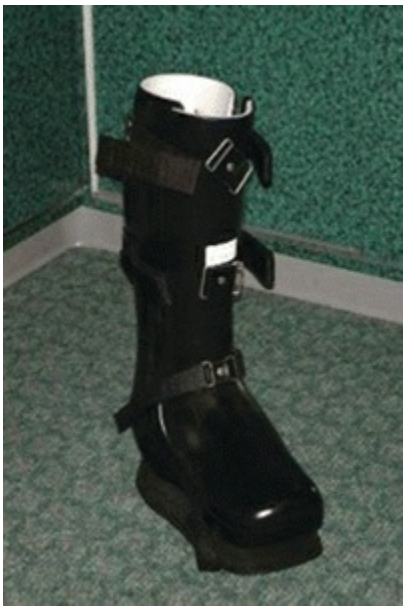


Figure 16-21. Charcot Restraint Orthotic Walker.

The primary drawback to custom-made devices is that they typically cost more than $1,000. If the structure of the foot changes or local edema resolves, the device can no longer be used. Since a number of less expensive, off-the-shelf products are now available to treat neuropathic wounds, custom ankle-foot orthoses are used less commonly. Off-the-shelf devices should be replaced at regular intervals because the materials in the insoles will lose their effectiveness over time.

Negative Pressure Wound Therapy

Negative Pressure Wound Therapy (NPWT) is widely recognized as an effective adjunctive therapy in diabetic foot wound healing and has been reported to increase the rate of healing and promote the formation of granulation tissue[93] (Fig. 16-22). NPWT utilizes the application of

subatmospheric pressure inducing micro tissue deformation that stimulates the healing cascade.[94] Although NPWT can be used for extended periods of time in both the inpatient and outpatient settings, there should be a clear goal of therapy in mind prior to its implementation. NPWT can be used to terminal epithelialization in some cases; however, in the opinion of the authors, NPWT should be principally used as an adjunctive wound bed preparation tool that decreases wound depth and provides a granular wound base for graft (autograft, allograft, or xenograft) acceptance.

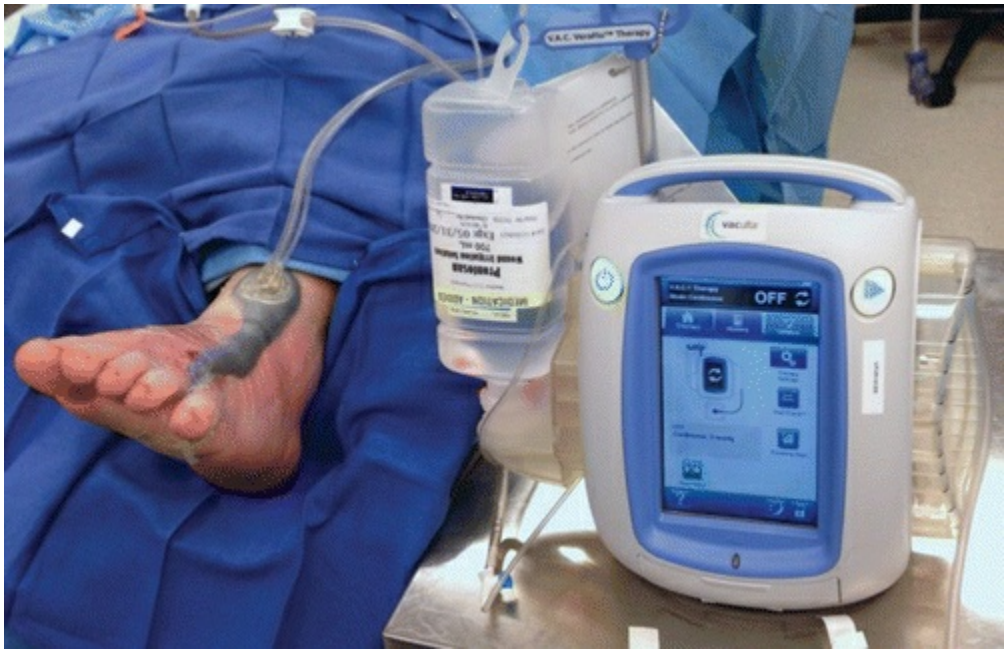


Figure 16-22. Negative Pressure Wound Therapy, inpatient setting. (Photo courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

The next generation of NPWT devices provides greater portability with more reliable mechanisms and potentially most impactful is the use of instillation of a solution in combination with NPWT. Negative Pressure Wound Therapy with Instillation (NPWTi) (Fig. 16-23) allows for preprogrammed cycles of installation of a solution and the application of negative pressure. Although NPWTi was reported in the literature in 1998 by Fleischmann et al. for the adjunctive treatment of infected wounds, this technique and technology was not widely adopted.[95] More recently, the next generation of NPWTi devices has gained much greater interest by the wound care community. Specifically, in the setting of infection, NPWTi may make a significant impact. This is in part because a choice of antimicrobial solutions can be instilled into the wound bed. Lehner et al. report greater than 80% salvage rate for acute and chronically infected total joint implants

utilizing NPWTi.[96] Further, Kim et al. report a significant reduction in the number of operative debridements and length of hospital stay for the treatment of the acutely infected wound requiring hospitalization.[97] The ideal type of solution, dwell time, duration of therapy, and other questions remain regarding NPWTi.[98] However, the growing evidence suggests that NPWTi is becoming a valuable adjunctive tool in wound healing.

Figure 16-23. Next-generation Negative Pressure Wound Therapy

Electrical Stimulation

Several studies have suggested a beneficial effect of electrical stimulation on the acceleration of wound healing, increase of cutaneous perfusion and cellular immunity.[75] Although there is a substantial body of work that supports the effectiveness of electrical stimulation in wound healing, most surgeons have a poor understanding of the technology and its potential applications in patients with diabetes. All living human cells are enveloped by a plasma membrane that operates on the electrochemical physiology of direct current exchange of ions.[99] Injury to the epithelial layer of the human skin disrupts the natural current of these ions, creating an electrical field and changing the movement of epithelial cells and fibroblasts.[100] This cellular migration may be reversed with the use of electrical stimulation by changing the polarity of the electrical field back in its natural electric current.[101]

The application of Transcutaneous Electrical Nerve Stimulation (TENS) to improve skin perfusion and venous flow in diabetic patients is reported frequently.[102] When the human skin becomes hypoxic and releases

cytokines, the expression of vascular endothelial growth factor (VEGF) is increased. Electrical stimulation initiates the release of stored VEGF followed by an increase in gene expression of VEGF. Daily electrical stimulation might allow secretion of VEGF to remain above basal levels throughout the healing process.

Bacterial load and infection seem to be important factors in chronic wounds and delayed healing of leg ulcers of people with diabetes.[103] The bacteriostatic effect of electrical stimulation is described in several experimental studies[104] and may lower the bioburden in the wound bed. Future clinical trials are needed to fully understand the influence of electrical stimulation on infected leg ulcers in diabetics.

Other Adjunctive Therapies

The suggestion that hyperbaric oxygen therapy (HBO) is applicable to lower-extremity pathology has been a point of discussion for many years among healthcare professionals. Most reviews of medical publications about HBO treatment and the diabetic foot report little evidence to support the choice of oxygen therapy as an adjunctive therapy for wound healing.[105] However, a high-quality trial has demonstrated a beneficial effect of HBO on the healing of diabetic foot ulcers.[106] Although a few limitations can be listed about the study, additional randomized control trials need to be repeated to investigate its further implications. Given the high-economic burden of this treatment modality, studies with full health economic analysis are needed before HBO sessions can be routinely incorporated into clinical practice.

Many products have been designed to correct aspects of the wound biochemistry and the cell biology of diabetic ulcers. Some data suggest the use of endothelial growth factor (EGF) to hasten healing.[107] Dressings, skin substitutes, even platelet-derived products are being studied to enhance the healing of chronic ulcers in the diabetic foot.[108]

Antibiotics

Without appropriate wound care, antibiotic therapy is often insufficient to treat infected ulcers. Only clinically infected wounds require an empirical antibiotic regimen, based on the severity of the infection and the likely etiologic agent(s). Mild-to-moderate infections in patients who have not recently received antibiotic therapy may be sufficiently treated with a

therapy aimed solely at aerobic gram-positive cocci (*Staphylococcus aureus, β-hemolytic streptococci*). Severe infections or recent antibiotic therapy require broad-spectrum empirical therapy, to overcome resistant organisms.[31]

Because of limited evidence with which to make choices among antibiotic regimens, the route of therapy and the duration of antibiotic therapy, no single combination appears to be superior to others.[109] In case of osteomyelitis, generally, a duration of at least 4 to 6 weeks is required, and probably a longer duration is needed if infected bone remains.

Topical Antiseptics and Wound Cleansers

Although antiseptics and wound cleansers are ubiquitously utilized, their importance to wound care has been underappreciated. Their value lies in their ability to remove devialized tissue as well as reduce bioburden. Unlike antibiotic therapy, antiseptics are less likely to develop resistance to microbes, a concentrated localized effect, are generally better tolerated, and are widely available. Commonly used antiseptics include Dakin's solution (dilute bleach), dilute acetic acid, polyhexanide, and dilute iodine. In vitro studies have repeatedly demonstrated significant efficacy against a wide range of bacteria, yeast, and fungus including resistant strains.[110,111] In light of the troubling trend of growing antimicrobial resistance, topical antiseptics and wound cleansers may provide an important treatment modality.

Antiseptic therapy can be applied in different ways. With every dressing change, the wound can be washed with an antiseptic solution. It can also be applied onto the wound surface multiple times daily via an antiseptic saturated contact dressing (e.g., 4 × 4 dressing). Continuous or intermittent delivery via NPWT devices can also be used in the acute setting to instill antiseptic solutions.

Along the same category, another widely available yet generally underutilized topical therapy in wound care are surfactants. Surfactants are detergents that break up debris and prevent adherence of foreign materials. Surfactants have been utilized for orthopedic application for many years in the removal of acute contamination as well as biofilm reduction on hardware.[112,113] For example, castile soap is a well tolerated, widely available and has been reported to be effective. Anglen reported that castile soap worked as well as bacitracin solution with less wound healing problems in 458 open lower-extremity open fractures.[114]

Surgery to Heal Wounds and Prevent Recurrence

Is there good evidence that elective or prophylactic foot surgery in patients who have diabetes will prevent ulceration in the future? Armstrong et al.[115] validated a four-tier surgery classification that consists of elective, prophylactic, curative, and emergent surgery. Elective surgery is planned reconstructive surgery in a patient with foot deformity to eliminate pain or to enhance function. Prophylactic surgery is intended to prevent ulcer recurrence. Curative surgery is intended to facilitate wound healing in a patient with an existing foot wound. Emergent surgery is intended to remove infection or devitalized tissue.[115]

There is no evidence that elective surgery reduces the risk of future ulceration. Patients with diabetes should undergo elective foot surgery only if they have severe deformity, pain, or functional limitations that warrant surgery rather than an expectation that surgery will prevent a foot ulcer in the future.

Prophylactic surgery includes toe and bunion deformity correction, Achilles tendon lengthening, and exostectomy. For example, percutaneous lengthening of the Achilles tendon[115] has been shown to reduce plantar foot pressures in subjects with prior ulceration (Fig. 16-24). This type of surgery has been used for both prophylactic and curative treatment. Several authors have reported on the use of Achilles tendon lengthening in patients with forefoot ulcerations due to a tight Achilles tendon associated with limited ankle joint range of motion. The rationale for surgery is that limited active ankle joint range of motion causes more pressure and shear stresses on the ball of the foot, leading to ulceration. Armstrong et al.[116] demonstrated a reduction of about 27% reduction in forefoot loading after lengthening the Achilles.

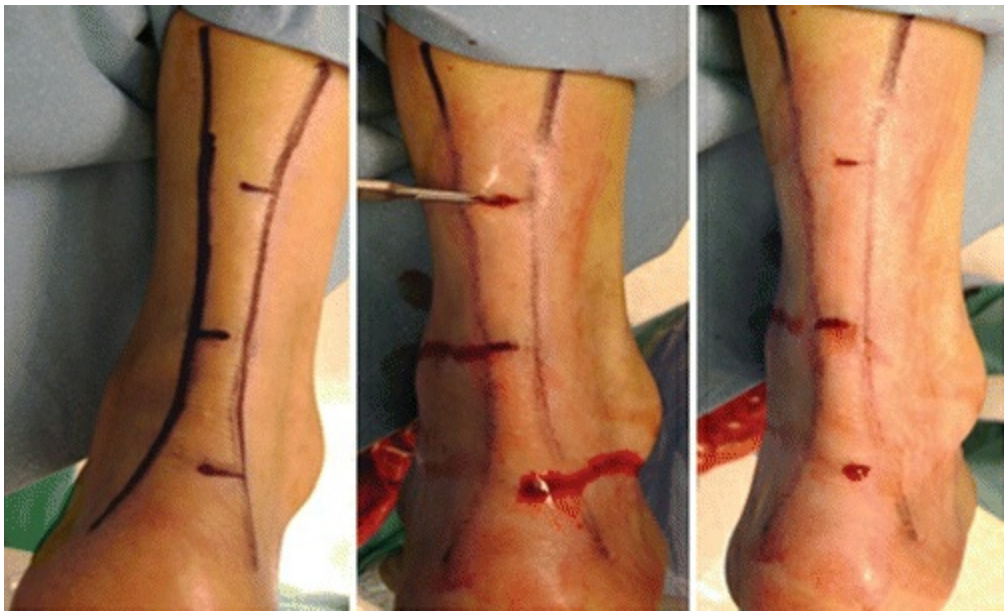


Figure 16-24. Percutaneous Achilles tendon lengthening can assist in forefoot ulcer healing or prevent recurrence by decreasing peak plantar pressure. (Photo courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

The question for physicians and patients is whether the risks of surgery are better than the risk of having a chronic foot ulcer. The risks of infection and amputation from a nonhealing foot ulcer are high. Approximately 10% to 20% of diabetic foot ulcers end in amputation,[117,118] 56% are treated for infection, and 20% develop osteomyelitis.[119–121] Ulcer recurrence, as previously discussed, is about 30% per year when standard preventative therapies are provided. The incidence of ulceration is 50% to 80% when no additional prevention is provided. On the other hand, several authors have reported the results of planned surgical procedures to heal foot ulcers. These studies suggest a high rate of wound healing (91% to 100%) and a low rate of ulcer recurrence after 2 years (0% to 39%).[122–124] If surgery is simply viewed as a prevention tool, in the correct subpopulation, surgery has the lowest reulceration rate. The goal of surgery is to reduce the long-term risk for reulceration by increasing joint motion where it is limited, reducing abnormal pressure points, and repairing structural foot deformities when they are an underlying cause of ulceration. The literature has several reports of retrospective case series, but there is only one randomized clinical trial that reports clinical outcomes with this type of approach. In general, surgery seems to be safe and effective at healing recalcitrant ulcers and reducing the risk of reulceration.

Several clinical studies have described the procedure and clinical

results of Achilles tendon lengthening to prevent ulcer recurrence. Lin et al.[125] reported on a cohort of patients with diabetic foot ulcers that failed to heal after a period of immobilization via a total contact cast (TCC). Using the Achilles lengthening procedure, 93% of patients (14/15) healed in an average of 39 days with no ulcer recurrence in the subsequent 17 months. In a randomized clinical trial, Mueller et al. compared neuropathic ulcer healing in patients using the same procedure versus TCC immobilization.[126] Ulcers healed in all of the patients who underwent surgery (n = 31) as compared with 88% (n = 33) of patients in the TCC group. Patients with Achilles lengthening had less than half the incidence of ulcer recurrence (31% vs. 81%) compared with patients in the TCC group.

Another example of selective foot surgery to reduce the risk of ulcer recurrence involves ulcers of the great toe. Foot ulcers of the great toe are commonly associated with hallux rigidus, or a reduced range of motion of the first metatarsophalangeal joint.[127,128] One of the most common surgical approaches to address hallux rigidus in persons with diabetes is a resectional arthroplasty of the first metatarsophalangeal joint, or more specifically resection of a portion of the base of the proximal phalanx of the great toe (Fig. 16-25). This is also referred to as a Keller bunionectomy. The Keller resectional arthroplasty removes the arthritic joint and allows a pseudoarthrosis to develop in its place. Like the surgery described to increase ankle joint range of motion by lengthening the Achilles tendon, arthroplasty of the great toe has been reported to increase healing of ulcers that have failed other therapies with a much lower rate of ulcer recurrence. Armstrong reported the results of a cohort study of 41 diabetic patients with great toe ulcers.[123] Patients either received resectional arthroplasty of the great toe or standard wound care. The surgery group had faster healing (24 vs. 67 days) and few recurrent ulcers after the surgery (5% vs. 35%).

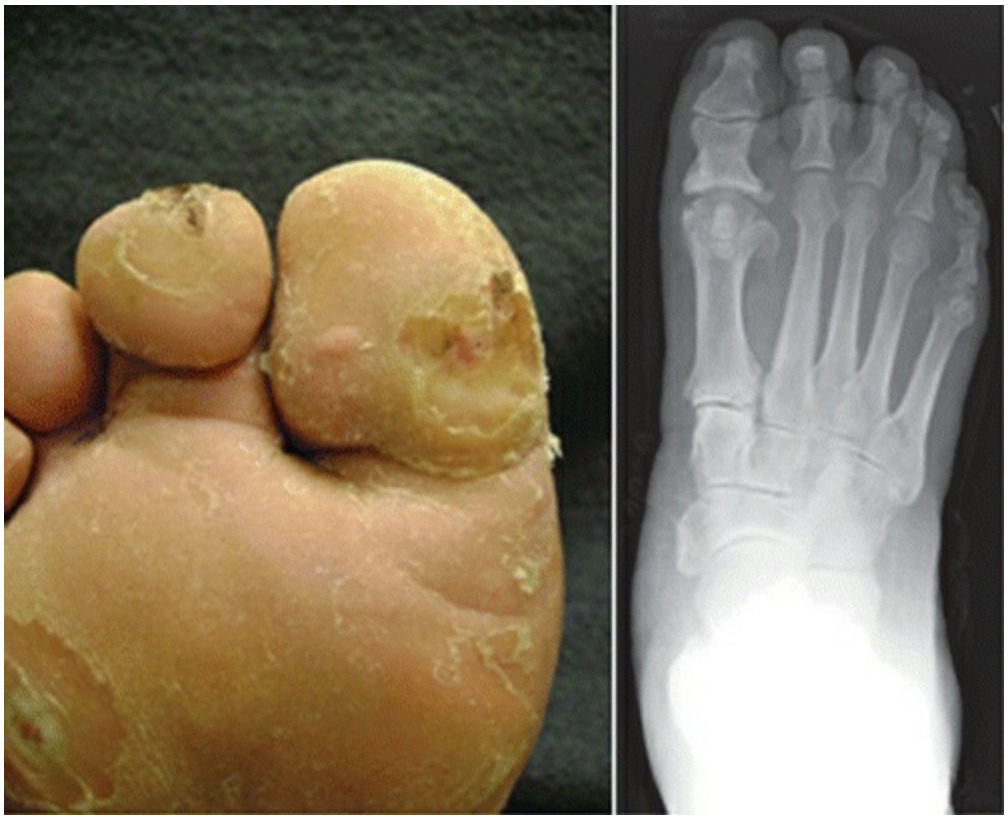


Figure 16-25. Plantar ulceration, healed after resection of the base of the proximal phalanx of the hallux (Keller arthroplasty). Notice the shortened proximal phalanx of the hallux. (Photos courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

Summary

Diabetic foot ulcer care is a challenge for both the patient and the healthcare provider. As the population continues to age, the incidence of diabetes will continue to increase, which will in turn lead to more diabetic wounds. A team approach—with total involvement of the healthcare system and the necessary partnership with the patient—will be the infrastructure for achieving better outcomes of care.

Early assessment for the risk factors for foot ulceration in persons with diabetes is essential. A variety of methods must be used to identify at-risk persons. Appropriate skin care and properly fitting shoes are mandatory for any person with diabetes. Clinicians can choose among many products available to off-load the diabetic foot and to treat the diabetic foot. Infection is an important concern in diabetic ulcers and warrants prompt identification and treatment. Adjuvant therapies coupled with debridement and appropriate dressings can be critical in salvaging the diabetic limb.

Future research is needed to determine appropriate therapy combinations and individual treatment plans for patients with diabetic foot ulcers.

PATIENT SCENARIO

A 51-year-old Caucasian male with diabetes and a neuropathic ulcer to the plantar aspect of his right foot arrives at the hospital (Fig. 16-26A). He was already treated for his foot ulcer in a local hospital wound care center with periodic debridement and hyperbaric oxygen therapy. He has a history of peripheral neuropathy and a high level of activity as he manages a ranch.

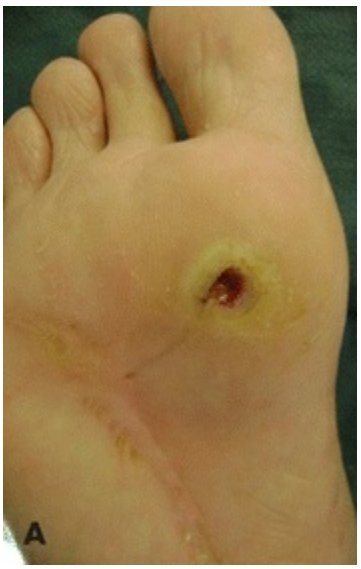
His past medical history includes control visits for his diabetes (12 years), hypertension, hyperlipidemia, and coronary artery disease. The wound on his foot has been managed with standard of care including: off-loading, debridement, enzymatic debridement, moist wound care. His current medications include metformin, glipizide, enalapril, and Toprol XL. He has a significant history of 40 pack-years of smoking, but quit 19 years ago. He denies any alcohol consumption.

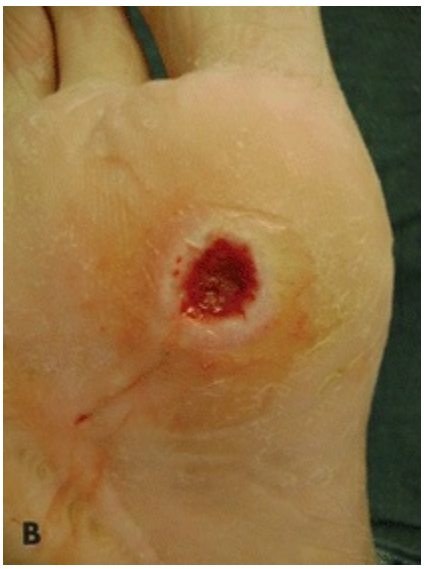
His physical examination reveals an obese man, very pleasant and oriented. He demonstrates barely palpable dorsalis pedis pulse, barely palpable posterior tibial pulse in both feet. The capillary filling time is less than 3 seconds. He demonstrates loss of protective sensation to 10/10 sites in both feet. He has absent pedal hair and dry, xerotic skin. The patient is unable to feel the Semmes-Weinstein monofilament at any site tested, revealing significant bilateral neuropathy. He has no pain or temperature sensation in either foot. His right foot has an ulcer under the head of the first metatarsal; the base of the ulcer is red with surrounding hyperkeratotic tissue, undermining the skin margins. The ulcer measures 1.9 × 1.2 × 0.3 cm after debridement (Fig. 16-26B). No bone can be palpated when the wound is examined with a sterile probe. Biomechanical evaluation reveals a plantar-flexed, non–weight-bearing first metatarsal with significant equinus bilaterally. Laboratory evaluation is within normal limits. Radiographs reveal no foreign body, nor signs of osteomyelitis.

The wound is assigned a grade of 1A on the University of Texas classification system (Table 16-3). There are concerns about his blood flow status. Noninvasive studies are ordered and reveal a slightly decreased left ABI of 0.91 and biphasic waveforms at the anterior tibial artery and the posterior tibial artery. Transcutaneous oximetry

measurements are performed and demonstrate an increase (33 to 54 mm Hg) at the midfoot level. The vascular surgeon is consulted and recommends that the ulcer has to heal without curative surgery.

The wound is stagnant for a period of 2 months. Off-loading devices attempted include removable CAM boot, and a half (wedge) shoe. Weekly to biweekly debridement with total contact cast allows the wound to heal in 6 weeks (Fig. 16-26C).





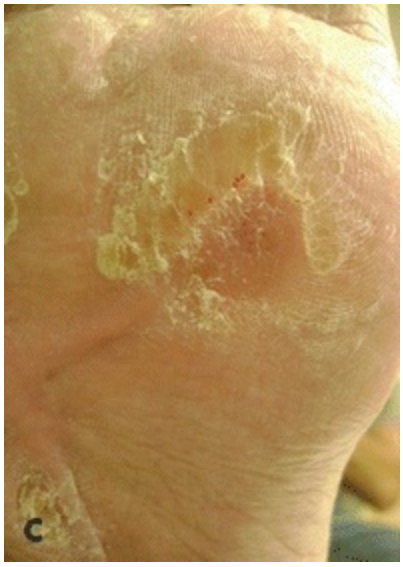


Figure 16-26. (A) Ulcer appearance at the time of presentation. **(B)** Ulcer appearance after debridement. **(C)** Complete epithelialization of ulcer at 6 weeks. (Photos courtesy of S. van Asten and J. Lafontaine, MD. Used with permission.)

Show What You Know

1. According to the ADA (2001), how many people are unaware that they have diabetes mellitus?

A. 50%
B. 30%
C. 75%
D. 25%

2. The single leading cause of lower-extremity amputation is:

A. diabetes mellitus.
B. lymphedema.
C. arterial occlusion.
D. venous disease.

3. According to the ADA, good skin care includes all of the following except:

A. keeping skin clean and dry.
B. applying moisturizers between toes.
C. avoiding very hot showers and tub baths.
D. checking feet daily for cracks or fissures.

4. Off-loading strategies must be tailored to the age, strength, activity, and home environment of the patient.

A. True
B. False

5. For which of the following treatment strategies have healing rates for diabetic foot ulcers been found to be comparable to those with Total Contact Casts (TCC)?

A. Wedged shoe
B. Half shoe
C. Wide-toe shoe
D. Walker boot rendered irremovable (ITCC)

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Sickle Cell Ulcers

17

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Objectives

After completing this chapter, you'll be able to:

- understand the pathogenesis of sickle cell anemia (or sickle cell disease)
- discuss the pathogenesis of sickle cell ulcers
- differentiate sickle cell ulcers from arterial and venous ulcers
- implement protocols for prevention and treatment of complications of sickle cell ulcers.

Sickle Cell Disease

Sickle cell ulcers are a complication of sickle cell disease, an inherited genetic disorder of the oxygen-carrying hemoglobin in red blood cells. Sickle cell anemia (or sickle cell disease) was first reported in 1910 by Dr. J.B. Herrick.[1] It is the result of a mutation on the β-globin gene of the cellular DNA. This causes an abnormal amino acid, valine, instead of glutamic acid to be inserted into the β-globin chain of the normal hemoglobin unit resulting in hemoglobin S or sickle hemoglobin.[2–5] This abnormality results in the polymerization of the hemoglobin when it is deoxygenated. This polymerization is associated with a loss of water from the cell. This polymerization and dehydration of the cells lead to the distortion and sickle shape of the red blood cells and damage to the cell membranes.[3,5] These abnormalities result in occlusion of small blood vessels and capillaries, causing ischemia of the tissues served by these vessels.

Sickle cell disease is primarily seen in black individuals and is more prevalent in the United States and Africa. The disease is seen in two main

forms: (1) homozygous sickle cell disease, the most severe form, occurring when the individual receives a gene for the abnormal hemoglobin (hemoglobin S) from both the mother and the father, and (2) heterozygous sickle cell disease, the less severe form, occurring when the individual receives only one gene for the abnormal hemoglobin from either the mother or father and the other gene is for normal hemoglobin.

Prevalence and Incidence

The patient with homozygous form of sickle cell disease is most likely to develop a sickle cell ulcer. Studies have shown that males are more likely to develop leg ulcers due to sickle cell disease than females.[6] The same study found that 5% of males with sickle cell disease who were over age 10 had sickle cell ulcers,[6] and 75% of patients over age 30 had a sickle cell ulcer at some time during the course of their disease.[7] With over 80,000 patients in the United States with sickle cell disease,[8] this makes the number of patients with sickle cell ulcers significant.

Practice Point

Lower extremity ulcers in young black patients, especially males, could be due to undiagnosed sickle cell disease.

Ulcer Pathogenesis

The abnormal hemoglobin molecule in the red blood cell in the patient with sickle cell disease does not affect the amount of oxygen the red blood cell can carry. After the red blood cell and its hemoglobin give up the oxygen to the tissues, the abnormal hemoglobin causes the red blood cell to lose its water content and polymerize resulting in the red blood cell distorting, becoming rigid, and deforming into the sickle shape (Fig. 17-1). Unfortunately, while the cells are in the sickled shape, they tend to increase blood viscosity and become "sticky." This results in slowing of the blood flow in small vessels and subsequent clotting of the vessels especially in the postcapillary venules,[5] causing ischemia of tissues and organs. Over time, the patient suffers repeated episodes of pain, tissue damage, and, eventually, organ failure. Because the cells become damaged while they are in the

sickled shape, they are removed from the circulation faster than normal, resulting in anemia (Table 17-1). When the red blood cell is reoxygenated and regains its water content, it can regain its normal shape, but a significant percentage of sickle cells do not regain their normal shape and remain irreversibly sickled despite being fully oxygenated.[5] This leads to an even more rapid removal of these irreversibly damaged cells from the circulation.

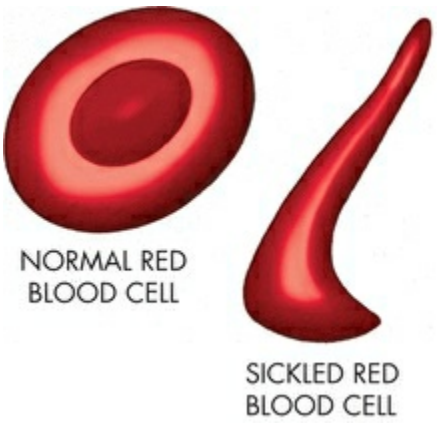


Figure 17-1. Sickled cell. The diagrams show a normal red blood cell and a sickled cell.

Table 17-1 Conditions Associated with Sickle Cell Anemia

Complication	Cause
"Crisis" with fever and pain	Sickling of cells due to abnormal hemoglobin
Pain in bones, joints, and back	Sickling of cells and ischemia of tissues
Severe abdominal pain	Sickling of cells and ischemia of tissues
Pregnancy problems Fertility problems	Uncontrolled sickling of cells
Increased infections Pulmonary Urinary tract	Deficient immune response
Salmonella osteomyelitis	Ischemia of bones, bone infarcts, sepsis
Chronic leg ulcers	Sickling of cells and ischemia of tissues
"Hand–foot" syndrome	Sickling of cells and ischemia of bones
Avascular necrosis of femoral or humeral head	Ischemic necrosis of bones due to sickling
Visual problems	Ischemia of retina due to sickling
Pulmonary infarction	Ischemia of lung due to sickle cell emboli
Congestive heart failure Cardiac murmurs EKG abnormalities	Myocardial ischemia
Jaundice	Hemolytic anemia Gallstone production and obstructive jaundice
Cirrhosis of liver	Ischemia of liver and cell necrosis
Hepatitis	Multiple blood transfusions
Enlarged spleen (infancy only)	Increased blood production
Splenic infarction (late teens, adult)	Ischemia of tissue due to sickling of cells
Renal dysfunction Hematuria	Ischemia of kidney with infarction of tissue
Renal vein thrombosis	Sickling of cells
Priapism (especially in children)	Sickling of cells
Impotence	Damage of penis by priapism and ischemia
Anemia	Hemolysis of abnormal cells
"Aplastic crisis"	Failure of bone marrow to produce cells due to infarction of marrow
Folate deficiency	High folate requirement of hemolytic anemia

Adapted from Conley, C.L. "The hemoglobinopathies and Thalassemias," in P.B. Beeson, W. McDermott, eds. *Textbook of Medicine*, 13th ed. Philadelphia, PA: W.B. Saunders Co., 1971:1501-503, with permission.

Although the underlying pathophysiology remains ischemia due to occlusion of the microcirculation, sickle cell ulcers have been associated with homozygous hemoglobin SS disease, trauma, infection, severe anemia, warm temperatures,[9,10] venous insufficiency,[11,12] and indications of severe sickle cell disease as reflected by increased cardiovascular risk, pulmonary hypertension, and lower body mass index.[13]

These ulcers are most likely to occur in the malleolar area of the lower

extremities. Occlusion of the small vessels in the microcirculation results in chronic damage to the microcirculation of the skin around the ankle including injury to the capillary walls, thickening of the intimal lining of the capillary, and increase in permeability of the vessel wall, allowing macromolecules to escape into the tissues.[14–16] These changes can also result in ischemia of the tissues making them more susceptible to minor trauma and less able to heal.[14,17] As a result, these areas are more likely to be the sites of skin breakdown and ulceration. Recently, new information shows that the endothelial dysfunction in sickle cell disease may be due to impaired bioavailability of endogenous nitric oxide resulting in unrestrained vasoconstriction of the small vessels, ischemia of the skin, and skin necrosis.[17,18]

Diagnosis

Medical History

Evaluation of the patient with a suspected sickle cell ulcer is of utmost importance so that the correct diagnosis can be made and appropriate treatment planned. The patient's medical history should be recorded, and the events surrounding the ulcer development should be investigated. Is this the first ulcer the patient has had? How long has the ulcer been present? How did the ulcer first develop? Was there trauma to the area? How did the area first look? Any history of lower extremity edema; unexplained swelling of the hands, feet, or knees; osteomyelitis; episodes of abdominal or joint pain; episodes of severe unexplained pain; recurrent urinary tract infections or pneumonia; or anemia should be noted. Sickle cell patients are prone to develop unexplained episodes of fever, which tend to resolve without therapy. These patients may carry a diagnosis of FUO (fever of unknown origin).

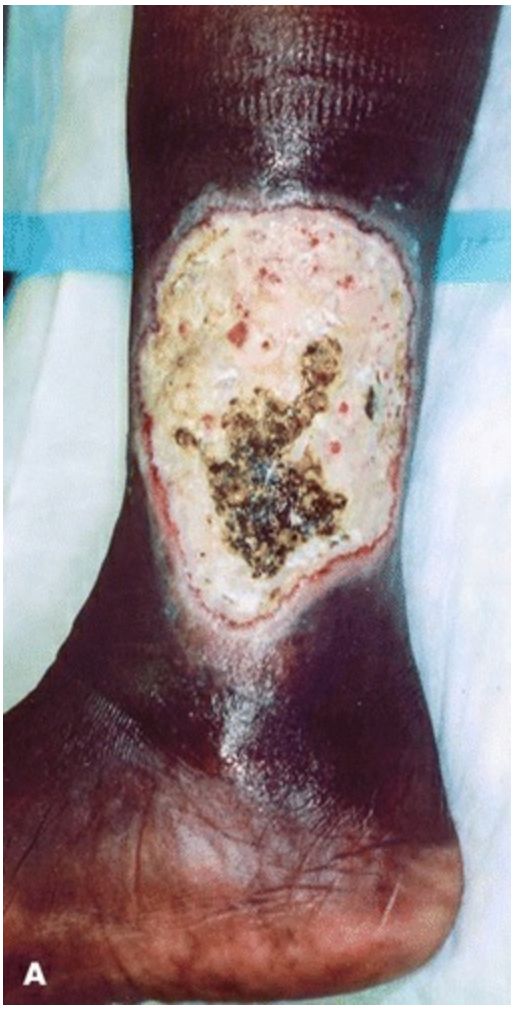
Physical Examination

A complete physical examination should be part of the evaluation of the patient. Vital signs, especially temperature, should be taken because, as mentioned above, unexplained fever may be a sign of sickle cell disease. Cardiac and pulmonary exam may reveal signs of cardiovascular disease and pulmonary hypertension. These conditions are more frequent and more severe in sickle cell patients with ulcers than in those without ulcers.[13]

Abdominal examination can detect enlargement of the liver. The spleen may be enlarged in early childhood as it is a primary blood forming organ but is usually small in later life due to ischemic infarction.

The presence of scars on the extremities indicates previous ulcer incidence. The location of the ulcer or ulcers is important as most sickle cell ulcers are found on the lower one-third of the leg and usually over the medial or lateral malleoli (or both) of the ankle[10]; however, less common sites include any area with little subcutaneous tissue such as the anterior tibial area, dorsum of the foot, and the Achilles tendon area.[19] The size of each ulcer should be measured by determining the length and width or by using one of the more advanced measuring modalities described elsewhere in the book. (See Chapter 6, Wound Assessment.)

The presence of an ulcer in a patient with varicose veins, venous insufficiency, and sickle cell disease can be especially troublesome as it may lead to misdiagnosis (Fig. 17-2). In addition, venous incompetence in the patient with sickle cell disease may predispose him to develop an ulcer and can be correlated with the development of a recurrent sickle cell ulcer.[12] Noninvasive venous studies have shown that venous insufficiency *is not* a primary contributing factor to the development of a sickle cell ulcer.[19] These studies can be helpful in establishing the correct therapeutic approach in these patients.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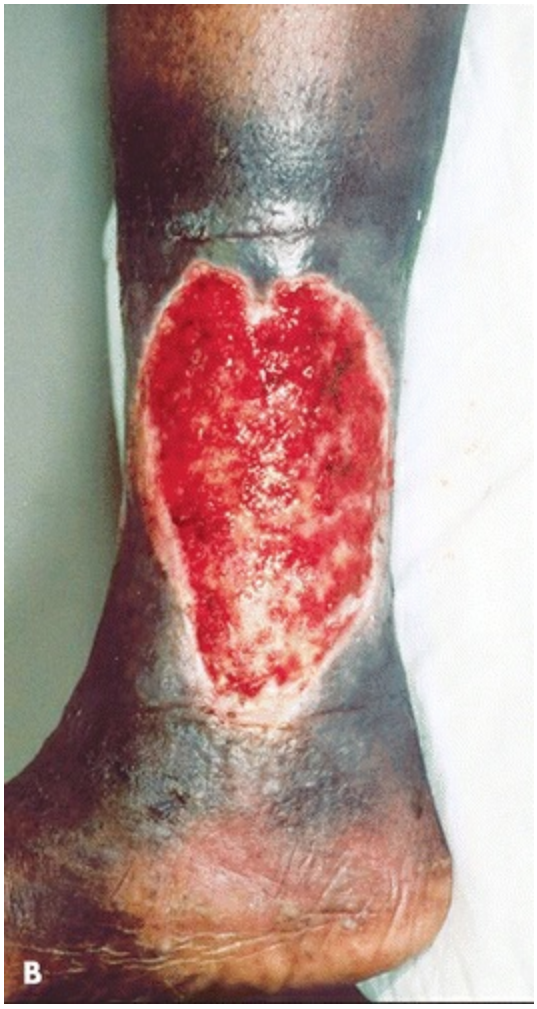


Figure 17-2. Undiagnosed sickle cell ulcer treated initially as a "venous ulcer." **(A)** Predebridement. **(B)** Postdebridement. (Used with permission from T. Treadwell, MD.)

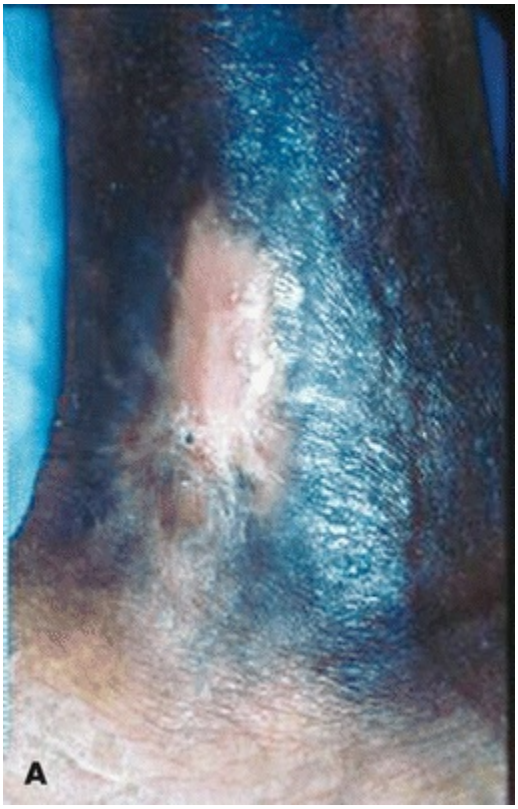
Practice Point

Misdiagnosis—and thus, mistreatment—of sickle cell ulcer as a venous "stasis" ulcer makes it imperative to get the diagnosis correct.

Ulcer Assessment

Examination of the wound bed is essential to determine the presence of granulation tissue or fibrinous material ("slough") (Figs. 17-2 and 17-3). In addition, the presence of periulcer erythema or cellulitis should also be documented. The presence and character of any drainage should be noted.

Tenderness of the lower extremity to palpation or the presence of pain in the ulcer or surrounding area should be also recorded. A vascular examination should always be performed by checking for the dorsalis pedis and posterior tibial pulses to be sure the patient has adequate blood flow. Documentation of their presence or absence is important. If there is a question about the adequacy of the circulation, the patient will need to be referred for noninvasive vascular studies or arteriography. The microcirculation in the periwound area can be evaluated with pulse oximetry, laser Doppler, and $TcPO_2$ measurements. Measurements of tissue oxygen levels by whatever method showed that readings are lower in patients with ulcers than sickle cell patients without ulcers.[13]



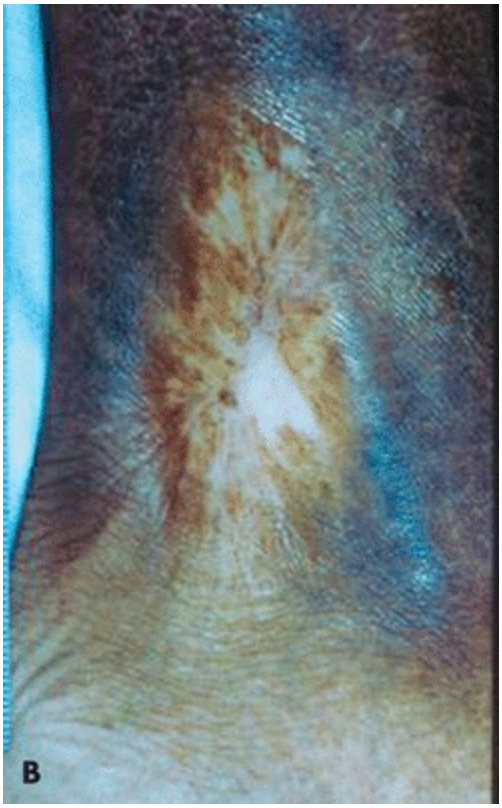


Figure 17-3. Recurrent sickle cell ulcer treated with tissue-engineered skin. **(A)** Healed 8 weeks postapplication. **(B)** Ulcer remains healed 8 months later—note improved scar. (Used with permission from T. Treadwell, MD.)

Practice Point

A complete history and physical examination are vital when evaluating a patient with a sickle cell ulcer.

Laboratory Assessment

Laboratory evaluation can help make the diagnosis if a patient is seen with a nonhealing ulceration of the ankle area. It, also, will help identify the severity of the patient's sickle cell disease. If the patient does not have a diagnosis of sickle cell disease, but it is suspected, blood tests that check for anemia, sickle cells, and abnormal hemoglobin should be ordered. This usually involves a complete blood count (CBC) with platelet count, sickle "prep," and a hemoglobin electrophoresis. Although hemoglobin electrophoresis is considered the diagnostic tool of choice, it has its limitations especially if the patient has had recent blood transfusions.[9] In

such a case, referral to a hematologist may be necessary. If the patient with known sickle cell disease has an ulcer, the laboratory workup should consist of a CBC with differential white blood cell count, platelet count, and reticulocyte count.

Laboratory evaluation has shown that sickle cell ulcer patients, in addition to lower hemoglobin levels, have higher markers of hemolysis such as elevated levels of lactate dehydrogenase (LDH), bilirubin, aspartate transaminase (AST), alkaline phosphatase, and uric acid, serum amino-terminal probrain-type natriuretic peptide, and higher reticulocyte counts than do patients with sickle cell disease with no ulcers.[13,14] High ferritin levels are noted and are felt to be due to the chronic inflammatory state of these patients with ulcers.[13] High levels of uric acid are seen in patients with sickle cell ulcers. It has been determined that high uric acid levels may be an indicator of microvascular problems even if there is no current sickle cell ulcer.[13]

Patients with sickle cell disease are especially prone to develop infections; therefore, it is important to obtain a wound culture using appropriate technique if the wound bioburden appears elevated. We have found that a significant number of patients with sickle cell ulcers have wounds covered by biofilm, which must be removed before the ulcer can heal (see Fig. 17-2). The most frequent way to remove a biofilm is with sharp debridement, but recently, we have turned to ultrasonic debridement techniques, which are less painful for the patient. Dressings that enhance autolytic debridement can, also, be helpful. Following debridement, the use of the enzymatic debriding agent, collagenase, will help maintain the debrided state.[20] Other methods for removing biofilms will be available in the near future. (See Chapter 7, Wound Bioburden and Infection.)

Infection and Osteomyelitis

Patients with sickle cell disease and a deep, painful ulcer should be evaluated radiologically for the presence of osteomyelitis especially if the patient has fever or leukocytosis. Patients with sickle cell disease are prone to developing Salmonella osteomyelitis.[9] Radiologic evaluation can be done by several methods. Plain film x-rays are the least sensitive method and usually don't show any evidence of osteomyelitis until late in the course of the infection. In addition, they can be confusing in the patient with sickle cell disease as the disease can cause periosteal elevation and other bone changes mimicking osteomyelitis.[9] Nuclear medicine bone scans are slightly

more helpful, but it must be remembered that routine bone scans only detect areas of inflammation. For this reason, if the patient has an ulcer overlying the bone in question, the bone scan is not helpful without special imaging techniques. Magnetic resonance imaging appears to be the imaging modality of choice in terms of sensitivity and specificity.

It has been suggested that bone biopsy and culture may be the only definitive way to determine if osteomyelitis is present,[10] but it should be done with great care so as not to cause an infection in the bone. Biopsy of the wound bed and wound margin may be advisable if the ulcer has been present for over 3 months, does not respond to therapy, or just doesn't "look right." This should be done to rule out the possibility of malignancy and can help with the diagnosis.[21]

Pain

It must be remembered that sickle cell ulcers are extremely painful. Pain assessment is an area that must not be overlooked as, often times, the patient is fearful of experiencing even more pain with your evaluation and treatment and may not want you to look at the ulcer much less touch it. This makes treatment of these ulcers very difficult. (A useful pain assessment tool for evaluating a patient's pain is outlined elsewhere in this book [see Chapter 12, Pain Management and Wounds].) It has been the author's experience that if the provider does not address the pain problem, many of the patients will not return for follow-up care. Indeed, most patients would rather keep their ulcer than deal with potentially being in more pain than they already are. For this reason, any procedure to the wound must be preceded by some type of anesthesia: topical anesthesia (Xylocaine ointment or EMLA cream), injectable local anesthesia, regional anesthesia, or general anesthesia. Some of these techniques will require hospitalization of the patient.

Pain control can be managed with topical anesthetic agents. Topical Xylocaine ointment or EMLA cream can be used on a regular basis for pain control. Applied every 4 to 6 hours, these agents can make the patient's daily activities much more manageable. It also makes dressing changes more comfortable. Other therapies, including opioid analgesics and regional medications (Xylocaine patches), are useful but many times have to be managed by a pain specialist. (See Chapter 12, Pain Management and Wounds.)

Because of the painful nature of sickle cell ulcers, debridement, biopsy cultures, or even dressing changes can be a challenge. If biopsy cultures are indicated, pain management will be required, but the practitioner should be

aware of recent studies about the effects of anesthetics on bacteria. Berg et al.[22] have shown that EMLA cream, a topical anesthetic agent commonly used before doing wound biopsies, is highly antibacterial. Within 1 hour of exposure to EMLA cream, most common bacteria are killed, including strains of *Staphylococcus aureus* (both methicillin-resistant and methicillin-sensitive strains), *Streptococcus pyogenes*, *Escherichia coli,* and *Pseudomonas aeruginosa.* Injected solutions of 1% lidocaine are, also, antibacterial for the same organisms but at greater than 2 hours after local injection. It is the recommendation of the author that EMLA cream not be used for anesthesia when biopsy cultures are being done. Local injection of preservative-free 1% lidocaine would be satisfactory to use if the biopsy culture is done within 2 hours of the injection and the specimens are promptly plated.[22]

Practice Point

Sickle cell ulcers are extremely painful. Evaluation and treatment of the patient's pain should be a top priority and should be the first steps in instituting therapy for the ulcer.

Treatment

Treatment of sickle cell ulcers can be challenging and frustrating. Even in this day of evidence-based therapies, it is noteworthy that there are no published trials of treatments of sickle cell ulcers.[10] One of the more interesting findings about the treatment of sickle cell ulcers is that most ulcers will heal with prolonged bed rest.[10] Obviously, this is not a practical therapy as extended hospitalization is no longer possible, and complete bed rest at home is not realistic. However, any therapy that results in a long period of immobilization, such as from operative intervention, must take into account that the bed rest is healing the ulcer, not necessarily the treatment. With this in mind, one must begin with the basics of what we know constitutes good wound care.

The basics of good wound care include debridement of devitalized tissue, control of infection, assurance of adequate circulation, maintenance of a moist wound environment, and control of the edema in the extremity. The major addition in the treatment of sickle cell ulcers is control of wound

pain. Many times, the ulcers are so painful that manipulation of the wound is impossible. Treatment of the wound with topical anesthetics has been previously addressed and must not be overlooked. If the therapy for the wound is painful, most people will forgo your advice and treat the ulcers themselves in ways that do not cause more pain. It is unfortunate that many of these patients are labeled as "noncompliant" when it is a poor choice of therapy by the clinician and not the compliance of the patient that should be in question. Once the pain is controlled, the wound can be debrided and treated, as indicated. Use of dressings that enhance autolytic debridement or enzymatic debridement should be strongly considered. Debridement with sterile maggots can be a consideration and is less painful, but convincing the patient may be difficult!

The evaluation of sickle cell ulcers for infection and the evaluation of the patient for circulation problems have been covered previously. The importance of moist wound care has been known since Winter's publication in 1963.[23] It is now known that wounds treated with wet-to-dry dressings do not heal well and removing a dry dressing that adheres to the wound causes pain and reinjures the wound.[24] Wounds treated by moist wound healing techniques and dressings heal faster, are less painful, and have less scarring than those allowed to dry out. There are numerous wound dressings currently available, which will maintain a moist wound environment. (See Chapter 9, Wound Treatment Options.)

If the wound is felt to be infected, topical antimicrobials should be considered. Oral or IV antibiotics are only indicated if the patient has a leukocytosis, cellulitis, or fever.[25,26] Silver dressings have become popular in the treatment of the wound with a clinically significant bacterial burden (critical colonization) or with frank infection. There are numerous silver dressings available for use on these wounds. It is suggested that once the bacterial burden is under control, the silver dressing should be discontinued and other moisture control dressings used. This is because of the potential for toxicity of the silver to the growing tissues.[27,28] Dressings containing Cadexomer iodine are useful in treating wounds critically colonized with bacteria or wounds, which are infected.[29,30]

Since a number of patients with sickle cell ulcers have a low body mass index and are underweight, attention to nutritional factors should be remembered.[13] Restoration of normal protein, vitamin, and electrolyte levels should be considered critical in the treatment of these patients.[31] It has been shown that many patients with sickle cell ulcers are zinc deficient.

Some early studies showed that oral zinc sulfate (200 mg three times/day)[32] is helpful in the treatment of patients with sickle cell ulcers.

Another major issue is to manage the edema of the extremity associated with the ulcer. It is known that edema fluid inhibits mitogenic activity and DNA synthesis, the cytokine environment in edema fluid is more proinflammatory, the protease activity is higher in edema fluid, and growth factor levels are decreased in edema fluid. All of these factors contribute to impaired healing of any wound including sickle cell ulcers.[33] Compression bandaging should be considered in the treatment of any patient with a lower extremity ulcer and edema.

The use of the tissue-engineered skin in the treatment of sickle cell ulcers has met with some degree of success.[34] Prior to applying any advanced therapy product, the wound bed must be well prepared, which means assuring that the wound bed has been debrided to remove all necrotic tissue, that infection has been controlled, and that the wound environment has been optimized.[35] To achieve the goal of wound environment optimization, Treadwell et al.[36] recommend pretreating the wound with a protease modulating agent for 2 to 3 weeks to reduce the abnormal protease levels. These agents include oral or topical doxycycline or an oxidized, regenerated cellulose (ORC) collagen product.[37,38] His early data show that pretreatment of any chronic wound with these protease modulating agents prior to application of human skin equivalent improves the healing rate.[36] Once treated with the tissue-engineered skin product, the patients can experience significant relief of their pain as well as healing of their ulcer (see Fig. 17-3A). It is also of note that sickle cell ulcers treated with tissue-engineered skin seem to have a more "normal" appearing and stable scar (see Fig. 17-3B).

The use of the split-thickness skin graft or pinch grafts to treat patients with sickle cell ulcers may be a reasonable therapeutic alternative. However, the procedures require hospitalization and anesthesia, thus making them less cost-effective. It has also been reported that split-thickness skin grafting has a very low success rate in healing sickle cell ulcers, and in those that do heal, the recurrence rate is very high.[39] Success has been noted with the use of muscle flaps, myocutaneous flaps, and free flaps to cover large lower extremity sickle cell ulcers[40,41] but not uniformly.[42]

A mainstay in the treatment of sickle cell disease is hydroxyurea, which is known to improve symptoms associated with the disease by increasing

the levels of fetal hemoglobin (HbF). This results in a significant decrease in the polymerization of hemoglobin S and sickling of the red blood cells.[5] Unfortunately, hydroxyurea is known to cause leg ulcers in patients taking this medication.[43,44] The medication must be stopped before the ulcer will heal.[44] Fortunately, it has been found that ulcers related to hydroxyurea therapy respond promptly to treatment with tissue-engineered skin.[45]

Transfusion therapy has been tried in the treatment of patients with sickle cell ulcers who have been resistant to all other therapies. The goal of the therapy is to keep the hematocrit between 30 and 35 volume percent and the percentage of normal hemoglobin (hemoglobin A) greater than 70% of the total.[39] The transfusions are continued until the ulcer heals or for 6 months at which time they are discontinued. Unfortunately, 20% to 30% of patients treated with multiple blood transfusions can develop antibodies to blood products minimizing their use when anemia is profound.[39] The possibility of iron overload with this therapy must be considered.[15]

Another interesting approach to treating sickle cell ulcers has been the use of IV arginine butyrate. The concept is that the arginine butyrate will change the concentration of abnormal hemoglobin, thus allowing the wounds to heal. Studies have shown reasonable success with this method,[46–48] but no randomized controlled trials have been done as of this time.

Other therapies for sickle cell ulcers include medications, such as pentoxifylline (Trental) and the topical growth factor, molgramostim (GM-CSF)[49]; negative pressure wound therapy; hyperbaric oxygen therapy; and electromagnetic stimulation. Other therapies are still considered experimental, and their utility in treating these patients with difficult wound problems will be determined by future studies.

Recently, it has been suggested that strategies for the treatment of sickle cell ulcers should fall into one of the following categories: (1) agents with reduce or prevent sickle cell dehydration, (2) agents with reduce sickle cell–endothelial adhesive events, (3) nitric oxide or nitric oxide–related compounds, or (4) antioxidant agents.[50] These strategies deal with the unhealed ulcer based on the pathophysiology unique to the sickle cell ulcer. Currently, most of the work on these strategies is still in the research phase but hold promise for future treatments.[5,50] One exception is the role of nitric oxide in the treatment of the sickle cell ulcer.

Nitric oxide is a strong vasodilator, which also effects activation of white blood cells, platelets, and endothelial cells. Nitric oxide is produced by endothelial cells in the vasculature from the amino acid, L-arginine. It has

been noted that nitric oxide levels are decreased in patients with sickle cell ulcers especially during episodes of crisis.[50–52]

Based on this information, Aslan[17] suggested that sickle cell ulcers may be due to a decrease in microcirculatory smooth muscle nitric oxide with resultant vasoconstriction, tissue ischemia, and necrosis and that nitric oxide supplementation may improve healing of the ulcers or even help prevent them. Because of this information, we have been treating patients with sickle cell ulcers with a combination of L-methylfolate, pyridoxal 5′-phosphate, and methylcobalamin, the combination of which has been shown to reduce vascular homocysteine levels and raise nitric oxide levels in the microcirculation of wound bed and periwound skin compared to the skin away from the ulcer.[52] An increase in nitric oxide in the microcirculation is reflected by vasodilatation and an increase in tissue $TcPO_2$. An example is the patient represented in Figure 17-4. The "pretreatment" evaluation shows the periwound $TcPO_2$ (#1) and the reference point (#2) levels of 16.0 and 69.1, respectively. The periwound level is below what would be expected for healing to occur. After 1 week of oral therapy with the medication ("posttreatment"), the periwound $TcPO_2$ has risen to 46.1 (#2), while the reference point (#1) has stayed essentially the same at 67.2.[53] With this improvement, the wound began to heal. For these reasons, we recommend all of our sickle cell ulcer patients be treated with this therapy. Recently, we have had the opportunity to utilize a device that delivers topical nitric oxide directly to the wound bed to assist in healing. This is the preferable way to deliver the nitric oxide so that systemic effects can be avoided in these patients.[54] It is our impression that the sickle cell ulcers heal faster with these therapies, but the actual healing rates and whether this treatment will reduce the incidence of sickle cells ulcers or their recurrence are yet to be determined.

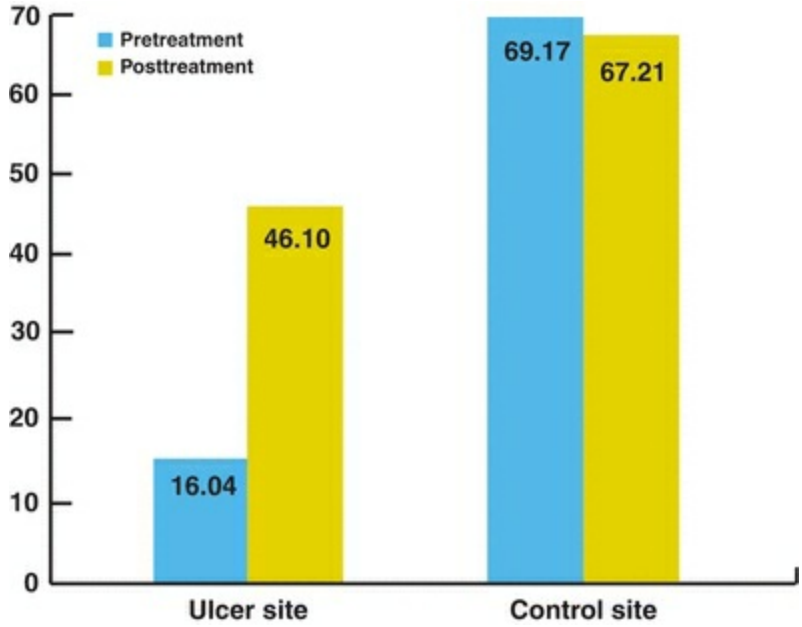


Figure 17-4. Treatment of sickle cell ulcer with nitric oxide–producing medication. Before treatment, $TcPO_2$ levels at the ulcer site and control site were 16.04 and 69.17, respectively. The periwound $TcPO_2$ level is below what would be expected for healing to occur. After 1 week of oral therapy with the nitric oxide–producing medication Metanx, there was marked improvement in the periwound $TcPO_2$ while the control site stayed essentially the same.[25] With this improvement, the wound began to heal. Based on this, we treat all of our sickle cell ulcer patients with this medication.

Sickle cell ulcers will respond better to any therapy if the sickle cell disease is under control. If the patient's anemia is profound (below 5 grams Hg/100 mL) or if the abnormal hemoglobin-containing cells represent greater than 50% of the total volume of red blood cells, the success of any therapy is problematic. This has recently been supported by work reported by Eckman and Platt.[39]

Preventing Ulcers

Prevention of sickle cell ulcers is paramount. The patient with sickle cell disease must be instructed in the importance of good skin care and methods to keep the skin moisturized and to avoid trauma to the lower legs and ankles. For example, the patient should use insect repellants to avoid insect bites. It has been noted that edema is the single most common occurrence prior to recurrence of an ulcer.[39] For this reason, the importance of treating lower limb swelling should be emphasized including the use of support hose

or elastic wraps. The patient should also be instructed to treat any minor injury to the lower extremities aggressively and seek medical care promptly. The clinician should inspect the lower extremities of sickle cell patients at each visit and review the preventative measures with them routinely.

Practice Point

Always look for edema on the lower extremities of patients with sickle cell disease as it is a critical indicator prior to ulceration. Move this Practice Point to page 14 after the paragraph on edema which starts "Another major issue is to manage…"

Summary

Sickle cell ulcers are a potential reality for any person with the inherited disease—sickle cell anemia. Treatment of these ulcers can be frustrating and requires multiple modalities. Unfortunately, recurrence is frequent. With new modalities to treat the underlying disease and the ulcers on the horizon, it is hoped that these problems eventually will be of historical interest both here and around the world. In the meantime, understanding of the patient's unique situation and use of evidence-based treatments will result in the best outcomes.

Show What You Know

1. Which of the following is NOT correct?

A. Sickle cell disease is an inherited disease of the blood's hemoglobin molecule.

B. Sickle cell disease is seen primarily in black individuals.

C. Higher risk of developing an ulcer is seen in individuals with heterozygous sickle cell disease.

D. The most severe form of sickle cell disease is seen when an individual inherits an abnormal hemoglobin gene from both parents.

2. Sickle cell ulcers are more likely to develop in males with sickle cell disease than females.

A. True
B. False

3. Which one of the following can signal that a patient with sickle cell disease might ulcerate?

A. Edema
B. Hemoglobin A_{1C} of 7
C. Absence of fever
D. Resolution of pain in the leg

4. Which one of the following is not part of the evaluation of the patient with a sickle cell ulcer?

A. History including a family history
B. General physical examination
C. Assessment of wound bed and pulses
D. A bone scan

5. Comprehensive treatment of the patient with sickle cell ulcer could include which of the following?

A. Dietary supplements to improve protein, vitamins, and electrolytes
B. Debridement of the ulcer
C. Wound pain management
D. Compression modalities
E. All of the above

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Surgical Reconstruction of Wounds

18

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Objectives

After completing this chapter, you'll be able to:

- explain the intrinsic and extrinsic causes of surgical wounds that have failed to heal
- describe the reconstructive ladder
- describe how the reconstructive ladder is used to achieve wound closure
- explain the surgical principles that guide care of patients who require skin, tissue, or bone transplantation for wound closure.

Surgical wounds are common in hospitalized patients; however, due to the body's amazing ability to heal acute wounds, hospitalized patients are seldom seen by wound care specialists unless their wounds have failed to heal. This chapter addresses common surgical wounds, complex wounds healed via surgical procedures, and surgical wounds that have failed to heal in a timely manner.

Extrinsic and Intrinsic Causes of Nonhealing Wounds

Causes of nonhealing wounds can be grouped into two large categories: extrinsic and intrinsic. Extrinsic causes are those factors that exist outside of the wound itself, such as pressure, ischemia, smoking, and malnutrition. Intrinsic factors exist within the wound, such as infection, tension, and arterial insufficiency. Determining which of these factors is present in a

wound is an important initial step in wound care. Removing any obstacles to healing is also important; local treatments won't be effective in healing the wound if the underlying cause of the disruption in healing is not addressed.

Wound care focuses on removing the obstacles to healing. Although wound care has seen technological advances, the great 16th-century surgeon Ambrose Paré is still correct in that we as healthcare providers don't heal wounds, we create them and nature heals them.[1] Indeed, our role is to support the body so that the wound can heal. Wound care practitioners must exercise caution when moving too quickly into treatments for wounds until the underlying causes of the delay in wound healing are fully understood.

Wound History

There are four major questions that need to be addressed by the clinician:

1. How and when did the wound occur?
2. When did the wound's healing trajectory change from normal to abnormal and why?
3. What known factors are obstructing the wound from healing?
4. What therapies have been attempted?

When examining a patient with a nonhealing surgical wound, it's imperative that the initial reason for surgery and the operation performed be fully understood. The answers to the following questions must be clear in the examiner's mind: What was the preoperative condition that required surgery? For example, did the patient have gastric bypass or repair of ruptured abdominal viscera? What was the patient's condition before surgery? Was the patient healthy or was the patient chronically ill due to diabetes or cancer? What was the intended operation? Were organs or tissue removed? Was the operation clean or contaminated (e.g., penetrating trauma, perforated diverticulum)? Were foreign materials implanted? Was the operation carried out as planned? For example, has the wound been closed with permanent sutures or mesh? Has healing been occurring along a normal trajectory until now? If so, what has happened to alter the course of healing? What medications is the patient taking that might impact healing? Has the nutritional status of the patient been normal since surgery? Was it normal prior to surgery?

From this initial assessment, a comprehensive understanding of the patient who has the wound, not just the wound itself, will be obtained. And, although treating the wound is of utmost importance, treating the patient, whose body will heal the wound, is equally imperative.

Goals of Care

For acute wounds, wound closure with the return of form and function is the usual goal of care. Left alone to close via contraction and scar formation, wounds seldom have either form or function and will often recur and look unpleasant. The ability to reach ideal and complete healing without scarring, such as seen in fetal wounds, is currently impossible. All full-thickness skin injuries heal with a scar. Therefore, the return of acceptable healing with sustained function and anatomic continuity becomes the ideal end point. The surgeon is constantly mindful of the consequences to leaving the wound open, balanced against the consequences of surgical closure and undergoing anesthesia.

Reconstructive Ladder and Planning Reconstruction

A decision-making process in choosing the appropriate method to achieve wound closure is based on the following information and the "rungs" of the reconstructive ladder (Fig. 18-1). Following are some important questions to consider.

Figure 18-1. Reconstructive ladder. The reconstructive ladder is used to determine the method to replace missing tissue from a wound bed. The ladder shows the simplest method—simply allowing the wound to heal on its own—at the bottom of the ladder.

Is the Wound Missing Tissue?

If the wound hasn't lost any tissue, it may be possible to close it primarily. Wounds are capable of healing by primary intention, which occurs when the wound edges are approximated (pulled together) and retained by sutures, staples, or glue. The dynamics of healing begin, and new tissue is synthesized. A healing ridge (an induration beneath the skin, extending to approximately 1 cm) forms directly under the suture line between 5 and 9 days after surgery. All forms of wound healing lead to scar, and primary wound closure is no different. If the scar is under tension, it is prone to exaggerated scar formation, such as hypertrophic scar.

What Kind of Tissue, If Any, Is Missing?

Wounds that only lack portions of skin may be allowed to granulate closed if they're small, or skin may be grafted to speed the healing process. Wounds that have lost tendon, muscle, or bone may require transplantation of this tissue to provide form and function. Such operations require flaps of skin, muscle, fascia, or bone. Such flaps are named for their composition, such as an osteocutaneous flap, which is a bone and skin flap.

Flaps (as opposed to grafts) bring their blood supply with them and either are locally transported to the wound or are from a distant site (free flap). The free flap employs the surgical technique of freely removing tissue from one area, detaching the nutrient artery and vein from their supply vessels, and moving the tissue to the recipient site where these vessels are reattached to new supply vessels. For example, a free radial forearm flap contains radius bone, overlying muscle, and skin. It is commonly used to reconstruct the face and jaw after wide excision of cancer of the mandible and floor of the mouth.

What Donor Site Morbidity May Occur?

Donor sites have some form of scarring and may exhibit some loss of function, depending on the tissue removed. Split skin graft donor sites should heal with minimal effort; there is no loss of function but a scar remains. When the breast is reconstructed using the latissimus dorsi muscle from the back, the woman may lose some functions of the shoulder. Although some patients perceive the loss of function as tolerable, other patients—for example, a tennis player—may undoubtedly perceive it differently. If a thumb is lost, a patient may opt to transplant the great toe to provide opposition for hand function; however, most people wouldn't want to sacrifice a thumb to replace the great toe. Some cultures (e.g., Japan) place great importance on the toes and prefer to use the second toe rather than the

great toe for thumb reconstruction. In other words, the degree of loss a patient is willing to experience is proportional to his or her need for the sacrificed tissue.

What's the Simplest Method to Achieve Wound Closure?

The easiest method available to close the wound is often used first. Wounds that can heal quickly on their own via granulation and epithelialization are allowed to do so. However, when large wounds or wounds over thin skin (e.g., sacrum) are allowed to granulate closed, the resultant scar is often unstable and prone to injury and ulceration. Skin grafting is the next simplest method and is used to treat wounds that are missing only skin. Skin grafting can be full or partial thickness, depending on the kinds of tissue missing in the recipient site and the condition of the site. Grafting of any tissue type relies on that tissue to revascularize at its recipient site. Wounds that could close on their own, but with accompanying contracture and loss of function, may also be grafted. If muscle is missing, muscle flaps may be used to fill the wound defect or cavity, but the muscle isn't typically made functional (i.e., an insertion, origin, and nerve aren't restored to create a functional muscle). One notable exception is the gracilis free flap to restore the function of the zygomaticus major after facial nerve injury. The blood supply to the muscle is restored so that it remains viable. The ample blood flow into the muscle is commonly used to treat complex wound problems such as osteomyelitis. Restored blood flow may also transport antibiotics and immune cells to the wound.

Muscle has been shown to supply overlying islands of skin through a series of vessels that perforate the muscle body. Surgeons can simultaneously transplant a muscle and the island of skin to both fill a wound cavity and provide skin coverage. Such a flap is called a musculocutaneous flap. These flaps are typically named after the muscle, as in the case of the tensor fascia lata flap, which is used to close a trochanteric pressure ulcer. If a large amount of skin is missing, the muscle may be transplanted and then covered with skin grafting to achieve the same effect. Such an operation is called a muscle flap and split-thickness skin graft, with the specific muscle being grafted added to the name.

Muscle is brought to the recipient site in one of two ways:

- It's rotated along the arc of its original blood supply left intact.
- It's freed from its blood supply and the artery and vein are reattached

via a microscope at the recipient site.

Freeing the muscle, skin, and other parts of a flap allows the flap to be used for reconstruction in areas that the muscle normally couldn't reach, such as the lower third of the leg. It's important to note that, as often as possible, tissue should be replaced with similar tissue. Similar hair bearing, appearance, and thickness improve aesthetic appearance on the reconstructed wound.

Wounds Without Missing Tissue

Some wounds don't require grafts because they aren't missing tissue, or local tissue can be undermined and lifted to close the original wound.

Simple Lacerations

Traumatic wounds are often missing little or no tissue and can be closed primarily; however, the full extent of the injury must be known before any closure is attempted. Laceration of arteries and veins is usually obvious by the amount of bleeding present. Facial lacerations are especially bloody due to the robust blood supply in the face. The patient with a facial laceration is assessed for function of motor and sensory nerves, muscle, and parotid and other salivary glands in the area prior to injection with lidocaine, which would obscure the findings. Repair of vessels, tendons, ligaments, and nerves is commonly completed in the operating room because it is a sterile environment and sometimes has the added benefit of an operating microscope. Wounds are irrigated copiously to remove any debris. Skin closure is accomplished by undermining surrounding tissue to facilitate its movement and suturing layers of tissue with minimal tension. Drains may be placed in wounds with contamination or large amounts of dead space. The appearance of the wound should ideally mimic the ipsilateral appearance. However, traumatic amputation or extensive debridement may leave an appearance that's less than acceptable.

Removing all forms of tension on the wound reduces the scar. Wounds that must be mobilized to gain function, such as incisions over joints, heal with wider scars, and this may be disturbing to the patient's self-image. Stented dressings, immobility (e.g., limited chewing or talking with facial lacerations), and thin applications of topical antibiotics may help to minimize scars. Moist wound healing techniques, used throughout the healing process, will minimize healing time and potentially the amount of scarring. However, it's important for the patient, the patient's family, and

members of the healthcare team to realize that scarring is inevitable and only after the scar has matured, which can take more than 1 year, will scar revision be attempted. Silicone-based dressings may be used over some healed wounds to help minimize scar buildup during the maturation phase. This has only been shown helpful with hypertrophic scarring. The reader is referred to an excellent evidence-based review of emergency wound management.[2]

Extensive Lacerations

Extensive lacerations, while unsightly, are seldom life threatening. Lifesaving care of the heart, lungs, and brain precedes definitive wound care. Initial wound care includes debridement of obvious dirt, glass, grass, or other foreign bodies. Massive lacerations are packed with moist dressings to prevent tissue desiccation, and, when the patient is stable, the wounds are debrided and surgically closed, if possible. Extensive wounds may require multiple debridements until viable tissue is present; these wounds may also require more complex forms of delayed closure, such as flaps.

Wounds that are not closed within the first 6 hours after injury are considered contaminated and cannot be closed primarily after that time. Some clean facial wounds are an exception to this rule, and their closure may be delayed longer in healthy patients who are on antibiotics.

Penetrating Abdominal Wounds

Penetrating abdominal wounds include stab wounds, impalements, gun shots, and so forth. These are serious wounds due to direct tissue injury, risk of contamination and sepsis, and the potential for excessive bleeding. Depending on the degree of contamination from environmental agents (e.g., dirt) or penetration of the bowel or bladder, the wound may not be closed completely. Frequently in traumatic injuries, these wounds are not closed external to the fascia. If the fascia cannot be closed, leaving the abdomen open is also a challenge. These patients require multiple re-explorations while simultaneously reducing or controlling abdominal fluid secretion and preserving the fascia for closure. Currently, no protocols exist for the management of the open abdomen, and care is based on clinical judgment.

Abdominal Compartment Syndrome

Abdominal compartment syndrome (ACS) is similar to compartment

syndrome of the extremity. ACS can occur during elective abdominal operations, following abdominal trauma or pelvic fracture, and with pancreatitis. Normal intra-abdominal pressure in surgical patients is 2 to 10 mm Hg; a value above 12 mm Hg indicates intra-abdominal hypertension, and a value above 20 mm Hg is considered an indicator of abdominal compartment syndrome. Intra-abdominal pressure can be determined using simple water column manometry with a bladder catheter. When pressure is mildly increased to 10 to 15 mm Hg, the cardiac index rises due to compression of the vena cava. With further elevations, there is progressive organ dysfunction due to intra-abdominal pressure on the abdominal organs and vena cava. Direct compression on the hollow intestine and portocaval system causes these structures to collapse. When the bowel is compressed, it becomes ischemic, allowing bacteria to thrive. Vasoactive substances such as histamine and serotonin increase endothelial permeability, further capillary leakage impairs red blood cell transport, and ischemia worsens. As pressure rises, ACS not only impairs visceral organs but also damages the cardiovascular and the pulmonary systems; it may also cause a decrease in cerebral perfusion pressure. Therefore, ACS should be recognized as a possible cause of decompensation in any critically injured patient.[3,4]

It is important to include ACS in a wound healing chapter because the treatment of ACS is not closure of the abdomen. If the abdomen is at all difficult to close, this procedure should be abandoned and alternative techniques applied. A good rule of thumb is as follows: when looking at the abdomen horizontally, if you can see the gut above the level of the wound, consider leaving the abdomen open and using temporary closure.

The easiest way to control the open abdomen is to use a silo-bag closure. This short-term therapy has increasingly been replaced by negative pressure wound therapy (NPWT). The use of NPWT has been shown to allow successful fascial closure in patients who in the past would have required mesh grafting for closure. While NPWT increases the use of primary closure, it also shortens time to recovery.[5,6]

Abdominal Wound Dehiscence and Evisceration

Wound dehiscence is the separation of the edges of a surgical wound. The strength of a wound lies in the musculoaponeurotic layer of the abdomen. In the early postoperative period, wounds stay closed as a result of the strength of the sutures used or through normal healing processes as muscles regain their strength. However, some wounds are susceptible to dehiscence, thus requiring tissue transplantation.

Risk factors for wound dehiscence may be technical or related to patient factors. Technical factors are due to the type of closure used. Indeed, wound dehiscence may occur because sutures break, sutures stretch or cut through the tissue, knots slip, the suture is too thin, or an insufficient number of sutures are used. Closure is best achieved when long-lasting, absorbable, or permanent sutures are used, with secure knots that do not slip or invite bacteria to harbor in them. Sutures should be placed about 1 cm from the abdominal wound and 1 cm apart from each other. This closure places the suture in healthy fascia that will not be cut by suture material. In wounds that are healing securely, a healing ridge of palpable thick tissue about 0.5 cm appears along the incision. This ridge is almost always absent in wounds that rupture.

Risk factors for abdominal wound dehiscence were reported in a large retrospective study.[7] The most significant patient-related factors for dehiscence were age over 50 years, male gender, emergency operation, longer operative times, postoperative coughing, hypertension, steroid use, cancers, ascites, chronic pulmonary disease, jaundice, anemia, and wound infection. A risk score was computed with the highest risk factors being age over 70 years; ascites; vascular surgery; surgery on the esophagus, stomach, or large bowel; coughing; and wound infection.[7]

Obesity, heavy coughing or retching, and ascites, all of which strain the wound during healing, predispose to dehiscence. However, many surgeons believe that if a wound is closed securely, these complications will not occur. Usually, the first sign of an impending problem is the sudden discharge of serosanguineous fluid from the wound, but some patients present with sudden evisceration following an episode of coughing or retching. When the edges of the wound separate and internal organs such as the gut are protruding from the wound, this is known as evisceration.

Evisceration is a frightening experience for the patient. Immediate treatment includes helping the patient to remain calm, medicating for pain, and holding the abdominal contents in the abdomen by keeping the viscera moist with a sterile moist towel. Exposed intestines should not be forced back into the abdomen. The patient should be kept NPO and prepared for immediate surgery. Lower the head of the bed so it is flat or no more than 20 degrees. Monitor the patient's vital signs and assess for signs and symptoms of shock.[8] The wound is explored urgently in surgery, devitalized tissue is excised, and the abdomen is closed with nonabsorbable interrupted suture taking secure bites into healthy tissue. Hernia formation is relatively common, reaching 30% in wounds that eviscerated and reclosed. If the

wound has dehisced and the patient cannot tolerate another anesthetic, the wound can be packed with dressings, an abdominal binder used, or a vacuum-assisted closure applied to obtain secondary healing. These wounds will inevitably develop a hernia.

Wounds Requiring Tissue Transplantation for Closure

Tissue transplantation is a phrase used to describe closure techniques for a group of wounds that have missing tissue. In order to achieve closure, various tissues (skin, muscle, fascia, bone) can be grafted or flapped into the wound. These wounds can be quite complex to manage.

Burns

Surgical management of burn wounds includes escharotomy, excision of eschar, skin grafting, and scar revision. Because many burned patients must undergo multiple operations, it is beneficial to have a dedicated team for these patients to reduce some of the anxiety associated with surgery, anesthesia, and pain management. It is wise to have burned patients go directly from their inpatient room to surgery, not stopping in preoperative areas.

Escharotomy

Escharotomy is a decompressive operation to reduce compartment syndrome in the extremities, respiratory compromise, abdominal compartment syndrome, and even orbital compression. Full-thickness burned skin cannot stretch; therefore, during fluid resuscitation, the fluid that moves into the interstitial spaces places pressure on vessels and nerves. Indications for escharotomy include compartment syndrome of the extremities (pallor, pain, pulselessness, paresthesia, and paralysis), falling oximetry with respiratory difficulty, increased abdominal pressure, and changes in vision (with increased periorbital pressures). Escharotomy is completed by incising the burned tissue along the longitudinal axis of the extremities or across the chest and/or abdomen. The surgical site is left open to allow for both edema and chest wall movement with respiration. Once the capillary bed has stabilized and fluid leaves the interstitial spaces, the escharotomy incisions will approximate. The incisions are not closed, but rather are allowed to heal secondarily; therefore, they should be packed

with saline-moistened gauze.

Excision of Eschar

Years ago, eschar was removed slowly with weeks of daily submersion in tanks and debridement with scissors. This process is still used for some burns, but many burns today are debrided with pressurized water knives, and some burns are completely excised and grafted early to promote rehabilitation. Burns of the hand are typically managed in this fashion in order to quickly mobilize the hand and restore function.

Skin Grafting

The most accepted way to permanently heal a full-thickness burn wound is to graft the wound. Skin grafts are portions of the patient's skin removed from a donor site of unburned skin or healed burned skin that are moved to cover an open burn wound. They are often referred to as split-thickness skin grafts. Skin grafts are harvested in the operating room and placed on the freshly debrided burn wound. The skin graft lives off of the serum in the wound bed; it does not have a blood supply (unlike a flap). As the capillary beds in the wound bed grow into the skin graft, it becomes pink in color. This pink color is called a "take" and often expressed as a percentage (e.g., 50% take of the split-thickness skin graft).

A crucial aspect of skin graft healing is immobilization of the graft onto the wound bed. Most surgeons use fibrin glue, splint the graft in place, or suture it to the edges of the wound. Because edema in the space between the wound bed and the skin graft can separate the graft from capillary ingrowth, NPWT is often used to promote attachment. If NPWT is used, the suction should be continuous and not interrupted.

The dressings are usually removed at 72 hours to inspect the graft. Dressings after that point vary, but many surgeons continue to dress the newly grafted site with an antimicrobial dressing.

Skin grafts fail for several reasons. The primary reason is edema in the wound bed that lifts the grafted skin off the bed. It is imperative that all precautions to keep the grafted area elevated be followed. Too many well-meaning patients have convinced nurses that during a quick trip to the bathroom, rather than the bedside commode, the leg will remain elevated. Unfortunately, those short periods of time with the leg down may be all that is required to allow edema to lift the graft off of the wound bed!

Patient Teaching

Instruct patients to use the commode rather than the bathroom after a skin graft to the leg.

Skin grafts can also fail due to infection in the wound. If infection is suspected, it should be ruled out with quantitative cultures of the wound bed prior to grafting.

Pruritus is a common and distressing problem in healed burn wounds. Skin grafts do not carry sweat or oil glands with them, making the transplanted skin prone to dryness and pruritus from heat retention. In addition, burn scars are not elastic, making them prone to injury. Persistent pruritus is estimated to occur in about 87% of burned patients.[9] Methods to treat pruritus have not been compared systematically, and the use of histamine receptor agonists is commonly based on the belief that pruritus is mediated by mast cells. Newer thoughts on the etiology of pruritus following skin grafting are that it is a variant of pain, and newer treatments for pruritus have examined eutectic mixture of local anesthetic (EMLA) cream, transcutaneous electrical nerve stimulation (TENS), colloidal oatmeal, massage, and sedatives. The problem of pruritus is in great need of ongoing study.

Scar Revision

Scars continue to mature over 6 to 12 months following a burn. Burned areas that were allowed to heal without grafting have the most robust scars. When the scars reach across joints, the joints can be pulled into contracture. Z-plasty is a procedure that can be performed if there is adequate tissue in one dimension and not the other, for example, adequate tissue in length but not width. Z-plasty transfers the excess tissue to the area in need of tissue, thereby releasing the contracture. Many times, however, there is not enough tissue in either direction, and so skin grafting is needed.

Nonhealing Surgical Wounds

Surgical wounds can be slow to heal because of underlying disease states, such as infection, poorly controlled diabetes, impaired arterial blood flow, protein–calorie malnutrition, and compromised immunity. By convention, any surgical wound of 3 to 4 weeks' duration that is not responding to

conventional therapy is considered nonhealing. From the surgical perspective, only debridement can be offered as treatment.

Infection, including the development of biofilms, is a common culprit of nonhealing. Biofilms promote inflammation, and inflammation delays healing. Common bacterial causes include *Staphylococcus*, *Pseudomonas*, and *Enterobacter*. Debridement, performed either traditionally or using low-frequency ultrasound, destroys the biofilm and is an important component of wound care.[10]

Median Sternotomy Wounds

Median sternotomy incisions are extremely complex wounds to close following surgery. Bone stabilization is usually done by rigid fixation and immobilization, but the sternum is often closed with wire and subjected to constant movement with respiration.

Infected medial sternotomy wounds following cardiac surgery are a dreaded complication. High-risk patients include those who are obese (body mass index over 30) and those with diabetes, heart failure, previous myocardial infarction, urgent operative status, hypertension, and/or have large breasts. Perfusion time over 200 minutes, use of an intra-aortic balloon pump, and three or more distal anastomoses are risk factors for very serious infections, but these conditions rarely occur.[11,12] Control of blood glucose and improvement of heart failure prior to surgery are helpful interventions.

Early wound infection, such as the aforementioned suppurative mediastinitis, appears as cellulitis, purulent wound drainage, and obvious tracking between the skin, sternum, and mediastinum. Left untreated, these infections smolder down into the mediastinum and may even extend into aortic suture lines, prosthetic grafts, and intracardiac prostheses. Local wound care is based on the condition of the wound and the condition of the patient. Wound bed preparation may include packing, debridement, or the use of NPWT, which may be used as a first line of treatment to drain the superficial infection down to the sternum because it splints the chest wall. NPWT can be used as a bridge to allow the patient to recover or stabilize. A systematic review with meta-analysis found that the use of NPWT was associated with reduced length of stay and NPWT may be more effective than standard therapy to manage surgical site infections.[13,14]

Caution is required when treating deep wounds that are packed. One continuous piece of rolled gauze is the preferred method for packing to avoid losing single dressings in the chest cavity. If foam is used for packing,

write the number of pieces of foam used on the outermost dressing so that they can be removed with future dressing changes. Further, if wounds require topical application of solutions, the large amount of open tissue will quickly absorb the fluids. Use of such products as povidone–iodine solution in these large wounds has resulted in iodine toxicity due to the large absorptive surface. Wounds that extend to or around the myocardium should be gently packed between heartbeats. Packing the wound tightly can constrict myocardial filling during relaxation; therefore, the wound must be tucked loosely with gauze.

Chest Wall Reconstruction

Reconstruction of the chest wall can range from skin closure to chest wall stabilization. Defects following excision of a tumor, tissue loss from infection of the pleural space, and dehiscence of sternal wounds after coronary bypass grafting are examples of chest wall wounds that usually require reconstruction. Failure to stabilize the chest wall can lead to paradoxical chest motion with breathing, which results in compromised respiratory function. Prior to surgery, the patient's cardiac and pulmonary function, nutritional status, and wound bed should be maximized.

When a defect is limited to skin and subcutaneous tissue, local skin flaps can frequently be used to close the wound. A skin flap can be rotated or advanced to cover the wound. The deltopectoral skin flap can also be used to cover chest wounds, although its blood supply is stretched to do so. The principal blood supply to the flap is from two large perforating arteries arising from the second or third intercostal space lateral to the sternal border. Therefore, any tension of the flap or chest can interfere with blood supply to the flap. The weight and torsion of the breast tissue or an obese chest can place tension on the flap, so a loose-fitting bra or binder should be used to support the tissue.

Loss of the sternum is especially complex because without the sternum, ribs are pulled inward with inspiration (e.g., flail chest). Patients can usually tolerate the loss of four ribs if the wound is closed with a muscle flap, such as the latissimus dorsi flap. Massive chest injuries or defects will require bony reconstruction; if bone is not used, acrylics or synthetic mesh can be used. However, the risks of infection and rejection are ever present when synthetic materials are used.[15]

Practice Point

Immediately report to the surgeon any signs of flap ischemia, such as pale, dusky, mottled, or cool tissue.

When the chest wall defect is large, several muscles may be needed for reconstruction, depending on the location of the wound. The latissimus dorsi, pectoralis major, rectus abdominis, and trapezius are the most common muscles used for chest wall reconstruction.[1] The pectoralis muscle is commonly used because it is near the defect. Its blood supply is through the thoracoacromial artery, located in the axilla. The muscle is also supplied with perforating arteries from the internal mammary artery. If the pectoralis muscle is used, the patient will lose some ability to adduct and rotate the arm and may have some weakness with lifting. Due to the severity of the defect, this loss of donor site mobility is usually justified.[15]

Following surgery, vascular inflow and outflow must be closely monitored. Arterial insufficiency in the flap is a major hazard, and problems can occur from too much tension on the artery, hematoma in the underlying tissue, extravasated blood, increased blood viscosity, spasm of the artery, or injury or disease of the arterial vessels. Venous outflow problems occur from edema in the flap or compression of the veins. A flap with arterial inflow problems will appear pale and feel cool. Flaps with venous congestion will look engorged and purple and feel tense or full.

Pressure Ulcer Repair

Repairing a pressure ulcer for surgical closure is a process that involves multiple steps that begin well before surgery. After the pressure ulcer bed is clean, it can be closed surgically. However, before any decisions are made for surgery, such plans must be considered in light of the patient's situation, condition, and goals. While various surgical options may be technically possible, performing these operations for the right reasons is an essential first step. Surgery shouldn't be entered into lightly. For some patients, not closing the wound surgically may be the best decision. With proper nutrition, pressure redistribution, and local wound care, deep pressure ulcers may remain stable for the duration of the patient's life. Adjunctive therapies, such as NPWT, electrical stimulation, and the use of bioengineered tissue products, may be considered to help with closure of

these chronic wounds.[16]

Preparing the Patient for Surgery

Prior to any surgery, the patient's nutritional status and comorbidities must be controlled. Although operative blood loss is usually modest with this type of surgery, general anesthesia is commonly used and the patient must be able to tolerate the stress. General anesthesia is also used in paralyzed patients. The need for general anesthesia for repairs in these patients is often an area of question, with the patient and/or family asking "why do I need anesthesia if I can't feel below my waist?" This question should be answered in the following way: "Even though you may not feel pain, reflex arcs are frequently intact and the stress of the surgery and blood loss make blood pressure erratic." Many pressure ulcers are repaired with the patient in a prone position, and general anesthesia is necessary to maintain a patent airway and oxygenation.

If malnutrition is the primary cause of nonhealing, surgery should be delayed until the patient has achieved positive nitrogen balance. Calorie counts provide clear data on actual intake of protein and calories, and adjustments can be made to reach ideal intake levels of 30 to 35 cal/kg of calories and 1.25 to 1.5 g/kg of protein.[17] Monitoring albumin is no longer considered a reliable marker due to albumin being increasingly recognized as an acute-phase reactant and thereby lowered by any inflammatory processes. With a half-life of 3 days, serum prealbumin is a better marker for assessing visceral protein status but also affected by any inflammation. In patients with impaired renal function, prealbumin is artificially high because it is not removed with dialysis. Therefore, patients on dialysis should be classified as malnourished if the prealbumin level is less than 30 mg/dL, whereas the usual normal range is 16 to 35 mg/dL. More accurate nutritional information on protein status can benefit the healing potential for the patient. Indirect calorimetry is helpful to obtain more exact information on nutritional expenditure. It's important to recognize that patients who are malnourished are often deficient in vitamins (especially vitamin C) and minerals (especially zinc and iron); these supplements should be given throughout the course of care. The guidelines on pressure ulcer treatment[16] do not advise the routine use of vitamins and minerals for healing; they are recommended here only if the patient is deficient in them.

Practice Point

Because prealbumin is not removed during dialysis, use the lab value of less than 30 mg/dL to determine malnutrition.

Again, the need for aggressive nutritional support is a common area for clinical questions. Families and patients may view tube feeding as "artificial life support" and resist its use. The healthcare team needs to explain that unless the patient is eating adequately, there will be no reserve to heal. Acute wounds need more calories and protein to heal, and tube feeding (or hyperalimentation) should be considered as a short-term method to increase the likelihood of healing (see Chapter 10, Nutrition and Wound Care, for additional information).

If the ulcer is due primarily to pressure (such as ischial ulcers in a paraplegic patient), pressure must be redistributed both before and after surgery. Long-term plans for continued pressure redistribution must be included in the operative plan, such as fitting the patient for a wheelchair and appropriate off-loading device and teaching the patient pressure-relief techniques to avoid high rates of recidivism.[18]

If the ulcer is due to a combination of erosion, shear, and pressure (such as ulcers on the sacrum in a patient with dyspnea and incontinence), all contributing factors need to be addressed. Similarly, long-term pressure reduction and proper skin care management must be included in the care plan. There are a finite number of available flaps; therefore, all efforts must be directed to prevent recurrent ulcers, especially through education of the patient. The social network of the patient with a spinal cord injury has been shown to be very crucial in maintaining a healed wound.[18]

Wound infection may not be evident from the surface appearance of the wound. The presence of osteomyelitis should be considered if the bone feels rough or soft and ruled out in all stage 4 ulcers. MRI is usually helpful to identify the infection. Biopsy after adequate debridement remains the gold standard in diagnosing osteomyelitis if still suspected and the above studies are negative.

Other infections must also be controlled prior to surgery. Urinary tract infections are common in people with diabetes, elderly women, patients with catheters, and those with sacral wounds. Urosepsis is a serious complication and should be considered as the cause of malnutrition and

changes in mental status or vital signs malnutrition. Chronic urinary antibiotics may be needed for persistent urinary tract infections. Wound infection can also lead to sepsis[19] (see Chapter 7, Wound Bioburden and Infection).

Spasms are often a contributing factor to both shear and friction injuries and may also complicate a postoperative course by putting undo tension on wound closure sites. Spasms are common after spinal cord injury due to loss of supraspinal inhibitory pathways. The higher the level of spinal cord injury, the more likely spasms will occur. People with cervical injury have almost a 100% chance of spasm compared with those with injury in the lower thoracic or lumbar spine, who have a 50% chance of spasm. Spasm must be controlled prior to surgery with medications such as baclofen (Lioresal) or dantrolene (Dantrium). Botulinum toxin (Botox) can also be injected into the muscle to attempt to reduce spasm; however, the efficacy of this treatment is unrecorded. Spastic limbs may lead to wound dehiscence and postoperative wound complications and therefore must also be controlled.

Contracture develops in patients with longstanding denervation caused by tightening of the muscle and joint capsules. Because hip flexors are so strong, hip contraction is common and can make positioning difficult, with bony prominences resting on each other and leading to ulcers. Contractures can be minimized with proper, persistent positioning, splinting, and a program of aggressive (often passive) range-of-motion exercises. Patients with significant contracture can't be placed in a supine position; if surgery is performed on one hip, only one side will be available as a turning surface, which will require a sophisticated pressure redistribution system to prevent complications or undue pressure at the other hip. A defined turning schedule is essential in these cases. If contractures can be released via tenotomy, the wound may heal. However, leaving a limb flaccid after tenotomy may leave the patient bedridden if he or she relies on the spastic limb for postural support while moving.

Preparing the Wound for Surgery

Debridement of nonviable tissue is an important first step in the treatment of full-thickness necrotic pressure ulcers or infected, dehisced surgical wounds. Debridement of adherent eschar in pressure ulcers is advised to reduce the wound bioburden and risk of sepsis. The true stage of a pressure ulcer can't be determined until adequate debridement has been performed because the true depth of the wound is obscured. Debridement can result in

extensive wounds. For example, pelvic pressure ulcers can extend to the scapula and to the vagina or trochanters. Removal of necrotic tissue may enhance the wound healing cascade and diminishes the risk of infection (see Chapter 8, Wound Debridement). Dry and stable gangrene should usually not be removed from foot ulcers in patients with impaired arterial supply. Changing a closed and stable wound to an open and ischemic wound creates a greater problem.

Wound debridement is most thoroughly completed in the operating room. Bedside debridement may be used to unroof hard eschar but can seldom be completed to the level of a clean wound bed. Enzymatic debridement will often provide reasonable wound debridement but may take many weeks. However, in the patient who is a poor surgical risk, enzymatic agents are a good alternative.

Determining which wounds would benefit from debridement is the first step. Necrotic tissue is generally understood to be a healing deterrent because it allows infection. Necrotic tissue has no blood supply, so antibiotics and antibodies are not present in it. The wound and surrounding tissue should be examined fully to assess for fluid collection, abscess formation, and extensions into surrounding tissue.

Whether or not heels should be debrided remains controversial. Most clinicians feel that stable, dry, adherent eschar on ischemic heels should not be debrided and recent data show healing of heel ulcers when eschar is left intact.[20] The foot, especially the posterior heel, has very limited blood flow and only a small amount of subcutaneous fat. Once the underlying fatty tissue is exposed to the environment, it may become infected quickly due to desiccation and its limited blood flow. If bone is exposed during debridement, osteomyelitis may be an inevitable occurrence. Stable, dry eschar that has no openings should be left intact, assessed often, and off-loaded completely by placing pillows under the calves or floating the heel in orthotic splints/boots. The phrase “float the heels” is an appropriate order to convey the idea that no pressure is to be applied to the heels. If the eschar softens or breaks or the tissue around the ulcer becomes fluctuant or inflamed, the tissue should be excised to prevent deeper bacterial invasion and sepsis. Chemical debridement may be needed to continue the process of wound bed preparation. Moist wound healing techniques should be instituted at this time. Off-loading should continue throughout the course of treatment.

Flaps for Pressure Ulcer Repair

Large areas of skin or skin and muscle may be missing from a pressure ulcer. Surgical repair options depend on what kind of tissue needs replacement. If skin is missing on a sacral wound, for example, rotational flaps of skin or skin and fascia can be rotated to close the wound. If muscle is missing, such as in a deep stage 4 ulcer, the gluteus may be used to provide padding and protection of the bony structures. The muscle moved into the wound doesn't function as muscle; it atrophies over time due to denervation. Muscle tissue provides padding and robust blood supply to combat osteomyelitis. Muscle also carries more skin with its named blood vessels, so more skin can be moved without ischemia developing (Fig. 18-2, Table 18-1).

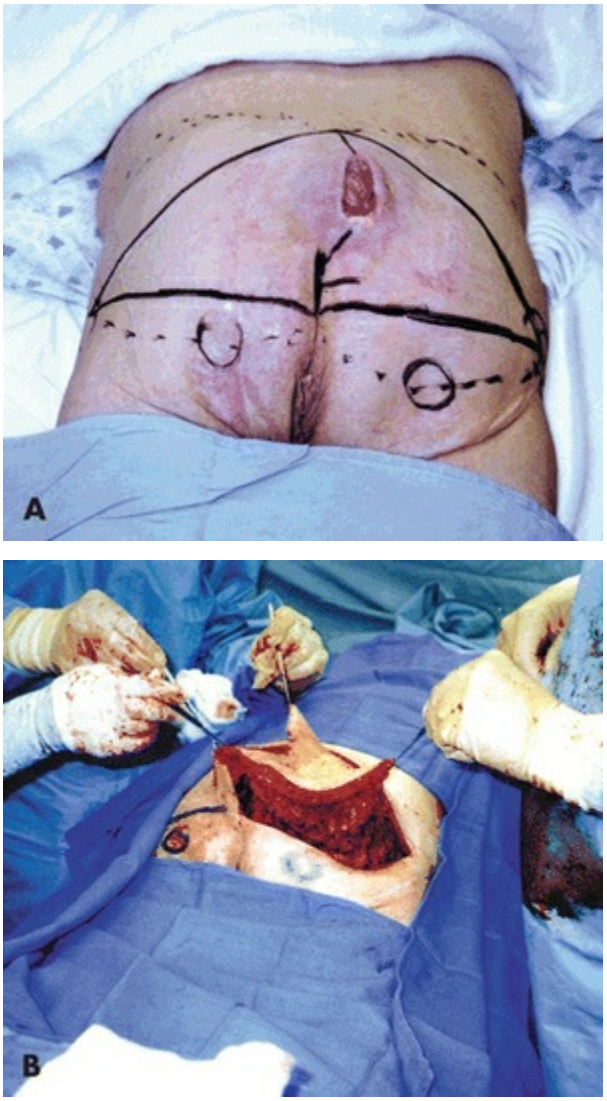

Figure 18-2. Surgical closure of a pressure ulcer. **(A)** This photograph shows markings made for gluteal fasciocutaneous flaps for surgical closure of a stage II pressure ulcer. **(B)** Shown here is surgical closure of the same ulcer.

Table 18-1 Flaps for Pressure Ulcer Repair

Pressure Ulcer Site	Muscle Flap Options	Skin or Fasciocutaneous Flap Options
Sacral ulcer	• Gluteus maximus (superiorly or inferiorly based) or V-Y advancement	Tensor fascia lata (may retain sensation)
Ischial ulcer	• Gluteus maximus (superiorly based) • Biceps femoris • Semimembranous • Semitendinosus • Gracilis	• Transverse or vertical lumbosacral • Posterior thigh advancement flap with skin graft of donor site • Posterior V-Y advancement flap • Medial thigh rotation flap
Trochanteric ulcer	• Tensor fascia lata (may retain sensation)	

This chart presents common closure options for pressure ulcers at common sites.

Postoperative Care

The transplanted muscle flap requires adequate perfusion of arterial blood and drainage of venous blood to survive. Because a limited number of flaps are available to reconstruct any wound, the consequence of flap failure is great. Early flap failure is most commonly due to arterial spasm or clots in the venous drainage. Flaps that have impaired arterial flow appear pale, have poor to absent capillary refill, and show sluggish bleeding when lanced. These wounds require quick restoration of their arterial supply by opening the wound and examining the arterial inflow. Occasionally, arterial spasm is the culprit. Spasm can be treated by positioning the flap dependently and warming the area.

Practice Point

Early signs of arterial flap failure are:

- pale color
- poor or absent capillary refill
- sluggish bleeding when lanced
- loss of audible pulses by Doppler

Flaps that have impaired venous drainage appear dark blue and swollen. The problem is seldom a faulty anastomosis site; usually, it results from the sluggish exit of venous blood. Venous congestion can be treated by elevation and application of leeches to drain excess blood from the flap.

NPWT may also be recommended to decrease fluid collection. Drains are commonly used to empty fluid accumulation in dead space and can be left in place for a week or longer until drainage has subsided.

Tension on the incision line can lead to dehiscence. Bolster dressings are typically used to close the wound with little tension. Suture lines are slow to heal, especially in the denervated patient, and sutures are left in place for at least 3 weeks. Due to poor approximation and tensile strength, great caution must be used when moving the patient to avoid pulling on the suture line. It's possible to tear open a late-stage surgical repair, which commonly leads to complete flap failure. Small areas of dehiscence are often left to heal via granulation tissue.

Pressure relief is crucial following flap repair. Surgeons usually prescribe bed rest on an air-fluidized beds or low-air-loss beds for 2 to 6 weeks. Be sure the patient is not removed from the bed without the surgeon's permission (e.g., sent for an x-ray procedure). Large skin flaps are especially prone to failure because of tension on the distal edges of the flap. If fecal incontinence is likely, the patient may be placed on a low-residue diet and constipating medications. Diverting colostomy may be required prior to flap closure in extreme cases to prevent contamination of the surgical site with stool. Usually, bed pans are contraindicated for the first few weeks.

The patient must be compliant with off-loading strategies after a surgical flap for wound closure to prevent breakdown of the surgical repair. This involves not only immediate postoperative pressure redistribution but continued interventions of pressure relief and reduction after complete healing has occurred, especially in patients confined to wheelchairs. Proper chair cushions and weight shifts are essential.[18]

When describing healing of flaps, the wound is a surgical wound, not a pressure ulcer. Its healing is primary and not secondary with granulation tissue.

Leg Reconstruction

Tissue defects of the leg can be reconstructed with muscle flap covered with skin grafts, myocutaneous flaps, or free flaps, depending on the location of the wound and available donor tissue. Attempts are made to salvage the leg unless there is irreversible nerve or vessel damage. Major soft tissue injuries with or without bone involvement provide an environment favorable for infection. Wound care is commonly completed in the operating room, where debridement can be performed in a sterile, controlled

atmosphere. Definitive wound coverage often includes rectus abdominis, gluteus, rectus femoris, gastrocnemius, and soleus muscle.

Following surgery, it's imperative to monitor the flap for signs of vascular compromise. These situations are emergent; without immediate intervention, the limb may be lost due to ischemia. Unusual findings must be reported promptly and accurately to the attending surgeon.

Practice Point

Monitor for the following signs of vascular compromise:

- Pallor and coolness
- Lack of pulses
- Pain with movement
- Slow or absent capillary refill
- Inability to move extremity

Necrotizing Fasciitis

Necrotizing fasciitis, also known as synergistic gangrene and "flesh-eating bacteria" infection, is a rapidly progressive soft tissue infection. Although beta hemolytic *Streptococcus pyogenes* is the most common causative organism, no single organism is responsible for the infection. Frequently, necrotizing fasciitis is caused by two organisms acting in concert, called synergistic gangrene. Gram-positive bacteria (including methicillin-resistant *Staphylococcus aureus*), gram-negative bacteria, anaerobic bacteria (including gas gangrene caused by *Clostridium perfringens*), marine *Vibrio*, and fungi have been identified. Necrotizing fasciitis caused by beta hemolytic streptococcal organisms is highly sensitive to antibiotics. However, antibiotics don't penetrate necrotic tissue, so delays in treatment can lead to a 73% mortality.[21]

Necrotizing fasciitis appears to develop following a breach in the integrity of a mucous membrane barrier, especially in the abdomen, perineum, and extremities Sacral pressure ulcers can develop necrotizing soft tissue infections also. A malignancy may also give rise to a portal of entry. In males, leakage into the perineal region can result in a syndrome called Fournier's gangrene, which is characterized by massive swelling and

tissue loss of the scrotum and penis with extension into the perineum, the abdominal wall, and the legs.

Pain out of proportion to the size and extent of the skin wound is the hallmark sign. Early presentation also includes a reddened, painful, swollen area of cellulitis and fever. More generalized swelling develops and is followed by brawny edema. With progression, dark red induration of the epidermis appears, along with bullae (filled with blue or purple fluid). Later, the skin becomes friable and takes on a bluish, maroon, or black color. By this stage, thrombosis of blood vessels in the dermal papillae is extensive. Extension of infection to the level of the deep fascia causes this tissue to take on the brownish-gray appearance of frank gangrene. Skin is normally a very effective barrier preventing bacteria from invading the body; likewise, immune cells in the skin are also able to trap organisms, preventing rapid spread along fascial planes and through venous channels and lymphatics. Patients in the later stages are toxic and frequently manifest septic shock and multiorgan failure. Progression may be rapid. Skin inflammation is rapidly followed by necrosis of superficial fascia, subcutaneous fat and, in some cases, muscle. Necrotizing fasciitis is commonly seen in conjunction with severe systemic toxicities.

Practice Point

The hallmark of necrotizing fasciitis is pain out of proportion to physical findings.

Diagnosis and Treatment

Aspiration cultures from the wound edge or punch biopsy with frozen section may be helpful if the results are positive, but false-negative results occur in approximately 80% of cases. There is some evidence that aspiration alone may be superior to injection and aspiration using normal saline solution. Extensive gas can be seen along the fascial planes on radiograph. However, deep biopsy and frozen section histopathology often confirm the diagnosis. Frozen sections are especially useful in distinguishing necrotizing fasciitis from other skin infections such as toxic epidermal necrolysis.

Practice Point

In cases of suspected necrotizing fasciitis, myositis, or gangrene, early and aggressive surgical exploration is necessary to:

- visualize deep structures
- remove necrotic tissue
- open compartments to decrease pressure
- obtain tissue for Gram stain for aerobic and anaerobic organisms.

Repeated debridements are usually necessary until all devitalized tissue is removed, and early debridement was associated with improved outcomes.[22] Intravenous broad-spectrum antibiotics are started, pending more specific culture results. The current empirical antimicrobial regimens advocate use of piperacillin–tazobactam with clindamycin and ciprofloxacin for infections from a combination of aerobes and anaerobes. For infections from group A streptococcus or *Staphylococcus aureus*, penicillin with clindamycin is recommended. For clostridial infections, combination of penicillin with clindamycin is effective. The final course of antimicrobial therapy should be based on the culture sensitivity reports. In cases of fungal infections, intravenous amphotericin B is given. Appropriate empirical antibiotic treatment for necrotizing fasciitis from group A streptococcal organisms is commonly clindamycin plus penicillin G and cephalosporin (first or second generation). Mixed aerobic–anaerobic infections can be treated with ampicillin and sulbactam, cefoxitin, or combinations of either clindamycin, metronidazole, and ampicillin or ampicillin, sulbactam, and carbepenem.[23] Hyperbaric oxygen treatment may also be useful as many of these infections have an anaerobic or microaerophilic component. Antibiotic treatment should be continued until all signs of systemic toxicity have resolved, all devitalized tissue has been removed, and granulation tissue has developed. Multiorgan system failure is not uncommon.

Following debridement, wounds are left open and packed. The packing can be extensive and may be completed in the operating room with daily debridement. Definitive treatment may include amputation or extensive skin grafting after the infection is under control and the wounds show evidence

of granulation. It's essential that the dressings stay moist to prevent desiccation of tissue. It may be helpful to use products with a higher absorptive capacity than gauze to manage fluid from draining wounds.

In one retrospective study, patients who survived an episode of necrotizing fasciitis had an increased risk of premature death due to infection, such as pneumonia, cholecystitis, urosepsis, or sepsis.[24] Women died much earlier than men in the study. In our experience, patients with necrotizing fasciitis have an increased frequency of colon cancer, and colonoscopy prior to dismissal has become part of the routine care provided.

Summary

Understanding the etiology of complex wounds is a critical first step in developing an effective treatment plan. Recognition of the unique characteristics of each of these wound categories will enable the clinician to correctly identify the wound. The goal of care for acute wounds is wound closure with the return of form and function. Use of the reconstructive ladder can assist clinicians in the decision-making process. Surgical management may be part of the care plan for complex acute and chronic wounds. Monitoring for signs of failure is imperative for patients with skin flaps and grafts.

PATIENT SCENARIO

Clinical Data

Andre is a 22-year-old man who became a T12 paraplegic following a motor vehicle crash 2 years ago. Andre had been the driver of the car, and his best friend was killed in the crash. Andre has been struggling with his residual paralysis and accepting responsibility for his injuries and his friend's death. He was treated initially at a trauma center for repair of a fractured spine, followed by rehabilitation at a spinal cord center. He often expressed the hope to "walk again" doing "whatever it takes." He uses a wheelchair with a gel cushion to allow for sliding transfers.

This admission to acute care began with treatment in the emergency department for fever and chills. He was diagnosed with sepsis, the

source of which was determined to be his ischial pressure ulcers. At the time of admission, the wounds were found to be dressed with newspaper. Andre said he did not have the money to buy dressings and uses paper towels or newspaper to absorb the drainage. He has no other chronic illnesses. He is protein malnourished and smokes 1 to 1½ packs of cigarettes daily.

Examination of Andre's wounds reveals extensive pressure ulcers throughout the perineum (Fig. 18-3). Both unstageable ischial pressure ulcers and healing pressure ulcers are present. Surgical consultation is recommended.

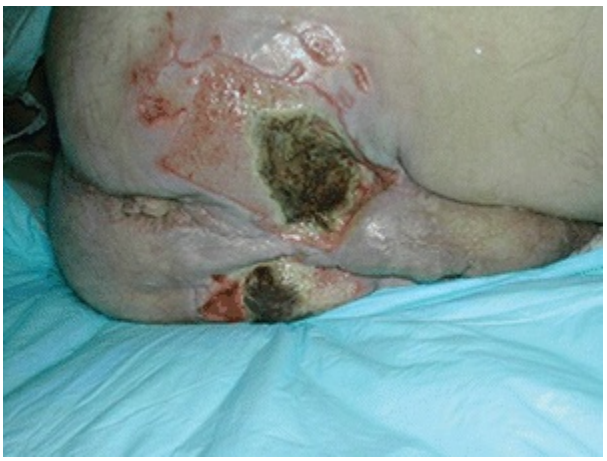

Figure 18-3. Extensive pressure ulcers throughout the perineum in a 22-year-old paraplegic man.

Case Discussion

Let's consider the following four questions as the basis for our clinical decision making regarding Andre's plan of care.

1. **What surgical treatment is likely to be recommended now?** Because the source of his sepsis is his wounds, these ulcers need to be debrided. Ideally, the debridement should be done in the operating room; however, if he is hypotensive from the sepsis, the wounds could be unroofed at the bedside. Eventually, the wounds will need to be evaluated to determine whether osteomyelitis is present. Infectious disease consultation should be obtained. Once certain anaerobic infection is controlled, NPWT could be used to promote healing.
2. **What type of pressure redistribution system is needed in the bed and in the chair?** An integrated bed system with low air loss

and patient movement or an air-fluidized system is needed. While these ulcers could be seen as affecting only one turning surface, the use of a standard foam mattress will likely not be adequate because his risk for new ulcers is also high enough to justify the expenditure for an upgraded surface.

3. **Is flap closure advisable? Why or why not?** Technically, there are flaps that could be raised to close these ulcers. However, it would likely be a multistage procedure and the flaps can only be used once. Unless Andre becomes more compliant with an off-loading program, the flaps are likely to fail. He has obviously had several ulcers and even a surgical debridement in the past 2 years. He may still be angry over the physical impact on his body and would benefit from mental health evaluation. A quick surgical fix to close these wounds would likely not benefit him in the long run.
4. **What social support is imperative in order for these wounds to stabilize? Heal?** Andre's social support network needs to be explored. If he lives alone, he might benefit from a group home with other persons confined to wheelchairs. The positive role modeling from others with spinal cord injury may be able to engage him to do off-loading and examine his skin for signs of breakdown. Social services should be working with Andre to ensure that he has a medical benefit program that can provide appropriate supplies. He also needs to be evaluated by physical and occupational therapists to assess his wheelchair, wheelchair cushion, methods of transfer, and off-loading and other potential rehabilitation strategies. Impact of smoking on wound healing needs to be explored with Andre. Even if he is unwilling to stop smoking, he might be agreeable to reducing the amount of smoking. This could be a good place to start.

Show What You Know

1. Which of the following situations depicts the use of the simplest method for closure of a wound?

A. A skin graft of a venous stasis ulcer
B. Secondary healing of a calcium extravasation
C. Primary closure for a sternal incisional dehiscence
D. A free fasciocutaneous flap for a stage III pressure ulcer

2. Which of the following wounds might require a musculocutaneous flap for closure?

A. Large burn on the face
B. Calciphylaxis of the lower legs
C. Radiation necrosis of the chest wall
D. A stage II pressure ulcer on the trochanter

3. Which of the following signs might indicate arterial impairment in a flap?

A. Pain and coolness
B. Pallor and warmth
C. Slow capillary refill and pain
D. Slow capillary refill and pallor

4. You discharged a patient with ischial ulcers from the hospital after 6 weeks of local wound care. Upon return to the clinic, the wounds have recurred and are necrotic. Which of these causes of recurrence should be investigated first?

A. Presence of urinary incontinence
B. Inadequate pressure redistribution in seating
C. Deterioration of nutritional status
D. Development of ischial osteomyelitis

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Tube, Drain, and Fistula Management

19

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Objectives

After completing this chapter, you'll be able to:

- describe different types of tubes and drains
- state the etiology of fistulae
- discuss management options for drains, tubes, and fistulae.

Tube, drain, and fistula care may seem out of place in a wound care text. However, the realities of clinical practice sometimes dictate that a wound care clinician be consulted in the management of these special patients. In fact, it is not uncommon for people with wounds to have an assortment of concomitant conditions that include a fistula or the use of tubes and drains. Wound care is a science; drain, tube, and fistula management is an art based in science. As with all aspects of patient care, a holistic approach is essential.

Tubes and Drains

The purposes of tubes and drains include the promotion of drainage from a wound or body cavity, decompression, lavage, and medication administration. Placement of tubes and drains will depend on the type, location, and purpose of the tube or drain being used. Determining a management plan starts with an assessment of the type of tube being used and the manufacturer's guidelines for care; the location of the tube, including any associated skin folds that can impede the stability of the device; and a medical record review to determine the internal location and purpose of the tube and how well the device is functioning.[1] Depending on

the type, purpose, and location of the tube or drain, the insertion process will be facilitated by computed tomography, fluoroscopic illumination, or endoscopic guidance (Table 19-1). In the case of open or laparoscopic surgery, the tube or drain generally is placed during the procedure.[1]

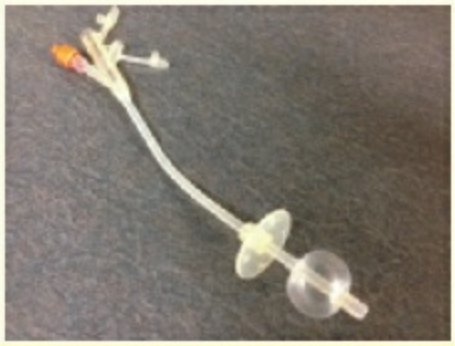
Table 19-1 Categories of Tubes and Drains

- **Simple tube/drain:** A catheter or a soft, flat, rubber drain. A rectal tube is an example of a simple tube that is inserted into the rectal cavity to relieve flatus, stool, or postoperative drainage. Simple tubes can be used for enema or medication administration.
- **Closed drainage catheter:** A catheter connected to a drainage container. In most cases, the collection container is considered sterile and sealed during the manufacturing process. Chest tubes, Jackson-Pratt drains, Hemovac drains, and indwelling urinary catheters (IUCs) are examples of closed drainage systems that attach to a bedside collection system.
- **Chest tube:** A sterile, closed drainage and suction system used in an emergency or surgical setting to relieve air or fluid from the pleural space. The closed drainage system is sterile and includes a water seal and negative pressure for suction.
- **Simple sump:** A catheter inserted into the abdominal cavity following surgery. It can be connected to a suction device at the distal end of an incision or can be used to instill an irrigant such as saline.
- **Nasogastric tube:** A tube that accomplishes nasogastric decompression when inserted through the mouth or nares into the stomach to remove gastric contents or administer food or medications
- **Feeding tube:** A tube that is inserted into the stomach or small intestine for enteral feeding
- **Indwelling urinary catheter:** A type of catheter that is inserted, using a sterile process, into the bladder to drain urine and decompress the bladder. A Lubricath Coude Catheter has a curved tip to ease insertion past the prostate.[1,2]

Premarking the drain site prior to insertion is sometimes helpful. With an enteral feeding tube, for example, premarking the drain site minimizes the possibility of the tube residing within a skin fold or crease. Tubes situated on a level plane of skin are easier to stabilize and manage.

A gap often exists in patient and family education regarding drains and tubes. Most people are aware of what to expect as far as an incision is concerned, but the presence of a tube or drain is unanticipated and unwelcomed. Preparing the patient for the potential outcome of tube or drain placement reduces anxiety and gives the patient the opportunity to plan ahead[1] (Table 19-2).

Table 19-2 Common Types of Tubes

Gastrostomy feeding tube	
Jackson-Pratt–style drain tube end	
Mushroom tip catheter style	
Penrose drain	
Sump style tube with sheath	

Patient Teaching

Tell the patient and family the location and the number of tubes and drains that might be needed after surgery or to treat their medical condition.

Types of Tubes and Drains

Feeding Tubes

Patients who require enteral feeding are debilitated by disease, traumatic injury, extensive surgery, or malnourishment. Enteral feeding following a surgical procedure in which the patient is unable to take food by mouth is desirable to treat and prevent nutritional deficits and prevent atrophy of mucosal villi in the small intestine.[2] Types of enteral feeding systems include nasogastric, gastrostomy, percutaneous endoscopic gastrostomy (PEG), and jejunostomy. Tubes can be placed using several methods, including surgical, endoscopic, radiological, or manual by way of the nares (Table 19-3).

Table 19-3 Nursing Care of Feeding Tubes

Successful nursing care of feeding tubes depends on: • ensuring that the device is stable • providing appropriate nutritional management • meeting the patient's hydration needs • using the proper technique for instillation of medications.

Nasogastric Tubes

Nasogastric tubes are inserted manually and are the least stable of the enteral feeding options. The procedure involves selecting the appropriate feeding tube, measuring the patient's anatomy to determine the length of insertion, lubricating the tube, placing the patient in a high Fowler's position, having the patient swallow water (if responsive) during the insertion process, and educating the patient about the procedure. Be sure to maintain tube stability at the nares used for insertion, and check throughout

the insertion procedure and before instilling the liquid feeding that the tube does indeed end distally in the stomach.

Gastrostomy Tubes

Gastrostomy tubes (G tubes) are surgically inserted directly into the stomach and exit at the anterior abdominal wall. This type of procedure allows for proximal decompression with simultaneous distal enteral feeding. A number of commercially manufactured G tubes are available for use. The typical design includes a disk to stabilize the tube exteriorly against the abdominal wall and a balloon tip at the distal end to ensure internal stability.[2]

Percutaneous Endoscopic Gastrostomy Tubes

PEG tubes are inserted directly into the abdominal wall using an endoscope. The tube exits proximally at the abdominal wall. The main purpose of PEG tubes is to treat and prevent nutritional deficits associated with chronic illness complications, extensive surgery, and abdominal trauma. Tube stabilization is achieved with internal and external bumpers placed along the abdominal wall.[2]

Jejunostomy Tubes

Jejunostomy tubes are surgically or endoscopically inserted directly into the jejunum for the purpose of long-term enteral feeding in patients who are at risk for aspiration or for patients with esophageal, gastric, or duodenal disease processes. Tube stability is achieved with internal and external bumpers.[2]

Biliary Tubes

Biliary tubes are used to relieve obstruction and facilitate drainage from the bile ducts of the liver. The catheter is inserted into the liver and bile duct either as part of a surgical procedure or via fluoroscopy using contrast and a guidewire. Catheter holes are positioned above and below the obstruction. Biliary tract drains include cholecystectomy tubes, percutaneous drains of the biliary tract, T tubes, and endoscopically placed nasobiliary tubes. Nursing management of biliary tubes includes maintaining tube stability, containing the effluent (output), caring for the skin around the tube, and monitoring output.[2]

Esophagostomy Tubes

During esophagostomy, which involves surgical resection of the diseased part of the esophagus, the proximal end of the esophagus is brought to skin level and forms a stoma. The stoma is generally flush with the skin and located lateral to the trachea, resulting in an uneven surface that is difficult to pouch. Creative management solutions involve skin preparation and the use of moldable skin barriers attached to a pouching system to contain the drainage. A decompression tube can be added to aid patient comfort in cases of intractable nausea and vomiting.[2]

Indwelling Urinary Catheters

Indwelling urinary catheters (IUCs) have gained much attention in the literature and with regulatory agencies such as the Centers for Medicare and Medicaid Services (CMS) as a cause of increased cost to care and increase risk of infection.[3,4] Acute care hospitals, long-term care, and rehabilitation centers have also recognized the need to develop policies aimed at using IUCs more judiciously and for shorter periods of time to reduce the potential damage of IUCs on the lower urinary tract, reduce catheter-related sepsis, and promote urinary continence strategies.[4]

IUCs are used to monitor urinary output, manage urinary retention, decompress the bladder following surgery, and manage wounds complicated by urinary incontinence. They are used occasionally for long-term management of urinary incontinence.[1,5–7] A large variety of IUCs are available for use, including catheters made from latex or silicone and catheters coated with hydrogel, Teflon, or a silver alloy.[3] Common IUC properties include a double lumen, a balloon tip at the proximal end to maintain placement in the bladder, a distal end that will accommodate connection to a closed drainage system, and sufficient length to extend from the bladder and through the urethra to connect to a closed drainage system. The double lumen allows for balloon inflation and deflation as well as urinary drainage.

IUCs are one of the most frequent causes of nosocomial infection in acute care settings. Selecting the appropriate IUC begins with assessment of the patient. Considerations include allergies (such as latex) that will preclude the use of some types of catheters, the length of time the IUC is expected to be in place, and the mobility of the patient. In general, the smallest French size needed should be selected because the elastic urinary mucosa will conform to the catheter. Using larger-French-size catheters can

cause erosion of the uroepithelium and result in leakage of urine and permanent damage to the urethra. The newer silver alloy–coated catheters are intended to reduce bacterial growth. A review of the literature demonstrates limited effectiveness in the reduction of catheter-associated urinary tract infection (CAUTI) with short-term IUC use.[1,5] Nursing management of IUCs incorporates measures to prevent infection and limit the length of time the catheter is used.[1,5–7] The longer the catheter is in place, the higher the risk will be for bacteria to infiltrate the bladder. Most IUCs should be considered for short-term use (20 to 30 days). Local care of the urinary meatus with soap and water on a daily basis along with use of a closed drainage system has been shown to reduce bacterial migration as well as provide an opportunity for the nurse to monitor the condition of the perineal area.[1,6,7] Balloon size and inflation have also been discussed in the literature as important aspects of care. The purpose of the balloon is to stabilize the catheter in the bladder. For most adults, this can be achieved with a 5-mL balloon. Larger balloons (up to 30 mL) are manufactured with the intent of providing hemostasis by adding internal pressure following prostatectomy.[1,5–7] Additional nursing management strategies center on monitoring the patient for leakage (usually a result of bladder spasms), catheter-associated pain or discharge, and the color, odor, and amount of urine. Assess the patient for symptoms of CAUTI. Anchoring the catheter to the internal area of the patient's leg will provide additional stabilization and comfort and reduce the risk of accidental removal.[1,5,7]

Practice Point

Presenting symptoms of CAUTI include fever, chills, diaphoresis, hematuria, and pain (flank and suprapubic).

Nephrostomy Tubes

Nephrostomy tubes are used to relieve obstructive uropathy of the lower urinary tract. They are placed into the renal pelvis of the kidney at the flank using fluoroscopy in interventional radiology with the patient under conscious sedation.[2] Nephrostomy tubes provide temporary or permanent drainage for the urinary collection system. They also provide for insertion of stents, divert urine from urethral fistulae, and allow access for kidney

stone removal or biopsy.

Nursing Plan of Care

The nursing plan of care for patients with tubes or drains is developed using a holistic approach, which requires a thorough understanding of the anatomy and physiology of the body systems involved as well as the rationale for the selected tube or drain. Knowledge of these factors combined with assessment of the external factors affecting the patient provides a clear picture of what is needed to manage patient needs. External factors include the physical environment of care, socioeconomic issues, family support, and psychosocial concerns.[1,2,5–7] A comprehensive plan of care involves an interdisciplinary team approach that incorporates the reasons for the IUC with daily assessment and care of the skin around the tube or drain, early identification of problems, and removing the IUC when no longer required. Patient and family education is also an important aspect of care and includes nursing care needs along with signs of complications.

Interdisciplinary Team Approach

Achieving a comprehensive plan of care to meet the complex needs of patients with tubes or drains requires a collaborative team. This team can include, but is not limited to, the primary care physician, surgeon, radiologist, gastroenterologist, physical therapist, social worker, case manager, registered dietitian, primary nurse, and wound, ostomy, and continence (WOC) nurse. The care plan needs to include interventions aimed at tube stabilization, containment and measurement of effluent, skin management, nutritional support, patient mobilization, and effective communication practices with the medical team.[1,2]

The registered dietitian plays a vital role on the interdisciplinary team because he or she is educationally prepared to determine the appropriate formula for enteral feeding, hydration needs, and management of diarrhea. Nurses caring for patients receiving enteral tube feedings need to develop policies that include monitoring hydration, assessing for patient tolerance by evaluating gastric aspirates, maintaining the stability of the device, and engaging with the physician and pharmacist for optimal medication management.[2]

Education

Education and discharge planning are the next step in the plan of care. Each member of the collaborative team will contribute to the education needs of the patient or caregiver; understanding these needs in turn helps the team to determine discharge needs.[2] Education should be based on the principles of adult learning and include an explanation of the purpose of the drain or tube, the need for and how to stabilize the tube, management of the containment device, skin care, any feeding schedules and procedures, and the signs and symptoms of complications. Determine whether the complexity of care is beyond the ability of the patient and, if so, ascertain whether a caregiver is willing and able to provide support.[2] The details of care should be demonstrated, reviewed, and evaluated, and written instructions should be provided. What, if any, support will be needed after discharge? How stable is the patient? Is discharge to the home a realistic option?

Skin Care Around the Tube

The skin around the insertion site should be assessed daily for signs of skin breakdown, infection, or excoriation. Leakage of gastric contents onto the skin is not uncommon; when it occurs, the tube must be examined for leaks and stabilized to prevent movement in and out of the abdomen. If the tube migrates or stretches, leakage of gastric contents from the puncture site can damage the skin. For the first week after tube placement, the insertion site and external retention device should be cleaned with normal saline. Half-strength hydrogen peroxide can be used to remove crusts. After the first week, the stoma site and the area beneath the external retention device should be cleaned daily with a pH-balanced skin cleanser. pH-balanced solutions do not damage the skin, leave little residue, and protect the skin without changing its pH.

G tubes should be checked for their ability to be moved in and out of the abdominal wall; ¼ inch (0.5 cm) of movement is normal. If the tube cannot be moved even slightly, notify the physician. Often called “buried bumper syndrome,” the inability to move the tube may indicate that it has become embedded in the tissues and can erode into the stomach wall. G tube insertion sites should not be packed tightly with dressings between the tube and skin. Occasionally, hypergranulation tissue will develop at the puncture site. This tissue is not harmful and usually results from irritation from the tube.

Tube blockage is common with crushed medication, inadequate flushing (particularly with nasojejunal tubes, which tend to be longer and of finer bore), or precipitation of protein in the feeding. To avoid a clogged feeding

tube, thoroughly flush enteral feeding devices every 4 to 6 hours during continuous feedings and whenever feedings are on hold, before and after administration of feedings and medications, and after checking residuals. Always use a large syringe (30 to 60 mL) for flushing to prevent rupture of the tube. Irrigate the tube with 20 to 30 mL of tepid water. Tubes can generally be unblocked using a variety of solutions, such as water, carbonated soda, pancreatic enzymes, or commercially available products. No fluid has been found to be superior to water for maintaining patency. If the feeding tube has a Y-port connector, flush through the side port. Otherwise, disconnect the feeding infusion device and flush directly into the tube.

Practice Point

Medications that can be administered in liquid form are best suited for enteral feeding tubes. Water flushes between medications and feedings will help prevent tube obstruction.

Enterocutaneous Fistulae

An enterocutaneous fistula (ECF) is an abnormal connection between two epithelial surfaces.[8,9] This connection can occur between two internal organs or can lead from an internal organ to a body surface. The physiological origin and exit point of the fistula are the basis for naming and evaluating a fistula (Table 19-4).

Table 19-4 Types of Fistulae

Fistula	Connection
Colocutaneous	Colon to skin
Rectovaginal	Rectum to vagina
Entero-entero	Small intestine to small intestine
Enterocutaneous	Small intestine to skin

Although ECFs are not common, certain diseases or conditions will predispose a person to develop an ECF. Inflammatory bowel disease, such

as Crohn's disease, can cause spontaneous fistula development and increase the risk of a postoperative fistula.[8–10] These patients require medical support and regular monitoring for complications. Other predisposing factors include traumatic abdominal injury, peritonitis, small bowel obstruction, malnutrition (especially prior to abdominal surgery), the presence of devascularized tissue, and radiation enteritis.[8] Many times, these conditions can result in a large, deep abdominal wound with or without exposure of the bowel wall.

An ECF is one of the most challenging complications for the nurse to manage and devastating for the patient. Fluid and electrolyte imbalances, erosion of adjacent skin secondary to the corrosive nature of the effluent, malnutrition, dehydration, and the psychosocial well-being of the patient all require ongoing monitoring and evaluation for the best outcomes. The mortality rate for patients with ECF ranges from 12% to 25%, usually the result of sepsis, malnutrition, and dehydration.[7–11] The cost to managing a fistulae, including diagnostic tests, nutritional support, medical supplies, and medical care, is estimated at nearly $262,140.[12] With the current reimbursement of care environment, it is important to identify early on and provide nutritional, nursing, and medical support for the patient at risk of developing fistulae. Advances in nutritional support options, antibiotics, containment devices, and managing fluid and electrolyte balance have contributed to improved care and patient recovery from this type of event.[11]

Classification of ECFs

ECFs can be classified in a number of ways based on the type and amount of effluent present, the anatomy involved, and the complexity of the tract.[8] Classifying the fistula provides assessment and documentation parameters as well as clues to interventions aimed at both stabilizing and managing the patient. A simple fistula has a direct tract without an abscess. A type I complex fistula is associated with abscesses and multiple organ involvement.[8] Type II complex fistulae open into the base of an open wound; these are among the most challenging and devastating fistulae to care for.[8]

The amount of effluent is also important. Low-volume fistulae are defined by drainage amounts of less than 500 mL in a 24-hour period, while high-volume fistulae have drainage in excess of 500 mL over 24 hours.[9,11] Effective monitoring and measuring of ECF output are critical in managing

fluid balance. Generally, the more proximal the fistula is located, the higher the amount of output will be.[6,9,11]

The color and consistency of the effluent provide clues to the origin of the ECF. Gastric drainage is clear to light yellow-green in color with a watery consistency; the pH will be about 3.0.[8] Biliary drainage is a gold to deep green viscous liquid with a pH of 7.5.[8] Pancreatic drainage is clear and watery with a pH of 8.3.[8] Considering that the normal pH of skin is 4.5 to 5.5, the issue of protection of adjacent skin is a primary goal of care.[8,9]

Goals of Management

The goals of management for a patient with an ECF are complex and require the skills and attention of a collaborative team.[11] Early identification of the presence of an impending fistula provides an opportunity to manage complications in a timely fashion.[8,13] While an ECF is not a common event, paying attention to certain signs in the patient at risk provides clues to the need for early care. Following abdominal surgery or in cases of inflammatory bowel disease, signs such as fever, localized edema, induration, progressive local discomfort, changes in fluid and electrolyte balance, and altered mental state should be monitored closely and communicated to the physician or surgeon.[8,13,14] Once a person is predisposed to develop an ECF, some direct causes include a breakdown of the anastomotic site due to sepsis or tension, peritoneal abscess, altered blood supply, steroid therapy, and malnutrition.[15,16]

Once an ECF is suspected, it is important to identify the extent and source of the fistula. This can be accomplished through a fistulogram, computerized tomography, magnetic resonance imaging (MRI), or positron emission tomography (PET) scan.[8,15] Determining the extent of the ECF provides the information needed to develop a plan of care and guides surgical intervention if needed. Immediate stabilization of the patient by managing his or her electrolytes and fluids is a critical step.[8,15,17,18] The drainage from an ECF includes sodium, potassium, magnesium, zinc, proteins, digestive enzymes, and fluid, the loss of which will cause fluid and electrolyte depletion and malnutrition.[8,9,11,15,18] The corrosive nature of the effluent will erode the surrounding skin and cause pain.[8,9,13,15,16] Supportive efforts are aimed at correcting these problems through intravenous support as well as quantification and containment of effluent.[8] It

may be necessary to withhold oral intake (NPO) initially until all factors are identified and the extent of the ECF is determined.[15,16]

Medical Management

The medical management of fistulae has four primary goals: stabilize fluid and electrolyte imbalance, provide nutritional support, manage sepsis, and determine the exact location of the fistula. Supporting these goals provides optimal care for the patient and increases the chance of spontaneous closure of the fistula.[15,18] Although spontaneous closure of ECFs occurs in only about a third of cases, this is still the desired outcome. A number of strategies are recommended to achieve these goals. Involving a registered dietitian is critical. Along with the physician, he or she will monitor electrolytes and fluids for replacement needs as well as determine required nutritional support.[17,18] Initially, the patient may be NPO while total parenteral nutrition (TPN) is initiated. Resuming feedings through either the oral or enteral route will prevent mucosal villous atrophy in the small bowel and promote optimal nutritional absorption.[9,15,17–19] TPN is a viable option if enteral feeding is not possible; however, the risk of hyperglycemia is always present.[19–22] Continued monitoring of serum electrolytes and nutritional markers such as prealbumin and transferrin provides important clues to the effectiveness of the nutritional plan of care.[19] Pharmacologic treatments are also useful medical management strategies. The use of octreotide or somatostatin has been shown to decrease the volume from a high-output fistula in some cases,[8,9,19–21] although the results are not always predictable. Mortality rates increase with sepsis, so the identification and treatment of sepsis in these situations are an important aspect of care[23] (Table 19-5).

Table 19-5 Pharmacologic Options in Fistulae Management[20,21]

Classification	Examples	Effect on Effluent
Antimotility drugs	Loperamide, codeine, tincture of opium	Slows intestinal motility and allows more nutrient absorption time
Antisecretory drugs	Proton-pump inhibitors, histamine-2 receptor agonists, octreotide, somatostatin	Slows gastric secretions and slows intestinal motility
Supplements	Bile salts, pancreatic enzymes	Improve absorption of lipids and proteins

Spontaneous closure of an ECF is defined as closing with medical

management in 6 to 8 weeks.[20,22,24–26] If spontaneous closure does not occur within this time frame, surgical intervention may be indicated.[20,21,25,26] Situations that preclude spontaneous closure include sepsis, malnutrition, distal obstruction, a matured fistula (i.e., the fistula tract has epithelialized), mucosa exposure, and consistently high output of effluent. Recent attention has been placed on the use of biological fibrin glue (a combination of fibrinogen and thrombin) to close the fistula.[18] The procedure initially involves debridement of devitalized tissue and saline irrigation followed by injection of fibrin glue directly into the fistula[18,27,28]; more than one injection may be required. The expected result of this process is stimulation of coagulation within the fistula tract.

When all medical management options fail to result in closure of the fistula, surgical intervention may be needed.[21,29] Surgery must be planned carefully and be preceded by fluid and electrolyte balance, nutritional support, and sepsis and inflammation control. This will reduce the complications of peritonitis and the development of additional fistulae.[30–32] Timing of surgical intervention is critical. Surgical approaches are aimed at identification of the origin of the fistula and a surgical takedown. For those patients who are not stable enough for surgery, medical management should continue.

Nursing Management

A holistic approach to patient care begins with a thorough patient assessment prior to implementation of interventions.[8,9] Review the medical record for the history of the problem as well as the current situation. What baseline tests are available, and what should be continuously monitored? Assessment and documentation of issues surrounding the ECF include physical assessment of the affected area as well as the patient's psychosocial needs and discharge planning needs.[8,9] Take time at this point to consider options that will best facilitate the needs of the patient as a plan of care is developed. Skin care is of primary concern in these patients. Maintaining skin integrity reduces the need for pain management due to erosion of the skin, reduces infection, and improves the integrity of the management system.[8,9]

WOC nurses can be invaluable in caring for a patient with an ECF because these nurses add knowledge about a variety of pouching systems and skin care products to the plan of care.[8,9] Care is centered on

containment and measurement of effluent, odor control, perifistula skin care, cost containment, and education of those involved in care.[8,9,33,34] Management options include the use of ostomy pouches and accessories, skin barriers, wound care pouches and accessories, catheter holders, odor-controlling agents, and suction. Many times, a combination of these measures will be needed for optimal care. ECFs with an output of less than 100 mL/d can be managed with standard dressings, including charcoal-based dressings if odor is an issue.[8,9,33,34]

When the effluent is of high output and if the fistula is located within an abdominal wound, the containment plan becomes complex, and more than one person may be required to help in the application of the management system. Consider the high-output ECF located within a skin crease. The amount of effluent, the location of the ECF, and the skin crease pose serious challenges to maintaining a predictable seal of a pouching system. WOC nurses will employ the use of solid skin barriers, skin barrier paste, and powder as well as pouching system that facilitates accurate quantification of the output.[8,9] Fistulae presenting within an abdominal wound pose a particular challenge from the standpoint that not only is the fistula draining into the wound, but the wound must be managed and containing the effluent requires creativity and fistula management pouches for effective care.[8,9,34] Some authors recommend the use of an isolation technique in which the fistula is isolated using a combination of solid skin barriers, skin barrier paste, pouching, and accessory products followed by the application of negative pressure wound therapy (NPWT) for wound healing.[35,36] By creating a predictable wear time with pouches, dressings, suction, and NPWT, a budget can be set up based on how many dressing changes are required on a weekly basis and additional care goals can be achieved.[36–39] The complications of immobility are well documented in the literature, and a predictable wear time allows the patient more freedom to participate in therapy or other activities.

NPWT was introduced to wound care more than 20 years ago; its use as an adjunct in the management of ECFs has evolved more recently. While further study is needed to define the efficacy of NPWT in the management of ECFs, recent literature demonstrates the value of this option in reducing effluent in high-output fistulae as well as in optimizing the care of the patient. It is unclear whether NPWT increases the chances of spontaneous closure, but the improved tissue oxygenation and reduction of effluent are factors that improve outcome.[36–39] Also not clear are the circumstances

under which NPWT is most helpful. Some authors use the criteria of exposed bowel to preclude the use of this option, while others suggest the use of petrolatum-impregnated gauze as a contact layer prior to the application of the sponge and negative pressures of 75 mm Hg.[38] The use of NPWT in the care of ECF has been demonstrated in some cases to reduce effluent and allow the patient improved mobility and may be considered as a possible treatment modality. If NPWT will be used, it is important for the clinician to follow manufacturer guidelines for use. Consistent monitoring of serum electrolytes, nutritional status, effluent, and response to care coupled with physician communication provides information that can help determine the effectiveness of the plan of care.[36,38,39]

Principles of Pouching

In cases in which NPWT is not an option or has failed, pouching systems are available to support the plan of care by containing effluent and odor and providing for wound care. Evaluation of abdominal topography in the supine, sitting, and standing positions will expose any skin folds, creases, and scars that will undermine the pouch seal.[8,9] The use of skin barrier paste and moldable solid skin barriers will even out the skin contours and promote a level pouching surface. Solid skin barriers trimmed to form a wedge shape assist in evening out contours. The use of radial slits (darts) offers flexibility while simultaneously evening skin contours.[8,9] Each layer of wedges should be ½ inch larger than the preceding layer to prevent effluent from leaking between the wedges and pouch barrier. Apply the skin barrier as close to the wound margins as possible. If perifistula skin was left exposed, add protection to the exposed skin with barrier ointments and powders once the system is in place.[8] The use of pouches with windows facilitates this process.

Practice Point

Even the most skilled nurse will be challenged pouching a wound with an ECF, but an individualized approach, assessment of skin contours, creativity, and persistence will yield a predictable system that meets management goals.

Case Management

At some point in the care of the patient with an ECF, case management/discharge planning must occur. The high cost of care and implications for psychosocial well-being necessitate involving a case manager before the patient is discharged to home.[9,13] Certain questions should be asked in deciding when discharge to home is a viable option. Can an effective pouching/containment system be achieved for a predictable time (at least 24 hours)? Is someone available and willing to care for the patient and learn the management system? Can the caregiver demonstrate the procedures of care? Also consider the availability of supplies, the durable medical supplier, and whether insurance will cover the needed products. If the needed products are not covered, what financial arrangements can be made? Is home health care needed and an option?

Patients who develop an ECF are at high risk for complications from sepsis, malnutrition, dehydration, and skin breakdown.[8,9,15,18] The mortality associated with ECF demonstrates the need for early identification and management, which begins with assessing for patients with predisposing factors and early signs of ECF development.[8,21] The goals of care include stabilizing the nutrition and fluid and electrolyte status, containing effluent, managing sepsis, and employing medical and surgical management where needed.[8,9,15,18,21] Nursing care centers on continuous monitoring of serum electrolytes, communicating with the registered dietitian and surgeon, patient assessment and planning, and containment and quantification of effluent.[8,9,13,18,19,21] While wound dressings and pouching systems provide options in the care of these patients, newer treatments such as NPWT are fast becoming a standard option to consider to help reduce volume in high-output ECFs.[21,39–41] Continued review of current literature is recommended for healthcare providers caring for patients with ECF.

Summary

Understanding the various types of drains and tubes and when they are used is a critical first step in developing an effective treatment plan. Maintaining the patency of drains and tubes as well as assessing and protecting the skin around them are imperative. Fistulae are classified by the tissue involved, whether they are external or internal and simple or complex, and by the amount of effluent draining from them. The name given to the fistula

provides information about which two body areas are connected. Because fistula effluent is so odorous and caustic to the skin, skin protection is very important. Consideration must be given to fluid and electrolyte balance and nutritional management. Care of the patient with tubes, drains, or fistulae is challenging and requires the care of an interdisciplinary team.

PATIENT SCENARIO

Multiple Drains, by Linda Stricker and Amelia Agostinelli

Clinical Data

Mr. RM is a 55-year-old male who presented with sudden abdominal pain, nausea, and vomiting. His medical history includes newly diagnosed diabetes mellitus. On evaluation, he was found to have severe, acute pancreatitis, and his condition rapidly deteriorated with hypotension, tachycardia, and elevated temperature. He was medically stabilized and transferred to intensive care for further management. Shortly thereafter, he was intubated to support the airway and help manage the septic shock until stable enough for a surgical consult.

Case Discussion

Mr. RM was managed through aggressive fluids, respiratory care, blood glucose management, and nutritional support. Further evaluation demonstrated gallbladder involvement and duodenitis. His condition further deteriorated and was complicated by the development of necrotizing pancreatitis and a colonic leak. Abdominal drains were placed to help manage the situation. Secondary to massive sepsis, the drain sites started to leak large amounts of fluid around the drains creating a containment and nursing management problem.

As seen in the first photo (Fig. 19-1), the abdomen has two drain tubes; the exit sites have marked erythema, erosion, and hydrated tissue at the drain sites. Because the two drains are in close proximity, creating a reliable and sustainable containment system is challenging.

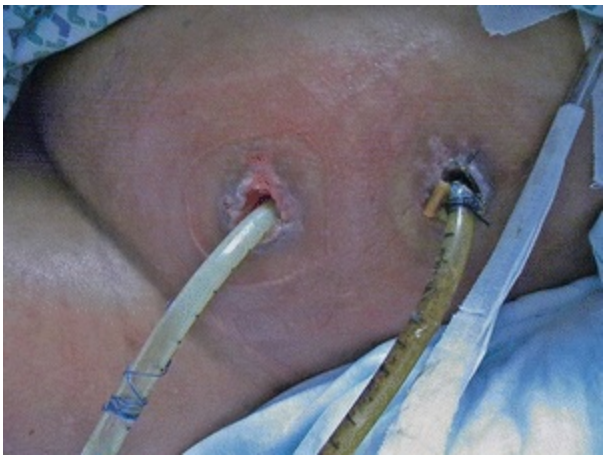


Figure 19-1. Initial presentation in critical care with two abdomen drains in place.

Our nursing goals to manage the drains included:

1. Stabilize the tubes to prevent further erosion of the abdominal tube sites
2. Contain the drainage through a reliable pouch seal while providing access for the tubes through the pouching system
3. Quantify the drainage
4. Promote optimal environment for wound healing

Tube Management

To both stabilize the drains and maintain a reliable pouching system, WOC nursing recommendations included the use of a two-piece pouch with a carboxymethylcellulose (CMC) skin barrier, gelatin-based moldable rings with tack on both sides, and a tube access port (Fig. 19-2). The pouch, rings, and paste were applied as follows:

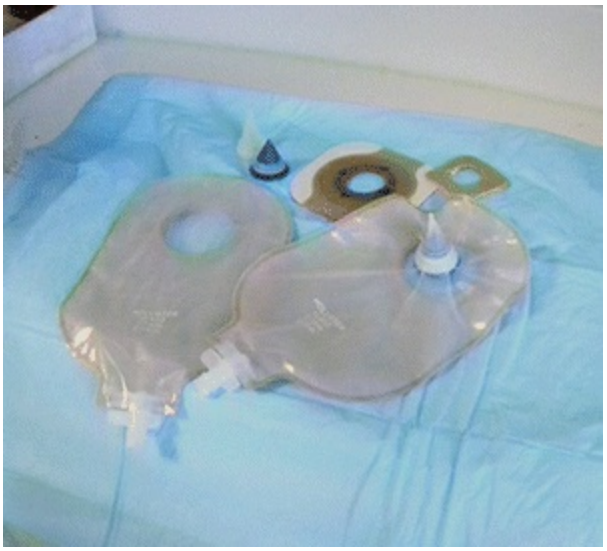


Figure 19-2. Pouching supplies.

1. The pouch was prepared with the addition of a tube access port through the anterior side of the pouch.
2. The gelatin-based skin barrier moldable ring was applied around the first drain tube (Fig. 19-3) and secured with the face plate of the two-piece pouching system to provide a reliable seal to the liquid drainage leaking around the tube (Fig. 19-4).
3. With the help of hemostats and a water- soluble lubricant, the drain was pulled through the drain tube access port to facilitate tube drainage (Fig. 19-5).
4. The pouch could then be coupled to the face plate (Fig. 19-6).
5. The second drain tube (not leaking) was managed with the gelatin-based skin barrier ring and face plate with tape collar trimmed to fit close the first drain tube (Fig. 19-7).
6. The pouch end was closed and allowed to drain by gravity. The spout end of the pouch allowed for ease of emptying and quantifying the drainage.

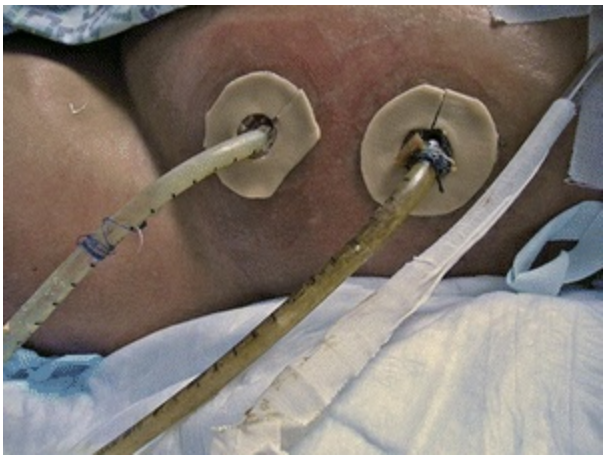


Figure 19-3. Moldable skin barrier rings around the tubes.

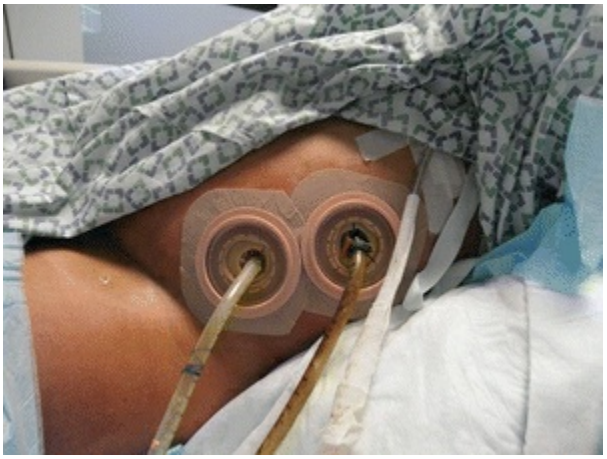


Figure 19-4. Face plate of the two-piece pouching system in place around the tubes.

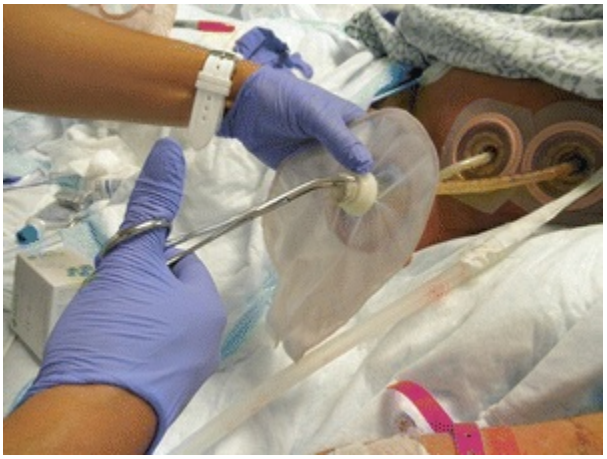


Figure 19-5. Applying the tube access port.

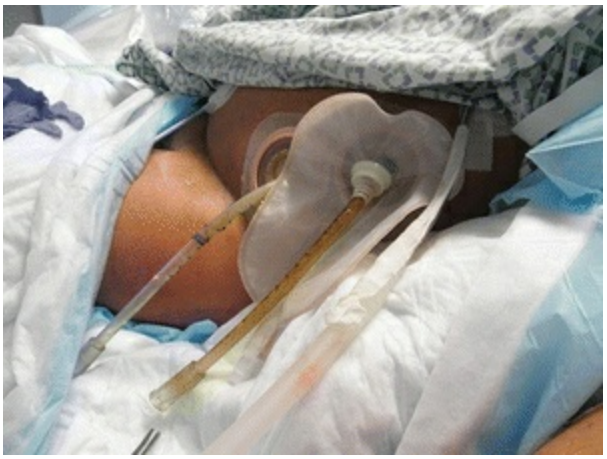


Figure 19-6. Pouch applied to face plate.

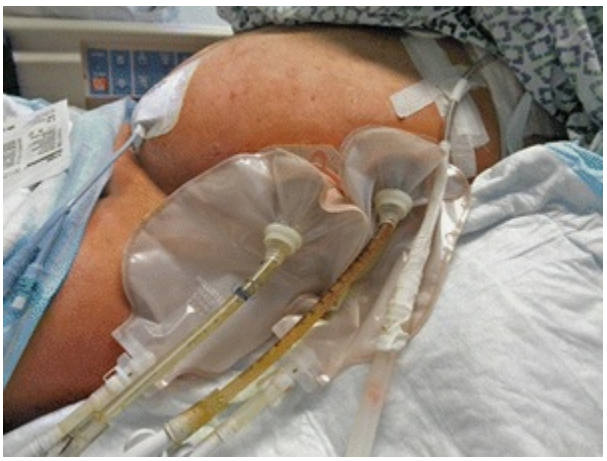


Figure 19-7. Both pouches secured.

Conclusion

This approach to management of leaking abdominal drains complicated by sepsis resulted in a 4-day seal. This system allowed for containment of drainage around the tube while facilitating tube drainage with tube stabilization. This provides an environment that prevents further erosion of the abdominal defect at the tube site while simultaneously providing a cost effect method to minimize skin damage and reduce the need for additional product to manage the drainage.

Show What You Know

1. Which statement about fistula care is TRUE?

A. NPWT is contraindicated for treating fistulae.
B. External fistulae empty into an organ.
C. Low-output fistulae produce less than 200 mL of effluent per day.
D. Fistulae that open into granulating wounds have the best chance of healing.

2. Optimal functioning of enteral feeding tubes depends on which primary nursing considerations?

A. Hydration, location of tube, diagnosis
B. Skin care, hydration, location of tube
C. Tube stabilization, hydration, nutrition
D. Nutrition, location of tube, continuum of care

3. Which of the following conditions predisposes a patient to formation of an enterocutaneous fistula?

A. Normal nitrogen balance
B. Albumin level of 4.0 g/dL
C. Blood glucose of 80 mg/dL
D. Crohn's disease

4. A patient with an enterocutaneous fistula is averaging an output of 9,600 mL over a 24-hour period. The direct care staff is concerned about pouch overflow and leakage. What would you advise?

A. Discontinue the pouch and switch to gauze dressings.
B. Connect the pouch to bedside gravity drainage.
C. Add more skin barrier paste and tape to caulk the leaks.
D. Restrict enteral and intravenous fluids.

5. Fistulae that begin in the ileum and end within an abdominal wound would be classified as:

A. Colocutaneous fistula.
B. Entero-entero fistula.
C. Rectovaginal fistula.
D. Enterocutaneous fistula.

6. You would expect fistulae originating from the pancreas to have what type of effluent?

A. Clear, watery, pH 8.3

B. Green, viscous, pH 7.5
C. Yellow-green, watery, pH 3.0
D. Brown, viscous, pH 7.8

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Atypical Wounds

20

Freya Van Driessche, MS
Robert S. Kirsner, MD, PhD

Objectives

After completing this chapter, you'll be able to:

- recognize the importance of identifying atypical wounds
- explain the need for wound biopsies in determining the etiology of an atypical wound
- describe the various clinical manifestations of atypical wounds.

Types of Atypical Wounds

Prolonged pressure (pressure ulcers), venous insufficiency (venous leg ulcers), complications of long-standing diabetes mellitus (diabetic foot ulcers), and poor vascular supply (arterial ulcers) are the most common causes of chronic wounds. Wounds resulting from uncommon etiologies, called *atypical wounds*, are less frequently encountered and less well understood. Their prevalence has not been studied extensively, but it is estimated that at least 10% of more than 500,000 leg ulcers in the United States may be due to unusual causes.[1,2] A variety of etiologies may cause atypical wounds,[3] such as infections, external or traumatic causes, metabolic disorders, genetic diseases, neoplasms, and inflammatory processes.

It is critical to recognize when a wound is caused by an etiology other than prolonged pressure, neuropathy, or abnormal vascular supply so that a correct diagnosis can be made and the appropriate therapy provided. A wound should be evaluated for an atypical etiology if:

- it is present in a location different from that of a common chronic

wound
- its appearance varies from that of a common chronic wound
- it does not respond to conventional therapy.

For example, the thigh is an atypical location for a pressure, venous, arterial, or diabetic ulcer and should raise the suspicion of an atypical cause. A wound on the medial aspect of the leg but extending deep to the tendon would be considered atypical despite being in a common location because the depth of this wound is atypical for venous ulcers. Finally, any wound that is not healing after 3 to 6 months of appropriate treatment should raise the consideration of an atypical cause, even if the distribution and clinical appearance are classic for a common chronic wound.

Once a wound is deemed atypical, a tissue sample is critical for histologic evaluation with special stains, tissue culture (for infectious causes), and immunofluorescence testing (for some inflammatory or immune-based causes).

Evidence-Based PracticeEvidence-Based Practice

Tissue samples are mandatory for atypical wounds because many of the unusual causes of wounds can resemble each other, making visual diagnosis alone difficult and risky.

Etiologies of Atypical Wounds

Some of the most commonly encountered etiologies for an atypical wound include inflammatory causes, infections, vasculopathies, metabolic and genetic factors, malignancies, and external causes (Table 20-1). However, a thorough medical history, including epidemiological exposure, family history, personal habits, medications, and concomitant systemic diseases, along with a thorough physical examination, histologic evaluation, and laboratory testing, will provide critical information necessary for a correct diagnosis of an atypical wound.

Table 20-1 Potential Etiologies of Atypical Wounds

Although not all-inclusive, this list presents some of the most commonly encountered etiologies for an atypical wound.

Inflammatory Causes
- Vasculitis
- Pyoderma gangrenosum

Infections
- Atypical mycobacteria
- Deep fungal infections

Vasculopathies
- Cryoglobulinemia
- Cryofibrinogenemia
- Antiphospholipid antibody syndrome

Metabolic and Genetic Causes
- Calciphylaxis
- Sickle cell anemia

Malignancies
- Squamous cell carcinoma
- Basal cell carcinoma
- Lymphoma
- Kaposi's sarcoma

External Causes
- Burns
- Bites
- Stings
- Radiation

Inflammatory Causes

Among the most interesting—and probably more common—causes of atypical wounds are the inflammatory ulcers. Although a variety of inflammatory and immunologic diseases affect the skin, two relatively common causes of inflammatory ulcers are vasculitis and pyoderma gangrenosum.

Vasculitis

Vasculitis is defined as inflammation and necrosis of the blood vessels, which can ultimately result in end-organ damage—in this case, the skin.[4] Although about half of the cases are idiopathic,[5] vasculitis is a reaction pattern that may be triggered by certain reactants, among which are underlying infections, malignancy, medications, and connective tissue diseases (Table 20-2). Clinically, vasculitis varies depending on the size of

the underlying vessel affected. For example, lesions may include a reticulated erythema due to disease of the superficial cutaneous plexus, or they may present as widespread palpable purpura, necrosis, and ulceration due to disease in larger, deeper vessels (Fig. 20-1). Often, vasculitic lesions on the skin are just one component of the patient's pathology—they may also have involvement of different end organs such as the kidney, lung, central nervous system, and gastrointestinal tract.[6]

Table 20-2 Potential Etiologies of Vasculitis

Although not all-inclusive, the most common causes of vasculitis are listed below.

Infections

- *Streptococcus* spp.
- *Mycobacterium tuberculosis*
- *Staphylococcus* spp.
- *Mycobacterium leprae*
- Hepatitis viruses A, B, and C
- Herpes virus
- Influenza virus
- *Candida* spp.
- *Plasmodium* spp.
- Schistosomiasis
- HIV

Medications

- Antibiotics (penicillins, cephalosporins, minocycline, sulfa drugs)
- Granulocyte macrophage colony stimulating factor
- Propylthiouracil
- Levamisole (often found in impure cocaine)
- Nonsteroidal anti-inflammatory drugs
- Thiazides
- Allopurinol
- Tumor necrosis factor–α inhibitors

Chemicals

- Insecticides
- Petroleum products

Foods

- Milk
- Gluten

Connective Tissue and Other Inflammatory Diseases

- Systemic lupus erythematosus
- Dermatomyositis
- Sjögren's syndrome
- Rheumatoid arthritis
- Behçet's syndrome
- Cryoglobulinemia
- Scleroderma
- Primary biliary cirrhosis

Malignancies

- Lymphomas
- Leukemias
- Multiple myeloma

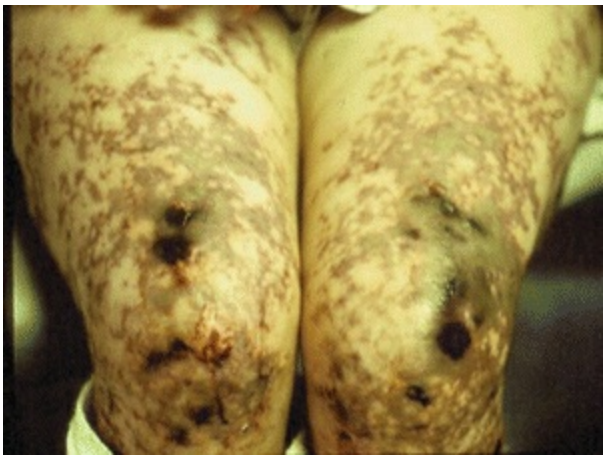


Figure 20-1 Vasculitis. This photograph shows reticulated erythema and necrotic ulcers on the thighs of a patient with vasculitis.

Circulating immune (antibody–antigen) complexes, which deposit in blood vessel walls, are the cause of many types of vasculitis.[7] Tissue biopsies will confirm the presence of vasculitis if performed early, and biopsies of perilesional skin may detect the type of immunoglobulin involved in the process. Biopsies performed later in the course of lesion development may fail to reveal immunoreactants or inflammatory cells, and their by-products will degrade immunoglobulins. Tissue culture may be helpful if the vasculitis is due to an infectious process. A histologically confirmed diagnosis mandates evaluation of other organ systems and an attempt to determine the etiologic factor.

If identified—and if possible—the causative agent should be addressed. Additionally, treatment of the vasculitis is based on the extent of the disease (Table 20-3). Mild disease that is limited to the skin can be treated with supportive care, such as leg elevation and dressings. Treatments with limited adverse effects, such as colchicine, dapsone, antihistamines, or nonsteroidal anti-inflammatory agents, also may be used. If skin disease is extensive or systemic involvement is present, more aggressive treatment such as systemic steroids, anti-inflammatory agents, or immunosuppressants may be needed[8] (Table 20-4).

Table 20-3 Diagnostic Tests for Vasculitis

Use the following tests to determine the etiology of vasculitis.

- Antineutrophilic cytoplasmic antibody
- Rheumatoid factor
- Antinuclear antibody
- Hepatitis A, B, C profile
- Complete blood count
- Antistreptolysin O titer
- Cryoglobulin level
- Serum protein electrophoresis
- Chest radiograph
- Purified protein derivative test
- Throat culture
- Tissue culture

To determine the extent of disease, use these tests:

- Urinalysis
- Stool guaiac
- Chest radiograph
- Renal function tests
- Liver function tests
- Complete blood count

Table 20-4 Vasculitis Treatment Options

Extent of Disease	Treatment Options
Mild	• Leg elevation • Compression dressings • Antihistamines • Nonsteroidal anti-inflammatory drugs • Anti-inflammatory antibiotics • Topical steroids • Support stockings • Dapsone • Colchicine • Potassium iodide
Extensive or systemic	• Dapsone • Systemic steroids • Stanozolol • Cyclophosphamide • Methotrexate • Azathioprine • Cyclosporine • Plasmapheresis • Mycophenolate mofetil • Tacrolimus • Other anti-inflammatory or immunosuppressant drugs

Pyoderma Gangrenosum

The term *pyoderma gangrenosum* actually refers to a disorder that is neither infectious nor gangrenous. Rather, it is an inflammatory process causing painful skin ulcers of which the etiology is still not fully understood. It is believed to be due to dysregulation of the innate immune system and alterations in neutrophil chemotaxis. Pyoderma gangrenosum is characterized by the appearance of one or more chronic ulcerations with violaceous undermined borders[9] (Fig. 20-2). It mainly affects adults, and its usual course is that of recurring, destructive ulcers, which begin as pustules and resolve with cribriform scars. Several clinical variants of pyoderma gangrenosum have been described, including ulcerative, pustular, bullous, vegetative, and peristomal types.

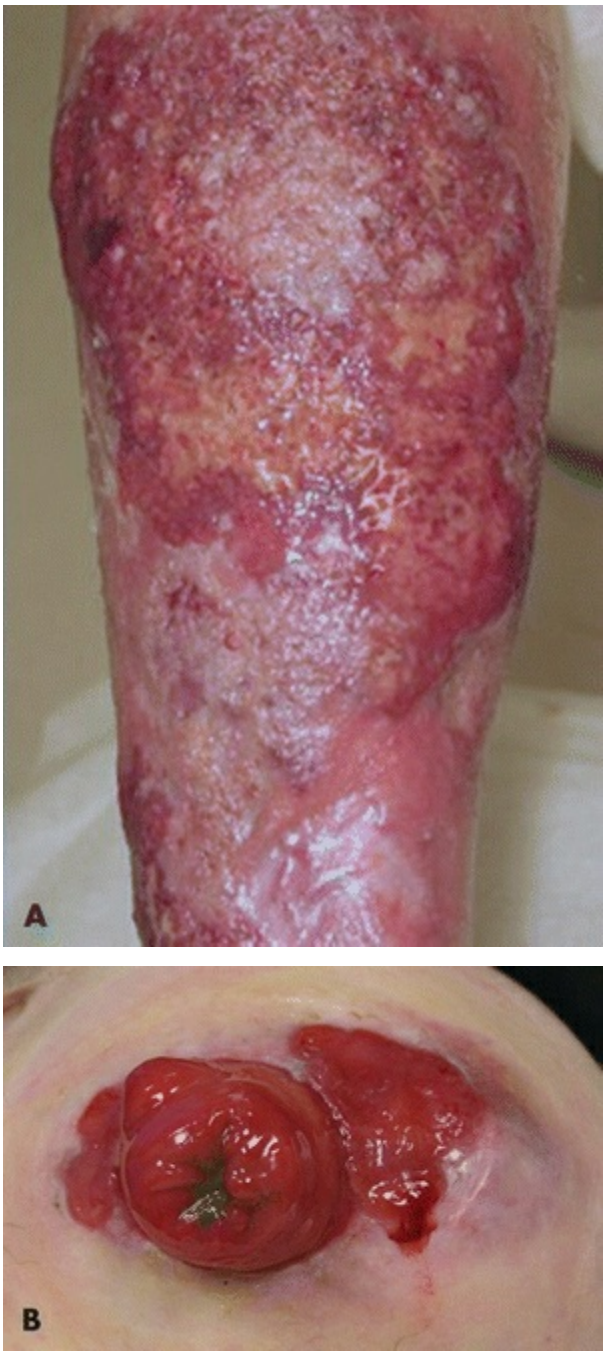


Figure 20-2. (A) Pyoderma gangrenosum in a patient with inflammatory bowel disease and pyoderma gangrenosum, this ulcer on the lateral leg shows areas of cribriform scarring. **(B)** Peristomal pyoderma gangrenosum.

Practice PointPractice Point

Because a diagnostic test to confirm pyoderma gangrenosum does not exist and a number of other conditions may resemble it

clinically, a correct diagnosis relies on the clinical presentation and exclusion of other causes.

It is important for the clinician to search for underlying diseases when a diagnosis of pyoderma gangrenosum is rendered because it is associated with other conditions in more than 50% of patients[10] (Table 20-5). Among these are inflammatory bowel disease, arthritis (seropositive and seronegative), monoclonal gammopathies, and other hematologic disorders and malignancies. Pyoderma gangrenosum lesions can occur around the stoma in persons with inflammatory bowel disease[11] (Fig. 20-2).

Table 20-5 Systemic Diseases Associated with Pyoderma Gangrenosum

No diagnostic test exists to confirm the presence of pyoderma gangrenosum. In addition, pyoderma gangrenosum is typically associated with other conditions.[8] Reported associations are listed below.

Inflammatory Bowel Disease
- Ulcerative colitis
- Regional enteritis
- Crohn's disease

Arthritis
- Seronegative arthritis
- Rheumatoid arthritis
- Osteoarthritis
- Psoriatic arthritis

Hematologic Abnormalities
- Myeloid leukemia
- Hairy cell leukemia
- Myelofibrosis
- Myeloid metaplasia
- Immunoglobulin A monoclonal gammopathy
- Polycythemia rubra, polycythemia vera
- Paroxysmal nocturnal hemoglobinuria
- Myeloma
- Lymphoma

Immunologic Abnormalities
- Systemic lupus erythematosus
- Complement deficiency
- Hypogammaglobulinemia
- Hyperimmunoglobulin E syndrome
- Acquired immunodeficiency syndrome

Pathergy (the development of lesions in areas of trauma) plays a role in the development of pyoderma gangrenosum wounds. In susceptible people, even minimal trauma to the skin can result in the production of pyoderma gangrenosum lesions, such as pustules or ulcers.

Curative treatment does not exist. The course of pyoderma gangrenosum waxes and wanes; however, corticosteroids usually are helpful.[12] For limited or mild disease, topical or intralesional steroids may be used. For more severe or widespread disease, systemic steroids can be used, although their adverse effects limit long-term use. In addition, immunosuppressant or anti-inflammatory agents may also be of value; for example, cyclosporine also appears quite effective in treating this disorder (Table 20-6). Infliximab, a monoclonal antibody to tumor necrosis factor-α, has been shown to be useful for patients with pyoderma gangrenosum in a controlled, randomized, double blinded study.[13] Infliximab is FDA approved to treat Crohn's disease, rheumatoid arthritis, ankylosing spondylitis, psoriatic arthritis, and ulcerative colitis.

Table 20-6 Pyoderma Gangrenosum Treatment Options

Type of Treatment Options	
Topical	• Topical steroids • Topical tacrolimus • Topical cyclosporine • Nicotine patch (case reports) • Intralesional steroids
Systemic	• Steroids • Antibiotics (dapsone and minocycline) • Cyclosporine • Infliximab and other biologics • Azathioprine • Methotrexate • Chlorambucil • Cyclophosphamide • Thalidomide • Tacrolimus • Mycophenolate mofetil • Granulocyte apheresis • Intravenous immunoglobulin • Clofazimine

Infectious Causes

Infectious causes of atypical wounds may be due to a variety of different organisms, some of which are not commonly encountered in the United States. For example, atypical mycobacterial infections (other than leprosy and tuberculosis) and fungal infections (other than dermatophytes and *Candida*) occasionally are detected upon diagnostic testing. Infection caused by *Vibrio vulnificus* may be responsible for lower leg ulcers in geographic areas where there is warm salt water.

Atypical Mycobacterial Infections

Atypical mycobacteria are ubiquitous in the environment and were not generally viewed as human pathogens until the 1950s, when several cases of disease caused by these organisms were reported.[14] Cutaneous infection usually results from exogenous inoculation, and predisposing factors include a history of preceding trauma, immunosuppression, or chronic disease. While *Mycobacteria marinum* is the most common agent of skin infection by atypical mycobacteria,[15] many others have been reported in recent decades (Table 20-7). The cutaneous lesions vary depending on the causative agent and may present as granulomas, small superficial ulcers, sinus tracts, or large ulcerated lesions localized in exposed areas (Fig. 20-3).

Table 20-7 *Mycobacterium* Species That Cause Skin Ulcers

Mycobacterium Species	Clinical Manifestations	Diagnosis	Treatment
M. marinum	• Swimming pool granuloma	Tissue culture	• Antituberculous drugs
M. ulcerans	• Subcutaneous nodule • Deep ulcers	Tissue culture	• Rifampin and Streptomycin/ Clarithromycin
M. scrofulaceum	• Cervical lymphadenitis • Fistulae	Tissue culture	• Surgical excision
M. avium-intracellulare	• Small ulcers with erythematous borders	Tissue culture	• Surgical excision • Chemotherapy
M. kansasii	• Crusted ulcerations	Tissue culture	• Antituberculous drugs • Minocycline
M. chelonae	• Painful nodules and abscesses • Surgical wound infection	Tissue culture	• Erythromycin • Tobramycin • Amikacin • Doxycycline
M. fortuitum	• Painful nodules and abscesses • Surgical wound infection	Tissue culture	• Amikacin • Doxycycline • Ciprofloxacin • Sulfamethoxazole

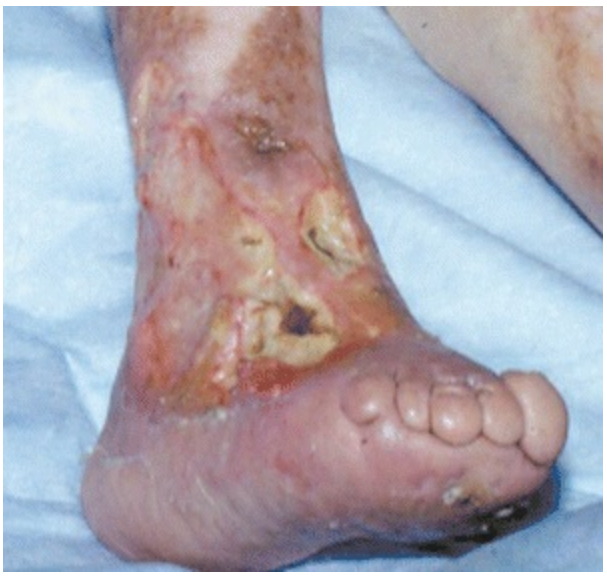


Figure 20-3 Hansen's disease. Shown here are the leg and foot of a patient with Hansen's disease caused by *Mycobacterium leprae*. In addition to neuropathic changes of the toes and plantar aspect of the foot, this patient has a large lateral leg ulcer.

Histologically, mycobacterial infections present as granulomas and abscesses that are difficult to distinguish from those of leprosy and cutaneous tuberculosis. Diagnosis invariably will depend on tissue culture or more recent techniques, such as polymerase chain reaction and gene rearrangement studies.

The appropriate therapy will depend on the causative agent because susceptibility to antibiotics varies. In some cases, simple excision of the cutaneous lesions or a combination of excision and systemic treatment often is most beneficial to the patient.

Buruli Ulcer

Buruli ulcer is a health problem in the tropical areas of many developing countries. Since 1980, Buruli ulcer has emerged as an important cause of human suffering. It was first encountered in 1897 when Sir Albert Cook described large ulcers in patients in Uganda. In the late 1940s, MacCallum identified an organism similar to *Mycobacterium ulcerans* in an ulcer in a 15-year-old in Bairnsdale, Australia. Large ulcers were subsequently observed in patients living along the Nile River in Buruli County, Uganda, in the 1960s, hence the name of the disease. Buruli ulcer is caused by *M. ulcerans*, from the family of bacteria that causes tuberculosis and leprosy.

The World Health Organization (WHO) has defined Buruli ulcer as an

infectious disease of the skin and subcutaneous tissue characterized by painless nodules, papules, plaque, or edema evolving into a painless ulcer with undermined edges and edema. Progression of the disease is associated with extensive sloughing and massive ulceration, particularly over joints, that may lead to contractures. Significant areas of the torso, face, or an entire limb may be involved. Amputation is often required when a limb is involved.[16]

Extensive cutaneous ulcers occur as the disease progresses from the early, more treatable, nodular form to the ulcerative form, which can result in the loss of skin and soft tissue. Although Buruli ulcer is rarely fatal, the associated disabilities and disfigurement are extensive and have a profound impact on quality of life in affected patients[16,17] (Fig. 20-4).

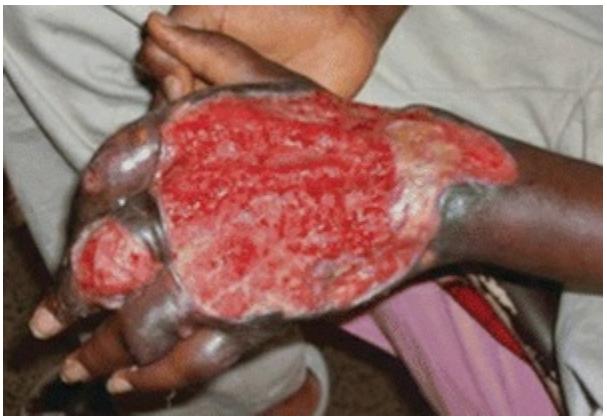


Figure 20-4 Buruli ulcer. This photo shows extensive sloughing and massive ulceration, typically leading to contractures and extensive disability and disfigurement.

The majority of those suffering from Buruli ulcer are between 5 and 15 years of age, but it can affect patients of any age. A prospective study showed that 8-week regimen of rifampicin and streptomycin (injectable) for patients with all stages of Buruli ulcer led to healing in 93% of patients without the need for surgery.[18] A more recent prospective study determined that clarithromycin (oral) could replace the injectable streptomycin after 2 weeks of standard rifampin/streptomycin treatment without loss of efficacy.[19]

Healing often is slow and can be associated with significant functional incapacity due to contractures and amputation. The course of disease can be complicated by the ability of the organism to suppress the immune system of the host. Disease progression is related directly to a decrease in the level of interferon-γ, a Th1 cytokine, as well as a concomitant increase in the Th2

cytokine interleukin-10.[16] Reversing the deficiency of interferon-γ and/or decreasing the interleukin-10 level has been identified as a novel therapeutic target for interventional development.

The disease, its treatment, and the resulting disabilities are a significant burden to patients, their families, and their society. In some countries such as Ghana, patients and families are reluctant to seek treatment due to lack of money or misunderstanding that treatment can lead to amputation.[17] Educational sessions for clinicians to recognize Buruli ulcer early and to change patient, family, and societal resistance to seeking early treatment have met with success, and the number of late cases of this disease in Ghana has declined over the past decade.[16,17]

Deep Fungal Infections

Deep fungal infections of the skin can be divided into subcutaneous and systemic mycoses. The subcutaneous mycoses result from traumatic implantation of the etiologic agent into the subcutaneous tissue, development of localized disease, and eventual lymphatic spread. In rare instances, hematogenous dissemination can occur, especially in immunocompromised hosts. As may occur with sporotrichosis or chromomycosis, ulcers from deep fungal infections are found worldwide and can present in a wide variety of clinical settings.[20]

Systemic mycoses are the result of systemic penetration of pathogenic fungi, with the lungs being the most common port of entry. These infections are restricted to the geographic areas where the fungi occur, especially tropical regions such as Central and South America. After an initial pulmonary infection, the fungi can spread hematogenously or via lymphatic vessels to other organs, including the skin. A decrease in immunity will lead to expression of the fungal infection, as is commonly observed in patients infected with human immunodeficiency virus (HIV).

Practice PointPractice Point

A thorough patient history assists the clinician in considering a diagnosis of systemic mycosis because of the limited area in which the causative fungi occur.

Sporotrichosis

Sporotrichosis is a subacute or chronic fungal infection caused by the fungus *Sporothrix schenckii*. Occurring as a consequence of traumatic implantation of the fungus into the skin, it is often associated with lymphangitis. Less commonly, inhalation of the conidia can lead to pulmonary infection and subsequent spread to the bones, eyes, central nervous system, and viscera. Systemic disease is seen in individuals with impaired immunity, such as alcoholics and AIDS patients.[21]

Sporothrix schenckii, a saprophyte in the environment, has been isolated in a variety of plants and other fauna as well as in animals (bites or scratches from animals, such as armadillos and cats). Individuals whose professional or leisure activities (such as rose gardening or landscaping) expose them to the environment are at greater risk of acquiring the infection. Lymphocutaneous sporotrichosis begins as a papule, which then ulcerates with time. The lesions spread and occur via lymphatic channels traveling from the inoculation site. Sporotrichosis is treated with systemic medications, including saturated solution of potassium iodide, itraconazole, terbinafine, and amphotericin B. Topically applied heat also may be used because the organism grows at low temperatures.

Chromoblastomycosis

Chromoblastomycosis is a subcutaneous mycosis caused by several pigmented fungi, including *Fonsecaea pedrosoi, Fonsecaea compacta, Phialophora verrucosa, Cladosporium carrionii,* and *Rhinocladiella aquaspersa.* These fungi are acquired through inoculation of the causative agents in the skin, after which a mycotic infection develops at the site of entry. These microorganisms can be found in soil throughout the world; however, the disease is most common in tropical and subtropical climates, with the majority of cases seen in South America.[22]

Primarily affecting men ages 30 to 50, the principal lesion is a slow-growing papule that eventuates into a verrucous nodule. Exposed areas are involved, with extremities—especially the lower limbs—being affected in 95% of the cases.[20] The surface of the lesion may be covered by scales or may be ulcerated with serosanguineous crusts. Black dots can be often observed; these dots are rich in fungi and represent the site of transepidermal elimination of necrotic tissue.

Diagnostic examinations should include scrapings from the lesion with potassium hydroxide 20%; tissue samples should be obtained from biopsies

for tissue culture and histology.

The disease tends to be chronic and difficult to treat and may lead to lymphedema and elephantiasis. Ulcerated and cicatricial lesions have been reported to develop into carcinoma. Small lesions can be cured by surgical excision; however, chronic lesions are often resistant to treatment.

Systemic antifungal agents, such as ketoconazole, itraconazole, terbinafine, and amphotericin B, have been used, both alone and in combination, with variable results. Oral flucytosine, alone or in combination with an antifungal, has also been reported to be therapeutic.[22] The new-generation triazole antifungals show promise, as demonstrated by the positive results with posaconazole, although they may be prohibitively expensive.[23,24] Cryosurgery has also been used alone and in combination with antifungal chemotherapy. In addition, local heat therapy of 42°C to 45°C can also be an effective therapeutic modality.

Paracoccidioidomycosis

Paracoccidioidomycosis (South American blastomycosis) is a chronic, infectious disease caused by the fungus *Paracoccidioides brasiliensis*, a saprophyte of soil and decaying vegetation found in tropical and subtropical climates. Infection occurs primarily via a respiratory route with occasional dissemination to other organs, including the skin. Rarely, direct inoculation into the skin can occur. Patients present with painful ulcerative lesions of the mouth, the face or, less frequently, the extremities. Involvement of regional lymphatics is characteristic.[25]

Diagnosis can be established by isolation and identification of the etiologic agent with direct mycologic examination, histopathologic or cytopathologic examination, or culture. Treatment includes trimethoprim—sulfamethoxazole, azole derivatives such as itraconazole and ketoconazole, and amphotericin B for severe cases.

Mycetoma

Mycetoma is a chronic infection of the skin and subcutaneous tissue characterized by local edema, sinus tract formation, and the presence of grains—hard concretions representing colonies of the etiologic agent. It occurs worldwide, but most commonly in tropical and subtropical regions. Mycetomas can be divided into eumycetomas, caused by fungi, and actinomycetomas, caused by actinomycetes. The most common agent in Central and South America is the bacteria *Nocardia brasiliensis*, which is

found in soil.[26] This agent is rarely found in the United States but when it does occur, the fungus *Pseudallescheria boydii* is the most commonly isolated agent.

Male rural workers ages 20 to 40 are most frequently affected. After trauma, a slow-growing, painless nodule develops, which may discharge purulent material and grains. Neighboring lesions may interconnect with each other, giving rise to the sinus tracts that are characteristic of the disease.

Diagnosis can be established based on clinical findings; additional examinations may include visualization of grains or filaments in discharge or biopsy and tissue culture. On ultrasonographic evaluation, the mycetoma grains, capsules, and the resulting inflammatory granulomas have characteristic appearances.[27] Treatment is difficult. Surgical excision, commonly in combination with systemic therapy, may be effective. Sulfonamides, tetracycline, aminoglycosides, rifampin, ciprofloxacin, amoxicillin, clavulanate, and oral azoles may be used depending on sensitivities of the etiologic agent. Recent preliminary evidence suggests that linezolid, imipenem, and the newer triazoles voriconazole and posaconazole also may be efficacious.[28]

Vibrio Vulnificus Infection

Vibrio vulnificus, a bacterium, is found widely in raw shellfish in Atlantic Coast waters.[29] It produces extracellular proteolytic and elastolytic enzymes and collagenases that favor tissue invasiveness. Wound infection with *V. vulnificus* occurs when contaminated seawater enters the body through a break in the epidermal barrier, commonly during fishing or water sport activities. Breaks in the skin relating to opening oysters is a common method of inoculation. Pustular lesions, lymphangitis, lymphadenitis, and cellulitis may ensue; in some cases, rapid progression to myositis and skin necrosis follows. Treatment of *V. vulnificus* wound infections consists of antibiotics, such as the combination of minocycline or doxycycline and ceftazidime, and wound care.[30]

Primary septicemia from *V. vulnificus* occurs 24 to 48 hours after the ingestion of raw oysters, especially in patients with hepatic cirrhosis, diabetes, renal failure, or immunosuppression. Clinically, fever and hypotension may be present, along with the development of bullous cellulitis and necrotic skin ulcers.

Necrotizing Fasciitis

Necrotizing fasciitis (NF) is an uncommon but life-threatening soft tissue infection characterized by rapidly spreading inflammation and necrosis of the skin, subcutaneous fat, and fascia[31] (Fig. 20-5). Mortality rates have exceeded 70% in the past,[32] although recent data have shown a dramatic decrease, with mortality rates below 10%.[33] Rapid, early intervention, including surgical debridement and antimicrobial therapy, is imperative to reduce morbidity and mortality.

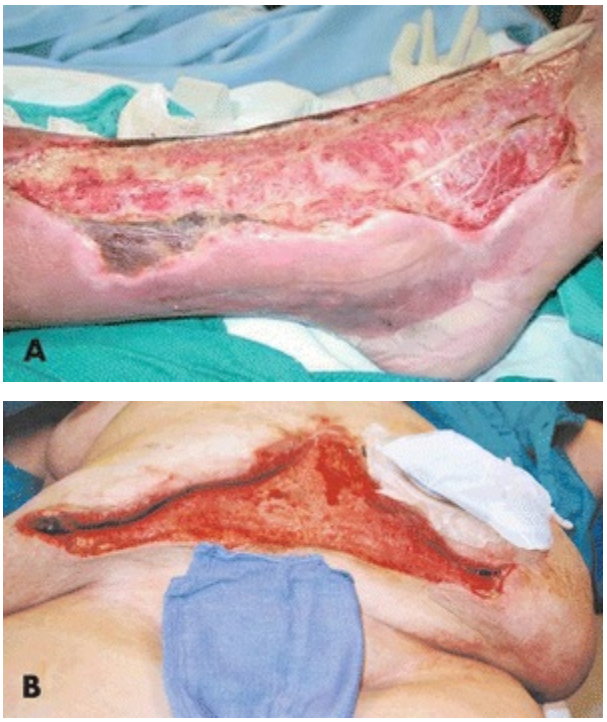


Figure 20-5. (A) Necrotizing fasciitis of the foot. **(B)** Necrotizing fasciitis of the abdomen.

The incidence of NF has been reported to be 0.40 cases per 100,000 population.[33] Although it is rare, certain conditions can predispose patients to developing the disease, including immunocompromised states such as diabetes mellitus (the most common), acquired immunodeficiency syndrome (AIDS), malignancy, obesity, peripheral vascular disease,[34] and trauma, such as burns, lacerations, or minor trauma (Table 20-8).

Table 20-8 Conditions Leading to Necrotizing Fasciitis

The conditions listed below place patients at risk for NF.[29,33,34]

- Age 50 and older
- Alcoholism
- Anogenital infection: anorectal, perianal, scrotal, ischiorectal, periurethral abscess
- Atherosclerosis: coronary artery disease, peripheral vascular disease
- Chronic obstructive pulmonary disease
- Cirrhosis or chronic liver disease
- Compromised skin integrity: psoriasis, herpes zoster, chronic leg ulcers
- Diabetes mellitus
- Hypertension
- Immunosuppression: HIV, corticosteroid therapy
- Intravenous drug use
- Malignancy
- Malnutrition
- Obesity
- Renal failure
- Smoking
- Surgery
- Trauma: needle puncture, insect bites, fish-fin injury, burns, lacerations, surgical wounds

NF spreads quickly along the fascia due to the relatively poor blood supply to this area. Thus, initially, overlying tissue may appear healthy even though the infection is rapidly spreading. NF is categorized as type 1, type 2, or type 3 depending on which organisms are cultured. Type 1 NF is caused by a polymicrobial infection from aerobic and anaerobic bacteria such as *Clostridium* and *Bacteroides* species. Type 2 NF consists of group A *Streptococcus* (*Streptococcus pyogenes*) with or without a coexisting staphylococcal infection. Type 3 NF is caused by a *Vibrio* infection from a puncture wound due to fish or marine insects.

NF can affect any area of the body, but it most commonly occurs on the extremities. Involvement of the genitalia is referred to as Fournier's gangrene and usually results from a polymicrobial infection.[35] There is a higher mortality rate when the head, neck, chest, and abdomen are involved because those areas tend to be refractory and, as such, are more difficult to treat.

Early on, patients generally present with a clinical picture similar to cellulitis. As with less aggressive cases of cellulitis, redness and edema can be seen at the site, with a spreading, diffuse inflammatory reaction that blends into the surrounding tissue. The overlying skin is shiny and tense

without any clear lines of demarcation. However, patient complaints of severe pain are usually out of proportion to the clinical lesion. This characteristic clue of NF may be the only hint of a deeper, more aggressive infection. It is critical to be alert for this sign because the earlier the diagnosis is made, the earlier treatment can be instituted and the better the chance of survival will be. Treatment involves immediate surgical intervention to remove infected and necrotic tissue in addition to systemic antibiotic therapy.[36]

Over time, frank cutaneous gangrene may extend beyond the skin and into the subcutaneous fat and fascial planes below. Separation of the necrotic tissue along the fascial planes with suppuration may occur. Myonecrosis develops in the underlying muscle. Lymphadenitis, lymphangitis, crepitation, and venous thrombosis are seen less often.

Metastatic abscesses have been reported in the liver, lung, spleen, brain, and pericardium, but these are rare. In addition to cutaneous manifestations of NF, there are systemic findings as well as those associated with progression of disease. Patients usually will appear toxic with high fever, chills, and constitutional symptoms. In fulminant cases, multiorgan system failure occurs.

Vasculopathies

A heterogeneous group of disorders is classified under this category. Vasculopathy is characterized by occlusion of small vessels within the skin due to thrombi or emboli, which leads to tissue hypoxia and the clinical manifestations of purpura, livedo reticularis, and painful ulcers. Cryofibrinogenemia, monoclonal cryoglobulinemia, and antiphospholipid antibody syndrome (APS) are among the causes of vasculopathies that commonly present as atypical skin ulcerations of the lower extremities.

Cryofibrinogenemia

Cryofibrinogenemia occurs as a primary (idiopathic) disorder or in association with underlying diseases, such as infectious processes, malignancy, or collagen, vascular, or thromboembolic disease. The clinical presentation is painful cutaneous ulcerations located on the leg and foot; these lesions are usually unresponsive to treatment (Fig. 20-6). Other cutaneous findings include livedo reticularis (a netlike erythema), purpura, ecchymoses, and gangrene. The pathogenesis of the lesions is related to the in vivo occlusion of small blood vessels initiated in the distal

extremities by the abnormal precipitate. This hypothesis is corroborated by the pathology findings of cryofibrinogen, consisting of thrombi within superficial dermal vessels due in part to protein deposition. Cryofibrinogen is a circulating complex of fibrin, fibrinogen, and fibronectin along with albumin, cold-insoluble globulin, and factor VIII. The complex is soluble at 98.6°F (37°C) but forms a cryoprecipitate at 39.2°F (4°C).[37] Additionally, this complex can be made to clot with thrombin. The mechanism by which cryofibrinogen is produced is not well understood.

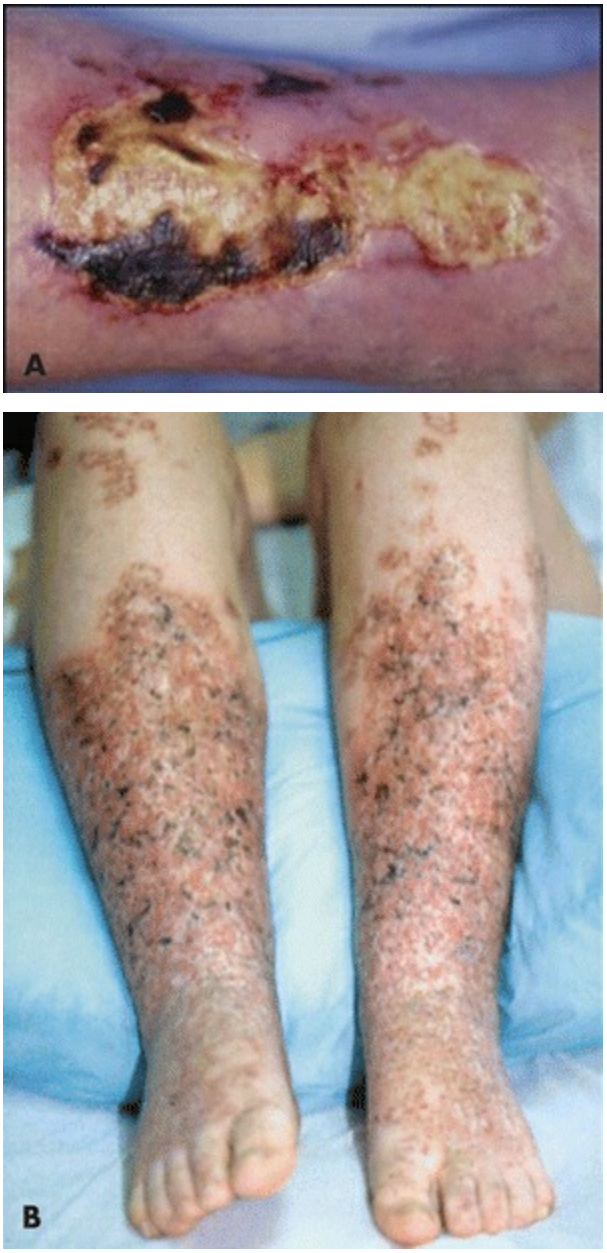


Figure 20-6. (A) Cryofibrinogenemia. **(B)** This patient has painful punctate ulcers on the feet and legs secondary to cryofibrinogenemia.

Treatment is symptomatic (avoiding cold, smoking cessation) or, in secondary disease, directed to the underlying cause.[38] Agents that lyse fibrin thrombi are helpful.

Stanozolol, streptokinase, and streptodornase have been used with success.

Cryoglobulinemia

Cryoglobulinemia occurs when deposits of cryoglobulins lead to the formulation of thrombi in medium and small vessel walls.[39] Three types of cryoglobulinemia have been identified; type I or monoclonal cryoglobulinemia may be seen in patients with malignant diseases, such as multiple myeloma, or benign lymphoproliferative conditions, such as Waldenström's macroglobulinemia. This classically leads to thrombotic phenomena but can clinically resemble vasculitis. Type II or mixed cryoglobulinemia combines polyclonal and a monoclonal immunoglobulin; this type of cryoglobulinemia is seen less often in association with malignancies and more frequently with infectious or inflammatory diseases. Type III is comprised of only polyclonal immunoglobulin and is most commonly associated with hepatitis C infection. Both type II and III cryoglobulinemia cause vasculitis, which can lead to skin ulcers.[40] Other skin manifestations include acral cyanosis, Raynaud's phenomenon, livedo reticularis, altered pigmentation of involved skin, and palpable purpura, which may progress to blistering and frank ulceration. Some patients may have systemic manifestations, such as arthritis, peripheral neuropathy, and glomerulonephritis. Diagnosis is based on skin biopsies, which show either vasculopathy or vasculitis, and subsequent detection of cryoprecipitate and analysis of cryoglobulins by immunoelectrophoresis.

Practice PointPractice Point

Evaluate the patient with vasculitis for type II or III cryoglobulinemia; these conditions can cause vasculopathy.

Treatment should be directed at the underlying cause in cryoglobulinemia. For example, interferon-a, ribavirin, and newer direct-acting antivirals are used to treat hepatitis C and may result in resolution of

the associated cryoglobulinemia.[41] Corticosteroids alone or in combination with immunosuppressive drugs (cyclophosphamide or mycophenolate mofetil) have been used to induce remission in patients with cryoglobulinemia. Plasmapheresis and rituximab can be used as well to treat severe cases of cryoglobulinemic vasculitis.[42]

Antiphospholipid Antibody Syndrome

APS is characterized by elevated titers of different antiphospholipid antibodies in association with venous or arterial thrombosis, recurrent fetal loss, and often thrombocytopenia. Antiphospholipid antibodies include lupus anticoagulant, anticardiolipin antibodies, and anti-β_2-glycoprotein-1 antibodies. All or any of the antibodies are part of the syndrome.[43,44] APS may present as primary disease or may be secondary to an underlying autoimmune disease such as systemic lupus erythematosus (seen in about 50% of cases). Elevated antiphospholipid antibody titers also have been associated with malignancy and infectious states. The exact pathogenic mechanism of APS remains unclear, but it is thought that once the antiphospholipid antibodies form, they act directly upon coagulation pathways and increase the susceptibility to thromboses. In addition, they may increase vascular tone.[45]

The clinical hallmark of this disease is the presence of livedo reticularis. Arterial and venous thrombosis may elicit a variety of skin lesions, including ulcerations (most commonly), superficial thrombophlebitis, and cutaneous infarcts. Any organ system can be affected. Placental vessel thrombosis and ischemia can result in miscarriage precipitated by placental insufficiency. A proposed mechanism responsible for this event is a disruption of annexin A5 (a cell surface protein with anticoagulant properties) binding of phospholipids on the trophoblast cells by antiphospholipid antibodies, thus promoting a procoagulant state that leads to thrombosis at the maternal–fetal interface followed by damage to the placenta and eventual fetal loss.[46] Treatment includes the use of aspirin, warfarin, and prednisone, but response is not uniform.

Metabolic Disorders

Metabolic diseases are uncommon causes of chronic wounds. One condition, called calciphylaxis, is commonly seen in a subset of patients undergoing long-term hemodialysis who develop secondary

hyperparathyroidism.[47] This leads to deposition of calcium within soft tissue and arteries and, eventually, tissue death.

Calciphylaxis

Calciphylaxis is a rare, often fatal condition that is characterized clinically by progressive cutaneous necrosis, which frequently occurs in patients with end-stage renal disease. Many eliciting factors have been suggested, but the most common linking phenomenon is the development of secondary hyperparathyroidism.[48] Secondary hyperparathyroidism causes elevated calcium–phosphate product and the development of vascular, cutaneous, and subcutaneous calcification that, in turn, leads to tissue death. Interestingly, most patients with severe hyperparathyroidism do not have skin necrosis, thus, other less well-understood mechanism are thought to play a role in the pathogenesis of calciphylaxis. Calciphylaxis is seen in approximately 1% to 4% of patients with ESRD on dialysis.[49,50] The prognosis for patients who develop calciphylaxis is grim, with an estimated 1-year survival rate of approximately 46%.[51,52] In addition to skin involvement, the pathophysiologic process may also occur within internal organs; this, along with sepsis from infected skin wounds, is a major cause of morbidity and mortality in patients with this condition.

The cutaneous manifestations begin as red or violaceous mottled plaques in a livedo reticularis-like pattern. This signifies a vascular pattern, and these early ischemic lesions often progress to gangrenous, ill-defined, black plaques (Fig. 20-7A). With time, the plaques ulcerate and become tender; indurated ulcers can lead to autoamputation. The ulcers of calciphylaxis are usually bilateral and symmetric and may extend deep into muscle. Vesicles frequently appear at the periphery of the ulcers (Fig. 20-7B).

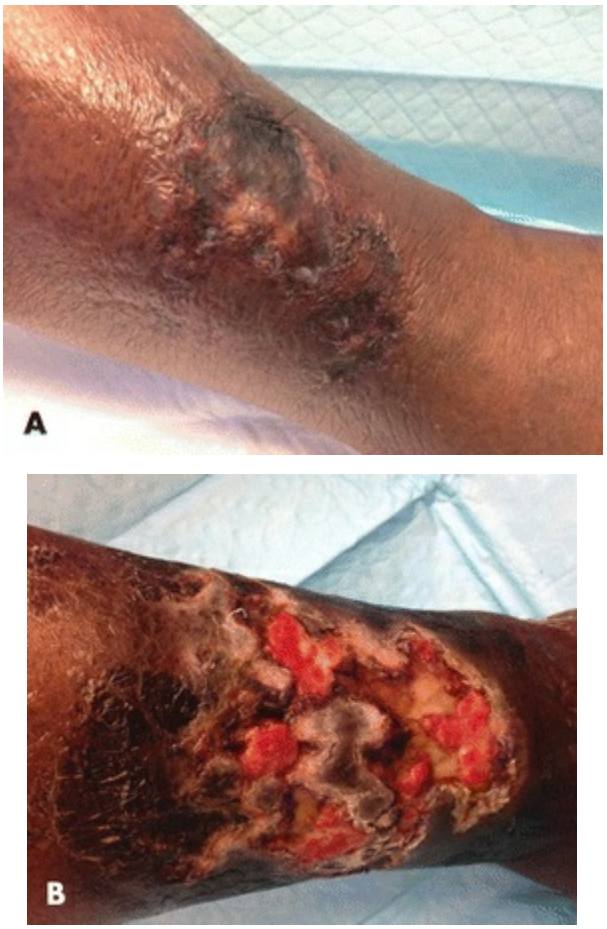


Figure 20-7. (A) Defined, black plaques of calciphylaxis. **(B)** Calciphylaxis lesion that has ulcerated.

The term *calciphylaxis* is considered a misnomer that originated from experiments in rats that resulted in interstitial calcification.[53] "Calciphylaxis" originally was coined by Selye in 1962 [53] after he produced ectopic calcifications in experimental animals using variety of sensitizing agents, including parathyroid hormone, vitamin D, and a diet high in calcium and phosphorus. Calciphylaxis should be distinguished from calcinosis cutis, which may be associated with significant morbidity but typically not mortality. Calciphylaxis has been given a number of other names, including uremic gangrene syndrome, metastatic calcinosis, azotemic calcific arteriopathy, and calcific uremic arteriopathy. Some authors prefer the term *calcific uremic arteriopathy*[54] because it reflects the histologic findings of calcium deposits in the walls of the small- and medium-sized vessels of lesions. The important distinction is that calcific uremic arteriopathy involves small vessel calcifications with intimal hypertrophy and small vessel thrombosis (Fig. 20-8).

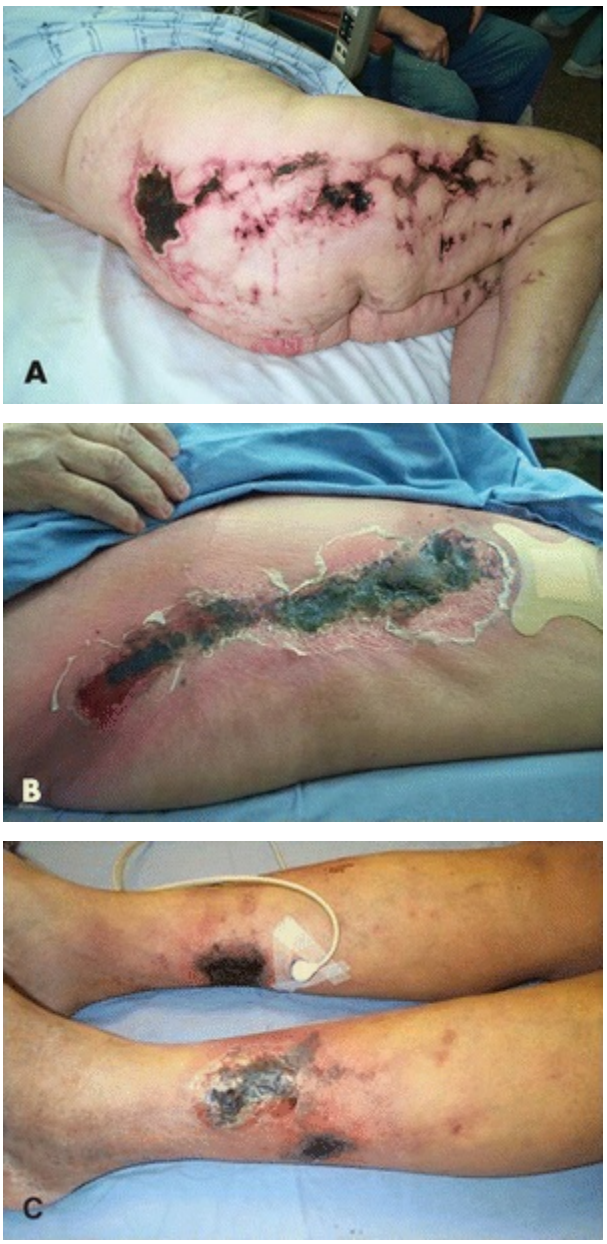


Figure 20-8 Calcific uremic arteriopathy (CUA). **(A)** CUA with livedo reticularis pattern. **(B)** CUA of the thigh. **(C)** CUA of bilateral extremities. (Courtesy of D. Beless, MD.)

The pathogenesis of calciphylaxis is still poorly understood. Recent studies have shown increases in the expression of bone-specific proteins, including collagen I, bone morphogenic protein 2 and 3, matrix Gla protein, osteopontin, osteonectin, and osteocalcin in calcified arteries. These proteins, together with elevated calcium and inorganic phosphate levels, may induce calcification or osteoblastic differentiation in pluripotent vascular smooth muscle cells.[47,48,54,55]

Diagnosis can usually be made based on clinical findings. Although

vascular processes may present similarly, laboratory evaluation for the presence of an elevated calcium, phosphate, or calcium × phosphate product confirms calciphylaxis. An elevated intact parathyroid hormone level along with radiographic evidence and consistent histology also help confirm a diagnosis. Histology showing calcification of the intima and media of small- and medium-sized vessels in the dermis and subcutaneous tissue are characteristic of calciphylaxis. Radiographic findings include pipe stem calcifications resulting from the calcium deposits outlining the vessels in these patients.

As secondary infections may have tragic sequelae, treatment of infected ulcers is critical. Swab and tissue cultures from the wound may aid in guiding antibiotic therapy.

The treatments used for patients with calciphylaxis can be divided into either medical or surgical therapies[47,48,54,55] and are often *used in tandem* (Table 20-9).

Table 20-9 Calciphylaxis Treatment Options

Medical Treatment Options
• Phosphate binders
• Decreased calcium in dialysate
• Antibiotics
• Low-phosphate diet
• Cinacalcet
• Bisphosphonates
• Sodium thiosulfate
• Avoidance of challenging agents
• Avoidance of systemic steroids
• Hyperbaric oxygen
• Anticoagulation
• Cyclosporine
• Stanozolol
Surgical Treatment Options
• Parathyroidectomy
• Wound care and debridement
• Amputation
• Renal transplantation
• Skin grafting using either autologous or tissue-engineered skin

Malignancies

Malignancies may present either as wounds or developing from wounds.

Nonmelanoma skin cancer, lymphomas, and sarcomas may ulcerate as they outgrow their blood supply (Fig. 20-9). Alternatively, chronic wounds may develop into a malignancy, most commonly squamous cell carcinoma. This phenomenon is termed *Marjolin's ulcer* after the author who first described cells from an edge of a chronic wound that had undergone malignant change —an occurrence that can be seen in up to 2% of chronic wounds[56] (Fig. 20-10). A similar phenomenon may also occur in scars, burn wounds, sinus tracts, chronic osteomyelitis, and even vaccination sites.

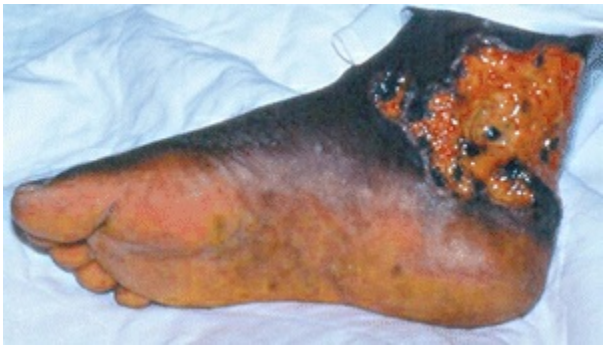


Figure 20-9 Malignancies. Shown here is a right medial leg ulcer in a venous distribution secondary to T-cell lymphoma.

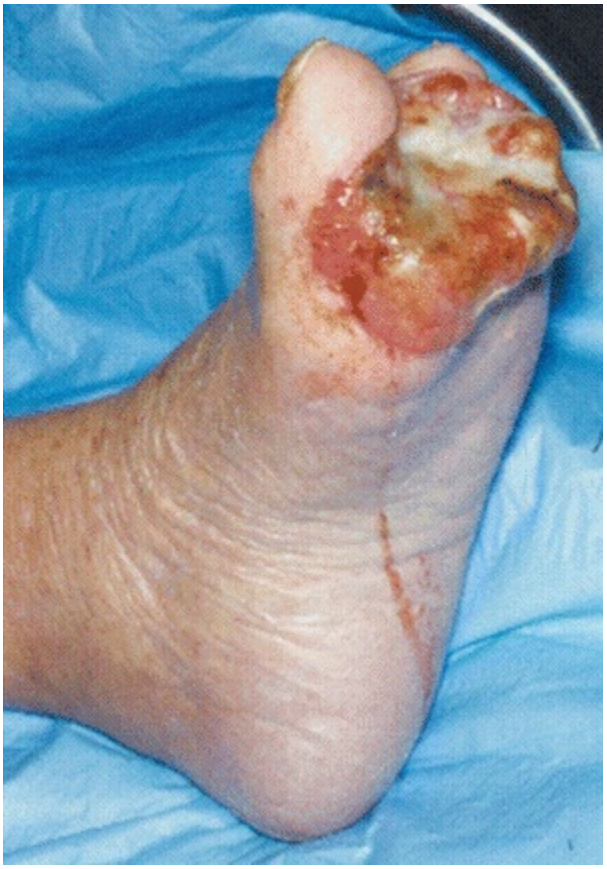


Figure 20-10 Marjolin's ulcer. Shown here is a chronic wound that developed malignant changes (squamous cell carcinoma).

The precise mechanism of malignant degeneration in chronic wounds is not known, although several theories have been proposed.[57,58] One such theory is that the weakened epithelium and the decreased vascularity of the wound make it susceptible to carcinogens. Another postulated idea is that possible toxins released from the wound tissue lead to mutations and eventual carcinogenesis. In addition to squamous cell carcinoma, basal cell carcinomas and other neoplasms, such as Kaposi's sarcoma and lymphoma, have also been found in chronic wounds.

Early identification of malignancy, typically via biopsy of suspected lesions, is critical. Treatment of biopsy-confirmed neoplasia includes excision with margins (usually at least 2 cm), possibly adjuvant therapy with radiation, topical 5-fluorouracil, methotrexate, L-phenylalanine mustard, and even lymph node dissection; amputation of the affected limb may be necessary in some cases.[57] Mohs surgery to ensure complete removal of the primary lesion has been used successfully to treat malignancy arising from chronic osteomyelitis.[59]

External Causes

External causes of atypical wounds include spider bites, chemical injury, chronic radiation exposure, trauma, and factitial ulcers. A thorough patient history is the most valuable tool in determining the etiologic agent in ulcers caused by external factors.

Spider Bites

At least 50 spider species in the United States have been implicated in causing significant medical conditions; however, the *Loxosceles* (brown recluse or violin spider) and the *Latrodectus* (black widow) species are the most well known to cause skin necrosis and ulcers in the Americas.

Loxoscelism

The bite of *Loxosceles reclusa* is usually painless and often goes unnoticed; however, some patients will experience progression to more significant wounds. In these patients, enlargement of the bite site occurs within 2 to 6 hours, with associated pain and general symptoms, such as fever, malaise,

headache, and arthralgia. As the disease progresses, a pustule, blister, or large plaque forms at the bite site. These wounds may present as a deep purple plaque surrounded by a clear halo (vasoconstriction) and surrounding erythema—the so-called red, white, and blue sign (Fig. 20-11). Viscerocutaneous loxoscelism, a systemic illness that can include diarrhea, nausea, vomiting, petechia, urticaria, and disseminated intravascular coagulation, can occur in 0.7% to 27% of patients depending on the geographic region.[60]

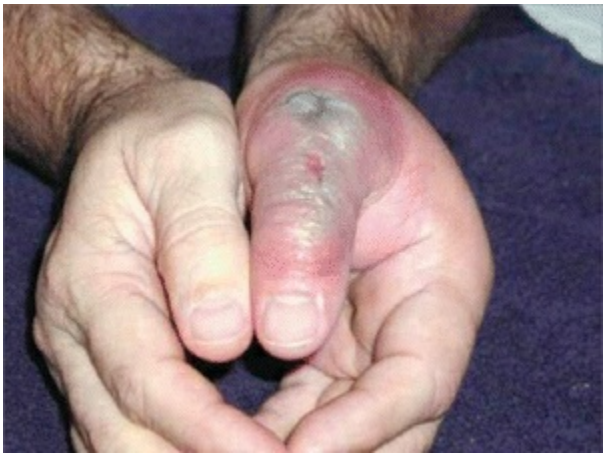


Figure 20-11 Loxoscelism. =Bite from a brown recluse spider (*Loxosceles reclusa*). Note the deep purple plaque surrounded by a clear halo and erythema, the so-called red, white, and blue sign.

With bites occurring in areas of greater fat content, such as the abdomen, buttocks, and thighs, necrosis develops more frequently. When the eschar is shed, an ulcer may result. Healing of the ulcer is generally very slow and may take up to 6 months.

The differential diagnosis includes foreign body reaction, infections, trauma, vasculitis, pyoderma gangrenosum, and methicillin-resistant *Staphylococcus aureus* infection. Treatment consists of cooling the bite site, elevation (if possible), and analgesics. The use of dapsone and corticosteroids is controversial.[45]

Latrodectism

The black widow spider, or *Latrodectus mactans,* is easily recognized by a bright red hourglass marking on the abdomen. The painless bite is followed by severe pain, swelling, and tenderness at the site where the bite occurred. Systemic symptoms may follow, including headaches and abdominal pain, but they subside in 1 to 3 days. Treatment includes local ice, analgesics, and

the administration of specific antivenin if severe. Black widow bites are rarely fatal; death may occur in children, those with comorbidities, or elderly people.

Chemical Burns

A variety of chemical products are capable of producing skin wounds.[61] Cutaneous injury caused by caustic chemicals progresses continually after the initial exposure and, if not properly cared for, may produce painful ulcers that are difficult to heal. The lesions caused by alkalis are usually more severe than those caused by acids; however, the severity of the burn is determined by the mode of action and concentration of the chemical as well as the duration of contact before treatment is initiated.[61] Prolonged irrigation with water for 30 minutes (for most chemicals) or longer is the most important initial treatment, followed by standard burn care.

Certain chemicals possess unique properties that require special additional therapy, such as hydrofluoric acid (application of 25% magnesium sulfate), chromic acid (excision of the affected area), and phenol (application of polyethylene glycol mixed with alcohol 2:1).[61]

Radiation Dermatitis

After exposure to ionizing radiation exceeding 10 Gy, local skin reactions characterized by mild erythema, edema, and pruritus may occur. This acute radiation dermatitis usually begins 2 to 7 days after exposure, peaks within 2 weeks, and gradually subsides. With exposure to higher doses, intense erythema with vesiculation, erosion, and superficial ulceration may ensue. Postinflammatory pigmentary abnormalities, telangiectasia, and atrophy are common.[62]

Hyperbaric oxygen is a possible treatment option for ulcers caused by radiation.

Factitial Dermatitis

The term *factitial dermatitis* denotes a self-imposed injury. The clinical appearance of these ulcers is usually particular, with sharp or linear edges in an area of easy access such as the extremities, abdomen, and anterior chest (Fig. 20-12).

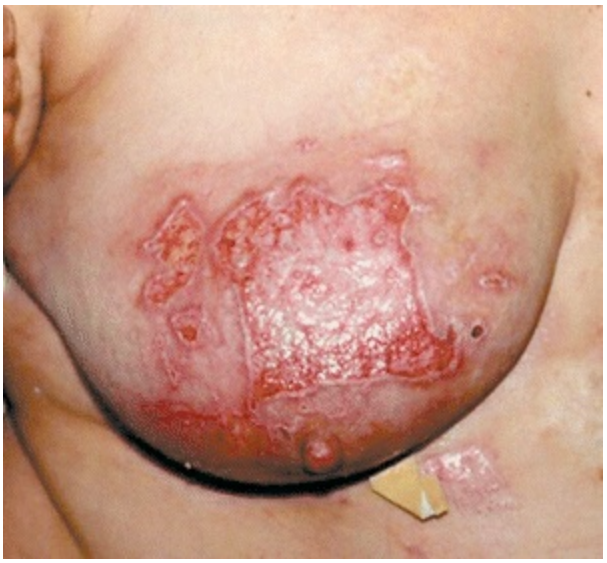


Figure 20-12 Factitial dermatitis. This photograph shows an angulated factitial ulcer on the breast. The term *factitial* denotes a self-imposed injury.

The care includes evaluation and treatment of underlying psychological diseases and limitation of accessibility to the wound, such as placing a dressing or cast over the wound.

Drug-Induced Causes

Coumadin Necrosis

Coumadin (warfarin)-induced skin necrosis is a rare complication of anticoagulant treatment. It is estimated that 1 in every 1,000 to 10,000 patients treated with this agent experiences this complication. Coumadin skin necrosis occurs almost exclusively between the 3rd and 10th day after beginning anticoagulation therapy in association with the administration of a large initial dose of the drug. Receiving the initial dose of Coumadin without concomitant heparin also increases the risk of necrosis. Although the precise nature of the disease is still unknown, advances in knowledge about protein C, protein S, and antithrombin III anticoagulant pathways have led to a better understanding of the mechanisms involved in pathogenesis.[63]

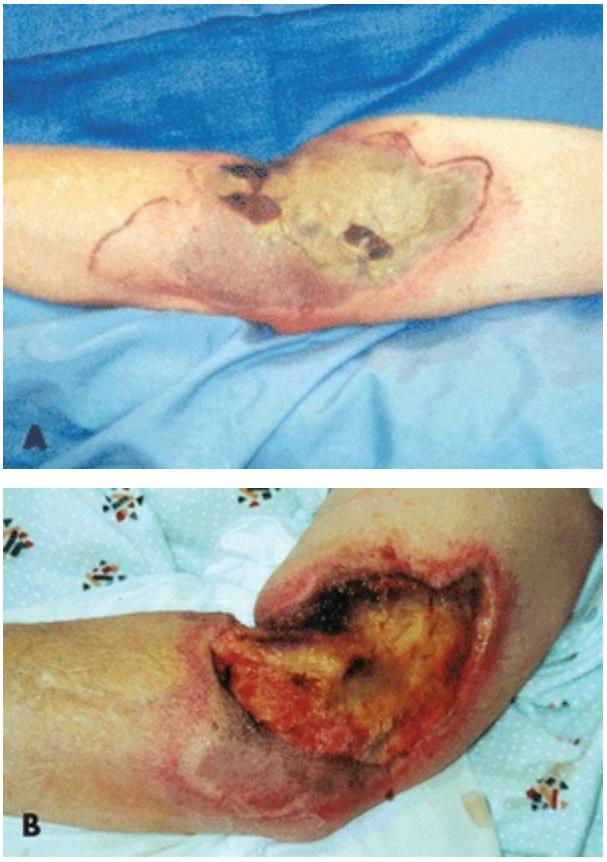
Postpartum women have a unique risk due to reduced levels of free protein S during the antepartum and immediate postpartum periods.[64] Manifestations range from ecchymoses and purpura to hemorrhagic necrosis; maculopapular, vesicular urticarial eruptions; and purple toes in all affected patients.

Wounds are painful and usually evolve into full-thickness skin necrosis within a few days. Differentiating between warfarin/coumadin-induced skin necrosis and NF, gangrene, and other causes of skin necrosis may be difficult.[63]

Cessation of warfarin administration is the first step in treatment, with heparin used instead if anticoagulation is needed. Local wound care can be conservative but may include debridement and grafting, depending on the extensiveness of the wound. Previously uncomplicated courses of warfarin therapy do not obviate the possibility of skin necrosis with future warfarin administration. Initiation of low-dose warfarin with heparin can reduce the likelihood of this disorder.

Extravasation

Solutions of calcium, potassium, bicarbonate, hypertonic dextrose, cardiac drugs, chemotherapeutic drugs, cytotoxic drugs, and antibiotics can lead to extravasation injury. Tissue loss can evolve into extensive wounds. Local wound care with debridement and eventual skin grafting is usually required for extensive skin and tissue loss (Fig. 20-13).



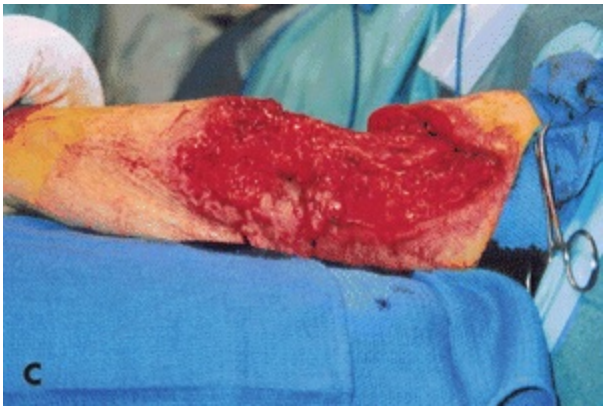


Figure 20-13 Extravasation. **(A)** Extravasation can cause tissue loss that may evolve into extensive wounds, as shown in this I.V. site 24 hours after infiltration of calcium chloride. **(B)** The same site 48 hours after wound debridement. **(C)** The same site after surgical debridement down to viable tissue.

Wounds with less tissue loss can be managed conservatively with the same outcome.[65] Because many extravasation injuries occur on the hands, scar management and return of function remain a problem. Proper administration through the correct needle size (small), vein size (large), and dilution of the medication is best. Infusion should be performed as slowly as possible to allow adequate dilution into the blood. Any complaints of pain during infusion warrant immediate cessation of the infusion, assessment of the intravenous site, and adherence to treatment protocols for extravasation as deemed necessary. Calcium gluconate is less likely to extravasate and should be used instead of calcium chloride for the management of low serum levels, especially those levels that are not life threatening. Because many of these cases may elicit external review and sometimes legal review, documentation is crucial to determine what care was given prior to and after the medication was given. Nurses need to be especially vigilant when administering medications prone to extravasation.

Patient TeachingPatient Teaching

Patients don't always give a complete history or believe that certain information is relevant to a clinician pertaining to their wound. Because certain diseases or treatments travel to certain locations, or even contact with certain insects and animals can be associated with some atypical wounds, telling a clinician about these factors can be important in determining the correct etiology

of the wound. Instruct patients to always tell the clinician if they:

- have inflammatory bowel disease, as this is often an underlying etiology in pyoderma gangrenosum
- lived in a developing country, as there could be a risk for Buruli ulcers
- visited or lived in a tropical region of Central or South America, as deep fungal infections, systemic mycoses, or exposure to soil (such as being on a farm in a subtropical location, especially in South America) could lead to chromoblastomycosis or paracoccidioidomycosis
- have eaten raw oysters or been fishing or participated in water activities in Atlantic Coast waters, as this can lead to *V. vulnificus* infection
- have a history of chronic renal failure and/or are on hemodialysis, as calcific uremic arteriopathy ulcers (calciphylaxis) can occur
- have an impaired immune system, such as from HIV or alcoholism, as sporotrichosis may be present
- are taking certain medications, such as warfarin (Coumadin), as this can cause necrosis.

Summary

Treating the underlying cause, when possible, is the initial step in caring for patients with atypical wounds. Anti-infective agents may be used for infectious ulcers, malignancies may require surgical removal, and anti-inflammatory agents may be used for inflammatory ulcers. In addition, the use of a moist healing environment, compression dressings (in the absence of arterial insufficiency) for leg lesions, off-loading areas at risk for prolonged pressure, and maximizing patients' nutritional status are essential.

Despite these measures, healing is often slow in patients with atypical wounds. Prolonged healing leads to increased morbidity and decreased quality of life as well as an increase in direct and indirect costs of care. Adjunctive therapies are often used, aimed at both increasing the number of patients who will heal (effectiveness of therapy) and the speed at which they heal (cost-effectiveness of therapy).

PATIENT SCENARIO

Clinical Data

Mrs. VS is a 43-year-old patient who presents to your clinic for the first time for treatment of a painful leg ulcer. She has had the ulcer for approximately 3 months. She states that it started as a small pustule on the lateral aspect of her lower left leg that ulcerated and slowly became larger. Two months ago, she went to a wound clinic close to her house. The doctor told her the wound appeared infected and needed to be debrided; she would also need antibiotics. Within the week after debridement, the wound almost doubled in size. Upon further questioning, you find out that Ms. VS has a history of ulcerative colitis. On physical exam, the ulcer is approximately 6 cm in diameter with irregular, violaceous, undermined borders. The base of the ulcer is purulent with ample exudate present.

Case Discussion

Several aspects of the history and physical exam should clue you in to the correct diagnosis of pyoderma gangrenosum. Pyoderma gangrenosum classically starts as a pustule that ulcerates and enlarges, although not all ulcers start this way. The appearance of the wound is also classic, with violaceous, undermined borders with a purulent base.

The patient's past medical history is very important in this case. Nearly 50% of patients with pyoderma gangrenosum have an underlying disease, the most common of which is inflammatory bowel disease. Other associated diseases include hematologic malignancies and inflammatory arthritis.

Ms. VS clearly demonstrates pathergy, the development of ulcers in an area of trauma or worsening of the ulcer with trauma; in this case, it was evident by the severe worsening of the ulcer following debridement. Other patients may report the development of ulcers following seemingly trivial trauma, such as hitting one's leg against a coffee table.

Treatment consisted of cleansing the wound with normal saline and dressing the wound with an absorbent dressing. The patient did not have any history of or physical findings or arterial insufficiency, so her

leg was wrapped in a four-layer compression bandage to minimize any edema and accelerate healing. High-dose systemic steroids were started, and the ulcer gradually resolved over the next 6 weeks, at which point the steroids were tapered.

Show What You Know

1. Which of the following is the most important reason to recognize a wound as atypical?

A. The wound may be contagious.
B. Treatment varies based on etiology.
C. Standard wound healing therapies don't apply.
D. To bill correctly.

2. Which of the following may be performed on tissue biopsies of wounds?

A. Histology
B. Culture
C. Immunofluorescence
D. All of the above

3. Which of the following should not typically be debrided?

A. Diabetic foot ulcer
B. Ulcers due to infectious causes
C. Pyoderma gangrenosum ulcers
D. Ulcers due to vasculitis

4. Sporotrichosis is a fungal infection caused by:

A. *S. schenckii.*
B. *M. ulcerans.*
C. *F. pedrosoi.*
D. *M. marinum.*

5. Cryofibrinogenemia is classified as a:

A. *mycobacterium.*
B. pyoderma gangrenosum.
C. vasculopathy.
D. metabolic disease.

6. Which type of wound is a rare, often fatal condition characterized by progressive cutaneous necrosis that occurs in patients with end-stage renal disease?

A. Calciphylaxis
B. Vasculopathy
C. Radiation dermatitis
D. Chemical burn

7. Factitial dermatitis is a:

A. rare condition.
B. self-imposed injury.
C. red rash.
D. dry, scaly scab.

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Wounds in Special Populations

21

This chapter addresses three special populations, spinal cord injury (SCI) patients, patients with HIV/AIDS, and bariatric patients, that are each divided into its own section with objectives, references, and show what you know questions. The following is a short summary of each of these three sections.

The first section of this chapter is on the SCI population. The patient with an SCI faces a lifelong risk for the development of pressure ulcers. Although mostly preventable, pressure ulcers are a deterrent to achieving rehabilitation goals, may contribute to a loss of independence, and interfere with the pursuit of educational, vocational, and leisure activities after an SCI. It's now possible to identify patients with an SCI who are at highest risk for pressure ulcers so that effective prevention strategies can be incorporated into their lifestyles.

The second part of this chapter addresses the HIV/AIDS population. In patients with human immunodeficiency virus (HIV) and acquired immune deficiency syndrome (AIDS), skin disorders are common. Accurate identification of skin lesions is critical so appropriate treatment can be implemented. Consultation with an HIV or AIDS clinician is helpful in the comprehensive care of these patients.

Finally, the third portion of this chapter is on the bariatric population. The bariatric population has increased dramatically. Across care settings, healthcare professionals will be caring for persons with higher BMI. Therefore, proactive planning, preparation, proper equipment, interdisciplinary teamwork, and sensitivity are important hallmarks in providing appropriate care to bariatric patients.

SPINAL CORD INJURY

POPULATION

Susan L. Garber, MA, OTR, FAOTA, FACRM

Objectives

After completing this chapter, you'll be able to:

- describe successful strategies for reducing the incidence of pressure ulcers in the patient with an SCI
- identify risk factors of pressure ulcers in the patient with an SCI
- discuss the major health complications of an SCI

Spinal Cord Injury Incidence and Prevalence

Approximately 273,000 persons are living with SCI in the United States today, of whom 17% (42,000) are veterans.[1,2] Twelve thousand new cases of SCI are reported every year.[2] The most recent data on the demographics of SCI derive from the National Spinal cord Injury Statistical Center (NSCISC).[2] The NSCISC provides statistics on five major categories of etiology:

- Motor vehicle crashes 36.5%
- Falls 28.5%
- Acts of violence 14.3%
- Recreational sporting activities 9.2%
- Causes that do not fit into any of these categories 11.4%[2]

SCI occurs most frequently in males ages 16 to 30 (55%), and males make up more than 80% of the total population of persons with SCI.[2] The average age at injury today is 42.6 years (up from 29 years at the end of the 1970s). Changes in race have been observed since 2010. Currently, 67% are Caucasian, 24.4% are African American, 7.9% are Hispanic, 2.1% are Asian, and.8% are Native American. This trend may be due, in part, to the trends in the general population of the United States. Almost half of the SCI population had completed high school at the time of injury. Given the young age at onset of injury, more than half were single. More than half were

employed at the time of injury; however, almost 12% were unemployed 1 year following SCI.[2] By 20 years postinjury, almost 35% are employed.

People with SCI are at risk for a number of complications. For instance, pulmonary complications are the most common cause of death during both the acute and chronic phases after SCI.[3] Other potential complications arising soon after injury—some of which may become lifelong problems—include pressure ulcers, urinary tract infections (UTIs), osteoporosis, fractures, and heterotopic ossification.

Pressure ulcers are among the most common long-term secondary medical complications observed at annual follow-up visits.[4] As such, they are a serious, costly, and potentially life-threatening complication of SCI. Clinical observations and research studies have confirmed staggering costs and human suffering, including a profoundly negative impact on the person's general physical health, socialization, financial status, and body image, compounded by a loss of independence and control.[5]

Reliable and current data on the incidence and prevalence of pressure ulcers in the person with SCI have been difficult to obtain, primarily because there were variations in the data collection methods, limiting standardization of the statistics. These limitations include the use of different classification systems to stage pressure ulcers, the inability to compare varied populations (e.g., acute or chronic SCI) presenting with or developing pressure ulcers, and the use of different methods of obtaining data, such as direct observation or retrospective chart review.[6] However, reported prevalence rates of pressure ulcers for persons with SCI residing in the community have ranged from 17% to 33%.[7]

The number of pressure ulcers increases significantly with duration of injury.[8] For these individuals, the incidence rate increased from 15% at 1 year postinjury to nearly 30% at 20 years postinjury.[9] This may be the result of a combined effect of aging and long-term SCI. Aging with SCI has been shown to negatively impact many health factors including the odds ratio for pressure ulcer development.[10]

Scope of the Problem

The database of the Spinal Cord Injury Model Systems is one of the most reliable resources from which to obtain data that reflect the scope of the problem. The National Institute on Disability and Rehabilitation Research sponsors the Model Systems Program, a federal extramural grant program of

selected research and demonstration sites. Model System sites provide exemplary, state-of-the-art care from the time of injury through acute medical care, comprehensive rehabilitation, and long-term follow-up and health maintenance services.[2] An individual is included in the database only if he or she was admitted to a system facility within 24 hours of trauma. Although the Model System database has included statistics on pressure ulcers in patients with SCI since 1981, these statistics do not appear every year in the Facts and Figures published data set.

An increased prevalence of pressure ulcers is seen in persons with longer SCI durations, especially among those 10 years or more postinjury.[11] Among veterans with SCI, over one-third report having pressure ulcers during the previous year.[12] Pressure ulcers remain the second leading cause of rehospitalization after urinary problems for persons with SCI enrolled in the US SCI Model Systems Database. Pressure ulcers account for 17% of the rehospitalizations in the first year after injury, 23% at 5 years, 29% at 10 years, and 38% at 20 years postinjury.[2]

Thirty-four percent of patients admitted to a Model System facility within 24 hours of an SCI developed at least one pressure ulcer during acute care or rehabilitation, with pressure ulcer prevalence increasing over time following injury.[13] On follow-up, 15% had a pressure ulcer at their first annual examination, 20% at year 5, 23% at year 10, 24% at year 15, and 29% at year 20. These numbers are based on 4,065 patients, 2,971 of whom developed pressure ulcers. In 2005, Chen et al.[11] reported that 27% (910) of 3,361 patients from nine Spinal Cord Injury Model System facilities had one or more episodes stage II or greater between pressure ulcers 1986 and 2002. Other investigators have reported prevalence rates ranging from 17% to 33% in populations of SCI patients residing in the community.[7,14] The occurrence of pressure ulcers is among the most common long-term secondary medical complications identified during annual follow-up visits in individuals with paraplegia (T1–S5, neurological level), who are more likely to be rehospitalized for pressure ulcers.[2,9,15] Risk factors include complete injury (classified as A, B, or C by the American Spinal Injury Association to denote complete vs. incomplete injury), tetraplegia, older age, comorbidities, and violent injury[9] (Table 21-1).

Table 21-1 American Spinal Injury Association (ASIA) Impairment Scale

A—Complete: No motor or sensory function is preserved in the sacral segments S4–S5.
B—Incomplete: Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4–S5.
C—Incomplete: Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade <3.
D—Incomplete: Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade of 3.
E—Normal: Motor and sensory functions are normal.

Recurrence Rate

High rates of pressure ulcer recurrence also have been reported, ranging from 21% to 79% regardless of treatment.[16,17] Epidemiological studies have found that 36% to 50% of all patients with SCI who develop pressure ulcers will develop a recurrence within the first year after initial healing.[7,14,16–18] Niazi et al.[16] reported a recurrence rate of 35% regardless of the type of treatment (medical or surgical) provided. Holmes et al.[19] found that 55% of their sample, most of whom had a history of severe previous ulcers, experienced a recurrence within 2 years after surgical repair. In a 20-year study in Canada (1976 to 1996), Schryvers et al.[20] studied 168 SCI patients admitted 415 times for treatment of 598 severe recurring pressure ulcers. Of these ulcer recurrences, 31% (185) occurred at the same site as the previous ulcer and 21% (125 ulcers) occurred at a different site. Goodman et al.[21] observed a recurrence or new ulcer development rate of 79% within a 1- to 6-year follow-up time frame.

Some investigators found that pressure ulcer history is a more viable measure of pressure ulcer outcome than measures taken at any single point in time over a brief period. Other studies reveal the protective mechanisms against pressure ulcer recurrence. For example, Krause and Broderick[22] reported that 13% of their sample of 633 subjects had recurring pressure ulcers (one or more per year) over a 5-year period. Their findings suggested that lifestyle, exercise, and diet were protective mechanisms against pressure ulcer recurrence.

Pressure ulcer recurrence in the person with an SCI has been associated with gender (male), age (older), ethnicity (African American), unemployment, residence in a nursing home, and previous pressure ulcer surgery.[22,23] Most of the literature describing recurrence following surgery

focuses on surgical techniques.[24–28] Investigators have reported recurrence rates of 11% to 29% in patients with postoperative complications and 6% to 61% in patients without postoperative complications.[24–30] Relander and Palmer[28] recommended that social factors be studied to determine the causes of pressure ulcer recurrence after surgical repair and suggested that patients who don't display the appropriate knowledge regarding pressure ulcer prevention should be counseled before consideration for surgery. Disa et al.[24] reported that high recurrence rates among patients with traumatic paraplegia were associated with substance abuse and the absence of an adequate social support system. They suggested developing more effective educational programs for both patients and caregivers.[24] In other studies, Mandrekas and Mastorakos[29] and Rubayi et al.[30] reported that inadequate patient education with regard to pressure ulcer prevention contributed to the recurrence rates.

Recurrence also is a significant problem for veterans with SCI. Guihan et al.[31] reported that there are four significant predictors of pressure ulcer recurrence in veterans. They are race (being black), more comorbidities indicating a higher burden of illness, Salzburg Pressure Ulcer (PrU) Risk Assessment Scale score (which measures 15 items associated with SCI, cardiovascular and pulmonary disease, albumin and hematocrit, and impaired cognition), and longer sitting time at discharge from the hospital following the treatment of a pressure ulcer.[31]

Financial Concerns

The financial burden of pressure ulcers is undoubtedly immense, although estimates of the cost of preventing and treating pressure ulcers in the patient with an SCI are not readily available. In 1994, Miller and DeLozier[32] reported that the total cost of treating stage II, III, and IV pressure ulcers in hospitals, nursing homes, and home care was approximately $1.335 billion per year. One could extrapolate from these data the financial implications of pressure ulcers for patients with SCI. However, the financial burden does not begin to reflect the personal and social costs experienced by patients and their families. These include loss of independence and self-esteem; time away from work, school, or family; and, ultimately, diminished quality of life. It is well documented that the development of pressure ulcers increases both hospital costs and length of stay for all patients.[33]

Throughout their lifetime, persons with SCI are at risk for the

development of pressure ulcers. Most of the reported costs deal with investigations of various treatment interventions, especially dressings.[34] In 2003, Garber and Rintala[35] reported that of 102 veterans' charts reviewed, a total of 625 visits were made to treat 400 pressure ulcers at a cost of $250 per outpatient visit (approximate costs from 1998[36]; costs are higher now). Stage IV was the most prevalent pressure ulcer seen, and pelvic ulcers accounted for almost two-thirds of the worst ulcers reported. The average number of clinic or home visits for pressure ulcer treatment was more than six per person. More than half of the study sample was admitted to the hospital for pressure ulcer treatment at least once during the study period. The average number of hospital admissions was two; almost 30% of veterans were admitted three times or more.[35]

Two recent reports have examined the direct costs of the treatment of pressure ulcers in persons with SCI. Chan et al.[37] studied the costs of treating chronic pressure ulcers among community-dwelling SCI individuals in Ontario, Canada. The average monthly cost was found to be $4,745 per community-dwelling SCI individual. Costs for hospital admission were found to make up for the largest percentage of the total costs.[37] In 2011, Stroupe et al. studied the costs of treating pressure ulcers among veterans with SCI.[38] The total inpatient cost per year for SCI veterans with pressure ulcers was $91,341, while that for a veteran without pressure ulcer was $13,754. The total outpatient cost per year for SCI veterans with pressure ulcers was $19,844, compared to $11,829 for those without a pressure ulcer.[38]

Total healthcare costs for individuals with SCI are higher than other groups at risk for pressure ulcer development and increasing rapidly.[39] The additional costs of treating veterans with SCI who have pressure ulcers have been estimated to be over $73,000 per annum due primarily to higher inpatient costs.[38]

Risk Factors

More than 200 risk factors for the development of pressure ulcers have been reported in the literature. Most of the risk factors were derived from studying elderly nursing home residents. However, many of the risk factors for these patients differ from those experienced by patients with SCI. Immobility increases the risk of pressure ulcer development in both populations. Unlike nursing home residents, however, patients with SCI are

encouraged to oversee or direct their own daily care and are expected to take primary responsibility for pressure ulcer prevention. As a result of the limitations imposed by the variables among studies, the literature is often contradictory regarding the effects of a particular risk factor or set of factors potentially responsible for the development of pressure ulcers. Different populations (e.g., acute or chronic SCI patients), inadequate sample sizes, different ways of standardizing the dependent measures, and poor or uncontrolled study designs all add confusion to the interpretation of study results.[40,41]

According to Chen et al., the risk of pressure ulcers seems to be steady in the first 10 years but increases 15 years postinjury.[11] Males, the elderly, blacks, singles, individuals who have less than a high school education, the unemployed, persons with complete SCI, and those with a history of pressure ulcers, rehospitalization, nursing home stay, and other medical comorbidities are at greater risk of developing pressure ulcers.[11] Although the number of days hospitalized and frequency of rehospitalizations decreased, the number of pressure ulcers increased over time.[11] Charlifue et al.[8] found that although the number of pressure ulcers increased as time passed, the best predictor of pressure ulcers over time was a previous history of pressure ulcers.

Despite these limitations, a number of pressure ulcer risk factors specific to the patient with SCI have been identified and described in the literature. Byrne and Salzberg[40] summarized the major pressure ulcer risk factors for the patient with an SCI as follows:

- Severity of the SCI (immobility, completeness of the injury, urinary incontinence, and severe spasticity)
- Preexisting conditions (advanced age, smoking, lung and cardiac disease, diabetes and renal disease, and impaired cognition)
- Residence in a nursing home
- Malnutrition and anemia

Characteristics associated with better pressure ulcer outcomes include maintaining normal weight, returning to work and family roles, and not smoking or having a history of tobacco use, suicidal behavior, incarceration, or alcohol or drug abuse.[42] The Consortium for Spinal Cord Medicine Clinical Practice Guidelines ("Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury: a clinical practice guideline for health-care professionals")[6] categorized pressure ulcer risk factors in patients with SCI

as follows:

- Demographic factors (age, gender, ethnicity, marital status, and education)
- Physical, medical, and SCI-related factors (level and completeness of the injury; activity and mobility; nutrition, bladder, bowel, and moisture control; and comorbidities, such as diabetes and spasticity)
- Psychological and social factors (psychological distress, financial problems, cognition, substance abuse, adherence to recommended prevention behaviors/strategies, context and health beliefs and practices)
- Support surfaces for bed and wheelchair

As far back as 1979, Anderson and Andberg[43] identified psychological factors associated with the development of pressure ulcers, including the patient's unwillingness to take responsibility for his or her skin care, low self-esteem, and dissatisfaction with life activities. Gordon et al.[44] also found poor social adjustment in the patient with an SCI and a pressure ulcer. The revised edition of the Paralyzed Veteran's Association (PVA) The Consortium for Spinal Cord Injury Clinical Practice Guidelines on Prevention and Treatment of Pressure Ulcers in Persons with SCI[6] lists the following psychological and cognitive risk factors:

- Psychological factors: Depression, anxiety, negative self-concept, poorly managed anger, and frustration may interfere with cooperation between individual and his/her care providers and may be associated with inactivity, self-neglect, and poor medical adherence.[45]
- Cognitive impairment: Associated traumatic brain injury and illegal drug use may interfere with cognitive function.[46]

Social, environmental, contextual, and circumstance factors: In recent years, there have been attempts to identify risk factors, other than obvious medical and SCI-related ones, within a person's life that contribute to the development of pressure ulcers, interfere with healing, and fail to prevent recurrence. Homelessness, gang membership, financial problems, and lack of social support have been shown to cause frequent disruptions, promote participation in high-risk activities, and may put the person in a state of danger. The lack of access to care, services, and supports can indeed influence the development of pressure ulcers.[47–50] In persons with chronic

SCI and pressure ulcers, it is important to assess the individual's motivation to stay ulcer-free.[49,50] Motivation drives the daily actions or inactions of the individual. Lack of motivation to perform preventative activities may be associated with depression, lack of social support, or poverty. Conflicting motivations, such as wanting to stay fully engaged in either vocational or recreational pursuits, may interfere with maintaining a strict regimen of pressure ulcer prevention. Understanding life context, daily routines, and central daily activities should result in a balance between ones daily occupations and pressure ulcer preventative routines. Therefore, in order to most effectively prevent and treat pressure ulcers in any individual, it is important to not just evaluate the usual risk factors as described throughout in this guideline but also to evaluate the context in which the individual with SCI lives, for, if these other factors are not addressed as well, any intervention is not likely to be successful.

Support surfaces for bed and wheelchair: Support surfaces that are old, worn-out, or inappropriate may put the person at risk for pressure ulcers.

The use of both a validated risk assessment tool and clinical judgment to assess risk is strongly recommended. The Salzberg Pressure Ulcer Risk Assessment Form for SCI is only one risk assessment form validated for persons with SCI.[18] However, in many facilities, the Braden scale, which is not specific to SCI, is used to determine persons at risk for PrUs.

Practice PointPractice Point

Innovative educational programs are needed to provide persons with SCI with information and the motivation necessary to regain control over their lives. It is necessary to determine how effective these programs are through testing knowledge and observing preventive behavior.

Risk-Based Prevention

A person with an SCI is at risk for the development of pressure ulcers from the moment of injury. Prolonged immobilization during the hours and days immediately after injury significantly increases the risk. Pressure reduction strategies to protect vulnerable areas of the body should be implemented soon after emergency medical intervention and spinal stabilization.

Practice PointPractice Point

Pressure ulcer development is a lifelong concern for the individual with SCI.

Prevention strategies must be implemented across the full spectrum of care. A comprehensive preventive plan should be developed for each patient and include information and instructions that are given to patients and their families.[51] (See *Patient Teaching: Preventing Pressure Ulcers in SCI Patients at Home*.)

Printed materials or visual media, such as DVDs, are used frequently to augment educational sessions. Some of these materials may be sent home with patients when they are discharged. Because most patient education programs are hospital based, little is known about what information the patient retains, which behaviors or activities are practiced routinely, and the compatibility of the patient's lifestyle with prevention strategies. In the 1970s and 1980s, a number of SCI centers established comprehensive pressure ulcer prevention education programs.[52,53] Both inpatient and outpatient programs advocated multidisciplinary, coordinated, structured, and wide-ranging approaches to prevention. Some of these programs still serve as models for practice today.[54]

Several educational needs have been described recently. These include (rank ordered by the authors of the study)[55]:

- awareness of the lifelong risk for developing pressure ulcers (including the ability to assess personal risk factors and how risk changes over time)
- ability to take charge of personal skin care regimen and to partner with healthcare providers
- ability to perform prevention strategies consistently that are compatible with level of function and activity and update practices as risk changes
- ability to coordinate social supports.

Effective prevention education and early detection of pressure ulcers are critical.[50]

The concepts of context and lifestyle as they pertain to pressure ulcer etiology, prevention, and management have been described recently in the

literature. Clark et al.[49] examined the daily lifestyle influences on the development of pressure ulcers among adults with SCI. In-depth interviews and observation were used to obtain detailed descriptive information pertaining to the development of recurring pressure ulcers as they relate to participants' overall lifestyle, daily routine, choices, challenges, and prevention strategies. These investigators concluded that prevention is dependent on the uniqueness of an individual's everyday life.[49]

In a qualitative study[50] of 20 adults with SCI and a history of pressure ulcers, investigators identified eight multifaceted, interconnected lifestyle principles that explain pressure ulcer etiology. They include perpetual danger; change/disruption of routine; diminished prevention behaviors; risk ratio of liabilities (risk factors or behaviors) to buffers (factors, behaviors, or people that reduce risk); individualization; presence of prevention awareness and motivation; lifestyle trade-off; and access to needed care, services, and supports.[50]

Patient TeachingPreventing Pressure Ulcers in SCI Patients at Home

Persons with SCI are at risk for developing pressure ulcers, especially after they leave your care. Teach patients and their families the following strategies to help them prevent pressure ulcers after they have returned home:

- Perform daily comprehensive visual and tactile skin inspection.
- Maintain good personal hygiene.
- Turn and reposition often, and perform frequent weight shifts.
- Use appropriate and well-maintained support surfaces for the bed and wheelchair.
- Maintain adequate nutrition.
- Maintain a healthy lifestyle (avoid alcohol, tobacco, and illegal drugs).

Skin Assessment

The SCI patient with a pressure ulcer should undergo two assessment phases. The first phase is a comprehensive evaluation and examination, including:

- complete social history including family, financial stability, or other support
- medical history including comorbidities
- physical examination including functional independence (ADLs, mobility, transfer skills)
- laboratory tests and nutrition
- assessment of psychological health, behavior, and cognitive status
- information on social and financial resources and the availability and utilization of personal care assistance
- assessment of positioning, posture, and related equipment
- assessment of lifestyle, including use of tobacco, past and present, and alcohol and drug use/abuse.[6]

The second phase of assessment consists of a detailed description of the pressure ulcer itself and the surrounding tissues, including the following factors:

- Etiology of the pressure ulcer
- Anatomical location and general appearance and characteristics of the wound base
- Size (length, width, depth, and wound area)
- Stage or severity
- Exudate
- Odor
- Necrosis
- Undermining
- Sinus tracts
- Infection
- Viable tissue (granulation, epithelialization, muscle or subcutaneous tissue)
- Nonviable tissue (eschar, clean nongranulating slough)
- Wound margins and surrounding tissue[6]

Photographs can be useful in these assessments and in monitoring.

The pressure ulcer should be monitored with each dressing change and the above factors reviewed at regular intervals.

Practice PointPractice Point

Patients with darkly pigmented skin are particularly vulnerable to undetected pressure ulcers. Although areas of damaged skin appear darker than the surrounding skin, tactile information must be used in addition to visual data when assessing persons with darker skin. The skin may be taut and shiny, indurated, and warm to the touch. Color changes may range from purple to blue. Remember, pressure-damaged dark skin doesn't blanch when compressed.[56]

Treatment

Prevention and treatment are inextricably linked across the continuum of care for the person with a pressure ulcer.[57] During rehabilitation following an SCI, the patient is exposed to a great deal of information about the major physiological changes that have occurred as well as how to prevent or manage potential secondary complications, such as pressure ulcers and UTIs. Unfortunately, much of this information isn't absorbed during this early posttraumatic phase, resulting in episodes of potentially life-threatening conditions once the patient returns to his or her home and community. Coupled with nonretention of information is today's significant decrease in length of stay, which makes structured education sessions during hospitalization very limited at best or totally absent.

Nonsurgical Treatment

The treatment of pressure ulcers is a complex process, based on a number of patient-related and pressure ulcer–related factors. Nonsurgical treatment for pressure ulcers consists of a number of sequential steps that become the treatment plan for that patient with his or her specific ulcer. The elements of a comprehensive treatment plan include cleansing, debriding, applying dressings, and assessing the need for (and appropriateness of) new technologies aimed at wound healing. Education, in the form of printed materials or discussions with healthcare professionals, is intended to prevent recurrence in the patient with an SCI. Enhanced, individualized pressure ulcer prevention and management education is effective in improving pressure ulcer knowledge during hospitalization for either

medical management or the surgical repair of a pressure ulcer.[58,59] Furthermore, initial individualized preventive intervention combined with structured follow-up within a person's individual everyday life setting may reduce the risk of pressure ulcers in persons with SCI.[60]

Initially, creating a physiologic wound environment is essential. Cleansing is accomplished with normal saline, sterile water, pH balanced wound cleanser, or lukewarm potable tap water. Other cleansing agents (such as diluted sodium hypochlorite ¼ to ½ strength for wounds with heavy bioburden for a limited time) and mechanical wound cleansing techniques are also employed depending on the status of the wound and surrounding tissue. Cleansing is followed by debridement of devitalized tissue and selection of wound care dressing. A number of adjunctive therapies have been identified. However, electrical stimulation is one of the very few to be supported by the scientific evidence. The treatment plan should be modified if the ulcer shows no evidence of healing within 2 to 4 weeks.

Surgical Treatment

Stage III and IV pressure ulcers are frequently treated surgically in patients with SCI. The goals of surgical closure include[6,61,62]:

- preventing protein loss through the wound
- reducing the risk of progressive osteomyelitis and sepsis
- preventing renal failure
- reducing costly and lengthy hospitalization
- improving hygiene and appearance
- expediting time to healing.

The surgical process includes[61,62]:

- excision of the ulcer and surrounding scar, underlying bursa, soft tissue calcification, and underlying necrotic or infected bone
- filling dead space with fascia or muscle flaps
- improving vascularity and distribution of pressure over bony prominences
- resurfacing the area with a large flap so that the suture line is away from areas of direct pressure
- providing a flap that leaves options for future surgeries.

Preoperatively, the rehabilitation and surgical teams coordinate their efforts to control local wound infection, improve and maintain nutrition, regulate the bowels, control spasms and contractures, and address comorbid conditions. Previous pressure ulcer surgery, smoking, UTI, and heterotopic ossification could affect surgical outcomes.[6]

New surgical techniques to repair pressure ulcers have been developed and are being used to improve surgical outcomes. Although these techniques are being evaluated,[63–67] reports of long-term follow-up of the status of the skin and recurrence have been limited. One study by Lee[63] used a new wound closure technique and followed patients for 102 days, after which 18 of 21 (86%) wounds in 13 patients remained closed. Sorensen et al.[68] suggested that thorough preoperative debridement, patient compliance, control of comorbidities, professional postoperative support, and sufficient pressure relief are essential if surgical success is to be achieved.

Evidence-Based PracticeEvidence-Based Practice

Postsurgical care includes keeping the surgical site pressure-free, preventing infection, preventing dehiscence of a flap, using specialty beds to maximize pressure reduction, mobilizing the patient progressively, and providing patient and family education.[6]

Support Surfaces

Support surfaces are devices or systems intended to reduce the interface pressure between a patient and his or her bed or wheelchair.[57] Support surfaces neither prevent nor heal pressure ulcers; rather, they are prescribed by a clinician and incorporated into a comprehensive pressure ulcer prevention and management program. Pressure redistribution products, such as active or reactive mattresses, mattress overlays, or specialty beds, may be used at various times to reduce the patient's risk of developing pressure ulcers. Reactive support surfaces are used for individuals who have a stable spine and who are able to reposition themselves enough to avoid weight bearing on all areas at risk for pressure ulcers. Active support surfaces and a high air-loss or air-fluidized reactive support surface are for individuals

who have pressure ulcers on multiple turning surfaces and/or status postflap/skin graft within the past 60 days.[6] Bed positioning devices and techniques must be compatible with the bed type and the individual's health status. Individuals should not be positioned directly on a pressure ulcer. Rather various positioning aids, such as pillows, and cushions should be used to reduce pressure on areas vulnerable to tissue breakdown. Although there is controversy regarding the frequency of turning or repositioning a person with a pressure ulcer, the literature supports the importance of individualizing a turning schedule. There are a great number of pressure redistribution bed support surfaces for individuals with or at risk to pressure ulcers. The use of these "specialty beds" is determined by the person's status and the individual facility's experience with these devices. These include air-fluidized and low-air devices.

Materials for wheelchair positioning and pressure ulcer prevention, such as foams and gels, used alone or in combination, and elements such as air and water, also used alone or in combination, are being used across inpatient and home environments. Wheelchairs and seating systems should be specific to the individual and allow that individual to redistribute pressure sufficiently to prevent pressure ulcers. The optimum wheelchair seating system should[6]:

- redistribute pressure
- minimize shear
- provide comfort and stability
- reduce heat and moisture
- enhance functional activity
- be inspected at regular intervals for wear.

Wheelchair cushions and seating systems of various materials and designs are intended to reduce pressure and maximize balance and stability when a patient is in a wheelchair. It must be stated emphatically that wheelchair cushions neither prevent nor heal pressure ulcers. Rather, they are used as one important tool in the clinician's armamentarium of prevention and treatment strategies. Power weight-shifting wheelchairs are often prescribed for individuals who are unable to independently perform an effective pressure relief while sitting. Full-time wheelchair users with or without a history of pressure ulcers should limit sitting and maintain an off-loaded position from the seating surface for at least 1 to 2 minutes every 30 minutes.[6] (For further discussion on support surfaces, see Chapter 11, Pressure Redistribution: Seating, Positioning, and Support Surfaces.)

Adverse effects of pressure on tissue are a major source of morbidity and mortality in persons with SCI.[6] Issues surrounding wheelchair sitting and associated seating and positioning devices have been studied extensively and therapeutic strategies developed to minimize pressure on the skin, especially in anatomical areas overlying bony prominences.[69–71] These efforts have led to major improvements in the technology of seating support and pressure-reducing devices such as wheelchair cushions and in mattresses for the supine patient.

Another area remains as yet uninvestigated, namely, the adverse effects of pressure exerted while sitting on a commode chair during bowel care procedures necessary for persons with SCI. Chronic constipation, difficulty in evacuation, incontinence, and damage to mucosa are all complications associated with neurogenic bowel in persons with SCI. One attempt to deal with the adverse effects of pressure during bowel management programs is the padded commode seat. This has led to some success in safety and in reducing the risk of pressure ulcers during the bowel management procedures.[72–76] Rates of rehospitalization following SCI remain high and result from complications in the genitourinary system, respiratory complications, and pressure ulcers.[15] Unfortunately, very little data exist on the specific nature of pressure ulcer development in association with bowel care sitting time or process. Research is needed on an equally relevant therapeutic approach that would focus on duration of sitting time during each evacuation attempt.

New Interventions

A number of adjunctive therapies have been reported in the literature with varying degrees of success in treating pressure ulcers in the patient with an SCI, including:

- electrical stimulation
- ultraviolet and laser therapy
- hyperbaric oxygen and ultrasound
- negative pressure wound therapy
- nonantibiotic drugs
- topical agents
- skin equivalents
- growth factors.

Among these, only electrical stimulation has enough reported scientific evidence supporting it to justify its use as a treatment for pressure ulcers in the patient with an SCI.[77,78]

Summary

Although SCI research has increased tremendously in recent years, designing and conducting randomized, controlled trials that are capable of producing compelling observational evidence on which to base management of pressure ulcers have been disappointing. Despite advances in pressure ulcer treatment, little scientific evidence points the way to preventing pressure ulcers in the SCI population. Randomized, controlled trials in real-world settings are the gold standard for assessing the effectiveness of prevention and treatment strategies. However, in complex, rapidly changing healthcare settings, blinding is infeasible and it may be impractical to control for every variable that influences a study's outcome. Furthermore, any assumptions that usual care is static are probably mistaken. Innovative approaches to maintain the integrity of the study design must be used, including flexibility in inclusion and exclusion criteria to support accrual, obtaining a better understanding of the important aspects of usual care that may need to be standardized, continuous improvement within the intervention arm, and anticipation and minimization of risks from organizational changes.[31] Research efforts should focus on prospective studies to prevent recurrence that include long-term follow-up programs promoting self-management.

PATIENT SCENARIO: SPINAL CORD INJURY POPULATION

Clinical Data

Mr. K is a 48-year-old African American man who sustained a complete SCI at the T8 level from a motor vehicle crash 7 years ago that resulted in paraplegia. He lives independently, is unmarried and unemployed, and has a history of hypertension. During his rehabilitation hospitalization, Mr. K was told about pressure ulcers and what he could do to prevent them. However, within 1 year after

discharge, he developed a stage III right ischial pressure ulcer for which he was hospitalized for surgical repair. The surgery resulted in a healed ulcer, and Mr. K. was discharged. Although pressure ulcer prevention was reviewed during this hospitalization and consumer-based written material was provided, he returned home and resumed previous habits, including sitting in front of the television for long hours, driving around with friends, not eating well, and smoking. Less than a year after the first surgery, Mr. K developed a stage IV pressure ulcer with osteomyelitis on his left ischium. He was readmitted to the hospital for the surgical repair of this ulcer. Compounding Mr. K's pressure ulcer history are the following: He does not have a support system to help and encourage him to be more proactive in preventing pressure ulcers; he seems depressed and unable to take control of his physical health; he would like to return to work as a computer programmer but has not put much effort into looking for a job; he often does not take his prescribed medication for hypertension; he developed type 2 diabetes; and he smokes and sometimes uses alcohol and illegal drugs. His bowel care required several hours on the commode, contributing to pressure ulcer recurrence on fragile skin.

Case Discussion

The first priority is to treat the infection (osteomyelitis) and surgically repair the ulcer. During this hospitalization, a case manager was assigned to coordinate discharge and follow-up plans. The case manager requested that an occupational therapist knowledgeable about SCIs and pressure ulcers work with Mr. K to develop strategies to prevent future pressure ulcers, help him to take control of the things he can control, identify appropriate support systems within the community and the healthcare system, and improve his quality of life. The occupational therapist worked with Mr. K to develop a pressure ulcer prevention plan that was acceptable and appropriate to Mr. K's lifestyle. Relevant actions and behaviors were included in a written plan so Mr. K could refer to it daily. These included weight shifts and turns, skin checks (with special attention to the changes in his darkly pigmented skin), nutrition, hygiene, limiting sitting in one place for long periods of time, consistency with prescription medications, limiting smoking and drinking, and not using drugs. Additionally, the occupational therapist performed a complete assessment of Mr. K's support surfaces used in the bed and wheelchair as well as the

wheelchair itself. His wheelchair and wheelchair cushion were in very poor condition. A new wheelchair was ordered and a pressure evaluation performed to identify the most effective wheelchair cushion for him. A mattress overlay, compatible with his bed at home, was also prescribed.

Mr. K was instructed on the routine care of his new equipment (wheelchair, mattress overlay, and cushion), especially with regard to changes in his skin that might reflect the deterioration of the support surfaces. He was re-evaluated for management of his hypertension, and a new medication regimen was designed. A program to manage his diabetes also was implemented. His bowel regimen was reviewed and modified, and a new commode/shower chair was ordered. A home visit by the occupational therapist identified ways to minimally adapt Mr. K's home environment to maximize his independence and safety. He was referred to a psychologist for counseling that seemed to result in better insight into his behaviors and lifestyle. Finally, Mr. K was given a list of resources he could contact with problems, including the case manager, equipment vendor, physician, and clinics. He also was given information on how to seek employment.

HIV/AIDS POPULATION

Carl A. Kirton, DNP, RN, ANP-BC, ACRN

Objectives

After completing this chapter, you'll be able to:

- describe the impact of highly active antiretroviral therapy (HAART) on the prevalence of skin disorders in the patient with human immunodeficiency virus (HIV) or acquired immunodeficiency syndrome (AIDS)
- describe six common infectious skin disorders and two common noninfectious skin disorders in the patient with HIV or AIDS that results in altered skin integrity
- discuss two of the neoplastic skin disorders seen in the patient with HIV or AIDS.

Skin Alteration in HIV and AIDS Patients

More than 90% of HIV-infected patients will develop at least one type of dermatologic disorder during the course of their HIV infection.[1] In fact, in the early 1980s, it was the identification of an unusual skin lesion in young homosexual men that prompted the search for the virus that causes AIDS. A broad range of infectious and noninfectious skin lesions may develop during both the asymptomatic and symptomatic courses of the disease. Alteration in the skin is often the first manifestation of an impaired immune system and may be a sign or symptom of a serious opportunistic infection. Skin alterations can also indicate advancing HIV disease.

Several points regarding HIV, skin disease, and its treatments are noteworthy. Lesions that are common in the non–HIV-infected adult population may present atypically in HIV-infected persons. In addition, skin disorders often aren't responsive to the usual treatments, may be present for longer than expected, and may develop into chronic, disfiguring disorders. Skin lesions may also be the precursor of a life-threatening illness.

Practice PointPractice Point

Prompt and accurate investigation of skin lesions in the HIV-infected patient is essential and often warrants collaboration with an HIV specialist.

The effective combination of several antiretroviral drugs to suppress viral replication with consequent repletion of the CD4+ lymphocyte count is the standard of care treatment for HIV infection. Antiretroviral therapy (ART) and drugs that prevent or treat opportunistic infections have contributed to a significant decline in HIV-associated morbidity and mortality. The introduction of ART has also resulted in a dramatic decline in HIV immunosuppression–related diseases including skin diseases, including Kaposi's sarcoma, eosinophilic folliculitis, molluscum contagiosum, bacillary angiomatosis, and condylomata acuminata. One study estimated that after the introduction of protease inhibitors (often called the post-HAART era), the total number of HIV patients with skin problems was

reduced by 50%.[2,3] Although ART and regimens to treat opportunistic infections have improved the quality of life for patients by controlling HIV replication, the increased use of pharmaceutical agents has led to an increased incidence of adverse reactions to these drugs.[4–6] The risk for adverse cutaneous reactions to certain drugs is greatly increased in patients with HIV compared with that of the general population (as much as 100 times) and occurs in tandem with the level of immunosuppression.[7] Drugs often associated with adverse effects include sulfonamides, co-trimoxazole, and tuberculostatics as well as many of the antiretrovirals. (See Table 21-2.)

Table 21-2 Adverse Cutaneous Reactions in HIV Disease

Drug Class	Dermatologic Manifestations
Nucleoside reverse transcriptase inhibitors (NRTI)	Lipodystrophy (especially zidovudine, stavudine), nail and mucocutaneous hyperpigmentation (especially zidovudine and emtricitabine), hypertrichosis of the eyelids, leukocytoclastic vasculitis Life-threatening skin diseases: Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN), abacavir hypersensitivity reaction (fever, rash—of any type, gastrointestinal disturbances, constitutional complaints, respiratory symptoms)
Non-nucleoside reverse transcriptase inhibitors (NRTI)	Most frequent class of antiretroviral therapy (ART) to cause morbilliform eruptions. Morbilliform eruptions related to nevirapine may become severe systemic hypersensitivity reactions Morbilliform rash (especially with efavirenz, etravirine) Less than 1% of patients will develop SJS
Protease inhibitors	Generally low rates of cutaneous manifestations. Morbilliform rash (especially nelfinavir, lopinavir) Maculopapular eruption (low rates with early administration of amprenavir and fosamprenavir) Generalized rash (especially with tipranavir as urticaria, morbilliform eruptions, photosensitivity, especially in children)
Fusion inhibitors	Injection-site reactions: erythema, cysts, and nodules at injection sites
Entry inhibitors	No significant reactions reported
Integrase inhibitors	No significant reactions reported

Cutaneous Drug Eruptions

Cutaneous drug-induced eruptions, not due to ART, can occur with some drugs used to prevent opportunistic infections. Bactrim (trimethoprim–sulfamethoxazole), the most effective drug in the prevention and treatment of *Pneumocystis jiroveci* pneumonia, is known to cause cutaneous eruption in patients with HIV infection. The rate of cutaneous eruption associated with trimethoprim–sulfamethoxazole in HIV patients is 20% to 80% compared with 1% to 3% in persons without HIV infection, possibly due to altered

drug metabolism, decreased glutathione levels, or both.[8]

Practice PointPractice Point

Approximately 50% to 60% of HIV-positive patients have been shown to develop a morbilliform eruption within 7 days of starting trimethoprim–sulfamethoxazole therapy. A trimethoprim–sulfamethoxazole drug eruption is characterized by the widely disseminated spread of fine pink to red macules and papules involving the trunk and extremities. The clinician must provide a complete head-to-toe examination of the skin within the first week of therapy and teach the patient to notify the provider of any unusual skin eruptions.

In its severe form, Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) may also develop. SJS is characterized by fever as well as widespread blisters of the skin and mucous membranes of the eye, mouth, or genitalia. TEN is a more serious manifestation of SJS that involves widespread areas of the skin with confluent bullae that can lead to loss of skin in massive sheets.

Practice PointPractice Point

TEN may lead to secondary infection with sepsis, volume depletion, and high-output cardiac failure as a consequence of widespread denudation of the skin. Patients who develop TEN must be treated aggressively in an acute care setting.

Immune Reconstitution Inflammatory Syndrome

Immune reconstitution inflammatory syndrome (IRIS) (also referred to as “immune reconstitution inflammatory syndrome” or “immune restoration disease”) is a syndrome characterized by a paradoxical clinical

deterioration in or an exacerbation of certain conditions, some of which may potentially manifest in the skin and may occur in as much as 40% of commencing ART.[9] IRIS is thought to be the result of an overzealous immunologic response to infectious or self-antigens as the immune system is restored with HAART. IRIS results in frank disease but often results in moderate to severe cutaneous eruptions, including follicular inflammatory eruptions, mycobacterial skin infections, and viral infections such as human herpes virus infection (e.g., herpes zoster). IRIS may even exacerbate an underlying autoimmune disease such as lupus erythematosus.

Infectious Skin Disorders

The immunocompromised status of patients with HIV or AIDS puts them at greater risk for infectious bacterial or viral skin disorders, such as herpes virus, cytomegalovirus (CMV), human papillomavirus, molluscum contagiosum, *Staphylococcus* and *Streptococcus* infection, and bacillary angiomatosis.

Herpes Virus

Breakouts of grouped blister-like lesions typically caused by the common herpes virus are easily recognized and common in patients with HIV at all stages of the disease. Herpes zoster may occur either early or late in the course of HIV-induced immunosuppression and may be the first clinical clue to suggest undiagnosed HIV infection. Herpes infection may occur on the oral and genital mucosa as well as in the perianal region. Lesions typically manifest as painful, grouped vesicles on an erythematous base that rupture and become crusted. History and clinical presentation are often all that's necessary to establish the disorder; therefore, confirmatory tests, such as viral cultures, HSV DNA, and HSV antigen detection assays, are rarely necessary. In patients with advanced HIV, a herpetic infection may develop into chronic ulcers and fissures with a substantial degree of edema.

Infections with human herpes simplex virus type 1 (HSV-1) and type 2 (HSV-2) are common, with a seroprevalence of HSV-1 among adults in the United States of approximately 60% and a seroprevalence of HSV-2 among persons aged ≥12 years of 17%. Approximately 70% of HIV-infected persons are HSV-2 seropositive and 95% are seropositive for either HSV-1 or HSV-2.[10] Herpes zoster can occur in HIV-infected adults at any CD4+ count, but the frequency of disease is highest in those with CD4+ counts less than 200 cells/μL. Unlike other skin disorders, the incidence of herpes

zoster infection is *not* reduced by ART.

Patient TeachingHerpes Virus Teaching Tips

- Scrupulous hand washing helps prevent the spread of infection.
- Use individual washcloths and linens.
- Mild analgesics may be necessary for pain associated with herpes infection.
- Topical soaks (such as Domeboro solution) can be used to help dry wet lesions.
- Impetiginization of skin lesions may be treated with warm compresses.

Uncomplicated zoster outbreaks should be treated for 5 to 10 days with acyclovir (Zovirax), famciclovir (Famvir), or valacyclovir (Valtrex). Painful atrophic scars, persistent ulcerations, and acyclovir-resistant chronic verrucous lesions may also develop. (See *Patient Teaching: Herpes Virus Teaching Tips*.)

Practice PointPractice Point

Healing of herpetic lesions is usually complete in less than 2 weeks. If they haven't healed within 3 to 4 weeks, the patient may have a drug-resistant virus. Acyclovir-resistant cases of varicella-zoster virus or herpes simplex virus infection require treatment with IV foscarnet (Foscavir).

Practice PointPractice Point

A patient with herpes zoster involving V1, the ophthalmic division

of the trigeminal nerve, should be referred to an ophthalmologist immediately due to the risk of corneal ulceration. Signs or symptoms of this condition, such as painful vesicular lesions on the tip of the nose or lid margins, should be considered an ocular emergency.

Cytomegalovirus

CMV is a double-stranded DNA virus in the herpes virus family that can cause disseminated or localized end-organ disease among patients with advanced immunosuppression. The incidence of new cases of CMV end-organ disease has declined by 75% to 80% with the advent of ART.[10] When the skin is involved, CMV may cause a number of different clinical manifestations, including ulcers, verrucous lesions, and palpable purpuric papules. Effective treatments for a CMV infection include oral valganciclovir (Valcyte) or IV ganciclovir (Cytovene), foscarnet (Foscavir), or cidofovir (Vistide).

Practice PointPractice Point

Ulcers are commonly secondarily colonized with CMV, and many patients have combined herpes simplex and CMV infections.

Human Papillomavirus

The most common skin complaint of HIV-positive patients is warts caused by the human papillomavirus (HPV). It has been shown that immune deficiency is associated with increased frequency of HPV infections, suggesting that the emergence of HPV is modulated by the patient's immune status.

Verruca vulgaris warts (common warts) appear as dull-colored papules that can erupt anywhere on the skin; verruca plana warts are flat-topped, skin-colored papules on the face and dorsal hands. Condyloma acuminata warts (genital warts) are characterized by soft, skin-colored cauliflower papules on the genital areas. Their appearance, size, and number vary with the site. Warts can range in size from less than 1-mm to 2-cm "cauliflower lesions." Verruca plantaris warts are hyperkeratotic papules and plaques

that appear on the soles of the feet. Certain types of HPV have oncogenic potential and are associated with cervical cancer in women, bowenoid papulosis of the penis, anal cancers, and invasive carcinoma. HPV-16 alone accounts for approximately 50% of cervical cancers in the general population and HPV-18 for another 10% to 15%, whereas the other oncogenic HPV types each individually account for less than 5% of tumors.[10] Unlike other HIV-associated infections, treatment with ART has not affected the incidence of HPV infection.

Treatment, although effective, rarely eradicates HPV entirely. Destructive measures—such as the application of topical chemicals (e.g., salicylic or trichloroacetic acid), cryotherapy with liquid nitrogen, and ablative surgery—are standard measures used for common verrucae (warts). Condyloma acuminata can be treated by using podophyllin resin 10% to 50% in tincture of benzoin, 3% cidofovir ointment, intralesional interferon-a, liquid nitrogen cryotherapy, electrodesiccation and curettage, or carbon dioxide laser.

Patient TeachingPatient Teaching

Imiquimod cream 5% or podofilox 0.5% or sinecatechin 15% is often prescribed for home application to prevent recurrence of HPV infection. Instruct the patient to apply the medication to his or her warts at night three times per week for up to 16 weeks.

Patients who use these creams may experience application-site reactions such as itching and/or burning. Skin reactions may be of such intensity that patients *may require rest periods from treatment*. Teach the patient to use the cream exactly as prescribed; using too much cream, or using it too often or for too long, can increase chances of having a severe skin reaction.

Patient TeachingPostprocedure Care after HPV Surgical Excision

Postprocedure patient teaching should include the following:

- Medication usually isn't needed after removal of lesions.

Topical anesthetic ointments may be used to minimize discomfort. Sitz baths may aid resolution when large areas are treated; silver sulfadiazine (Silvadene) ointment or antibiotic ointment may not only be soothing but may also reduce the possibility of superficial infection. No dressing is required, but some patients may request a sanitary napkin for treated genital lesions. Ice packs are helpful.

- Cryonecrosed lesions will progress from erythema to edema and then will turn black. The lesions will disappear within a few days, and healing should be complete in 7 to 8 days. For chemically cauterized lesions, the healing process is usually less than 1 week.
- Treated areas should be washed and dried gently each day of the healing process. Postcryotherapy management is similar to that for a superficial partial-thickness burn.
- If acute discomfort persists beyond 48 hours, instruct the patient to contact the physician.
- Rarely, a mixture of equal parts of 20% benzocaine (Hurricaine) ointment and topical antibiotics may be used. Lidocaine ointment 5% provides excellent relief and also keeps the tissues moist.
- Counsel the patient to report excessive discomfort or any signs of infection.[11]

Plantar verrucae are generally treated with topical 40% salicylic acid plaster applied daily, with paring of hyperkeratotic areas, although intralesional bleomycin and liquid nitrogen therapy have also been used. Verruca plana warts are commonly treated with topical tretinoin alone or in combination with 5-fluorouracil. Light electrodessication or liquid nitrogen application may be used as an adjunct therapy. Verrucous carcinoma requires excisional surgery. (See *Patient Teaching: Postprocedure Care after HPV Surgical Excision*.)

Molluscum Contagiosum

Molluscum contagiosum is a benign, usually asymptomatic viral skin infection caused by the poxvirus; it is spread by direct contact and causes no systemic manifestations. The diagnosis can usually be made from the

characteristic appearance of dome-shaped, umbilicated, translucent papules that may develop on any cutaneous site, especially the genital areas and the face. In the patient with AIDS, lesions may become widespread, disfiguring, and resistant to treatment. The lesions appear verrucous, pruritic, or eczematous. Once they become confluent, they can be difficult to treat. Although the exact incidence of molluscum contagiosum in patient with AIDS is unknown, it is estimated to be 5% to 18%.[12] Treatment is generally by destructive measures, including cryotherapy or curettage. (See *Patient Teaching: Molluscum Contagiosum*.)

Patient TeachingPatient Teaching

Molluscum Contagiosum

- Molluscum contagiosum can be transmitted through direct contact.
- The lesions are prone to autoinoculation, and in male patients, shaving the beard area has been reported to cause particularly severe infections, with lesions encompassing the entire face.
- Covering lesions with clothing and/or bandages is one effective way to prevent spread.
- Good hand hygiene and avoiding touching the lesions is another way to prevent spread of fomites.
- Cryonecrosed lesions will progress from erythema to edema and then will turn black. The lesions will disappear within a few days, and healing should be complete in 7 to 8 days.
- For chemically cauterized lesions, the healing process is usually less than 1 week.

Staphylococcus or *Streptococcus*

In general, most bacterial infections are caused by *Staphylococcus* and *Streptococcus* organisms and are commonly encountered in immunocompetent patients. Primary bacterial lesions manifest as vesicles, papules, and pustules and are often pruritic. It's the pruritic feature that often leads the patient to scratching, subsequently resulting in a break in the epithelial surface followed by excoriation of the lesion. Some lesions (such

as impetigo) may contain purulent fluid. Diffusely red, warm, tender areas in the skin suggest soft tissue cellulitis or a deep-seated infected wound.

In recent years, there has been an increase in the number of HIV patients with community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) infection. The incidence of CA-MRSA soft tissue infection is sixfold higher among HIV-infected patients than it is among HIV-negative patients.[13] It is now established that HIV disease is a major risk for CA-MRSA, possibly related to immunodeficiency.[14] Other recent factors include recent exposure to antibiotics, illicit drug use, recent hospitalizations, prior MRSA colonization or infection, and chronic skin disease. Lifestyle factors such as high-risk sexual behaviors, never using condoms, recent sexually transmitted infection, and public bath use have associated with colonization.[14]

Treatment with dicloxacillin, cephalexin, or ciprofloxacin is indicated in bacterial infections. Clindamycin or linezolid remains a commonly used treatment option for CA-MRSA. Wounds caused by bacterial infections should be assessed regularly and treated accordingly; incision and drainage may be indicated. (See *Patient Teaching: Preventing Staphylococcal or CA-MRSA Skin Infections*.) (Also see Chapter 7, Bioburden and Infection.)

Patient TeachingPreventing Staphylococcal or CA-MRSA Skin Infections

Practice Good Hygiene

- Keep hands clean by washing thoroughly with soap and water or using an alcohol-based hand sanitizer.
- Keep cuts and scrapes clean and covered with a bandage until healed.
- Avoid contact with other people's wounds or bandages.
- Avoid sharing personal items, such as towels, washcloths, razors, clothing, or uniforms, that may have come into contact with an infected wound or bandage. Wash soiled sheets, towels, and clothes with water and laundry detergent. Use a dryer to dry clothes completely.

For Patients Colonized with MRSA

- Mupirocin ointment may be administered to the nares to attempt to decolonize; it can also be applied to infected skin sites.
- Chlorhexidine 2% or 4% wash may be used to lower skin burden of colonization.

Bacillary Angiomatosis

Bacillary angiomatosis, a less common bacterial infection, is caused by organisms of the genus *Bartonella* (formerly *Rochalimaea*), specifically *Bartonella quintana* and *Bartonella henselae*. These cutaneous vascular lesions are characteristically small, reddish to purple papules that are tender to the touch. Lesions may ulcerate and then become covered by a crust. Complicated bacillary angiomatosis infections occur when the lesion is located deep in the subcutis, extending to involve soft tissue and bone. Infection with bacillary angiomatosis leads to systemic involvement. Biopsy followed by special staining is often necessary to definitively identify the organism. Treatment with erythromycin or doxycycline provides a prompt response. (See *Patient Teaching: Bacillary Angiomatosis*.)

Practice PointPractice Point

With the advent of ART, bacillary angiomatosis infections seem to have almost disappeared. However, these infections may mimic Kaposi's sarcoma, which should therefore remain the differential diagnosis until the actual causative agent is identified.

Noninfectious Skin Disorders

The immunocompromised status of patients with HIV and AIDS also puts them at greater risk for noninfectious skin disorders.

Pruritic Rash

Patient TeachingBacillary Angiomatosis

The most common reservoirs for the bacilli that cause bacillary angiomatosis are domestic cats and cat fleas. Clients with AIDS should avoid rough play with cats and situations in which scratches from cats are likely to occur. Cats shouldn't be allowed to lick open wounds or cuts. All cats should be treated for fleas, or other flea control measures should be followed.[15]

A rash that is predominantly papular may be due to pruritic papular eruption (PPE), eosinophilic folliculitis, nodular prurigo, drug reaction, syphilis, granuloma annulare, and atopic-like dermatitis.

PPE is often reported as the most common rash seen in HIV infection. The typical primary lesion is a firm, discrete, erythematous, urticarial papule. Anywhere from 18% to 46% of patients with HIV have this condition at some time.[16,17] PPE occurs in patients with a low CD4 count (<350 cells/mL) and usually in advanced HIV disease. It could be considered a marker for immunosupression.[18] Severe pruritus and subsequent scarred excoriations subject patients to HIV-related stigma. Topical or intralesional glucocorticoids are the treatment of choice. Other topical treatments, such as topical vitamin D_3 and topical capsaicin, have also been reported to be effective. Often, treatments provide minimal relief and can be disappointing.

Neoplastic Disorders

Patients with HIV and AIDS are at risk for a variety of neoplastic disorders. One of the most important risk factors for neoplastic disorders is immune suppression. Together, non-Hodgkin's lymphoma and Kaposi's sarcoma represent the majority of neoplastic disorders seen in patients with AIDS.

Lymphoma

Although lymphomas generally start in the lymph nodes or collections of lymphatic tissue in organs, such as the stomach or intestines, the skin may also be affected. Non-Hodgkin's lymphoma usually manifests as pink to

purplish papules or nodules. Deeply seated soft tissue involvement may expand superficially, forming dome-shaped nodules that often ulcerate. Cutaneous Hodgkin's disease appears similar to non-Hodgkin's lymphoma. The diagnosis is made by the identification of atypical cells having a Reed-Sternberg–like morphology. Treatments include methotrexate, prednisone, bleomycin, adriamycin, cyclophosphamide, and vincristine.

Kaposi's Sarcoma

Kaposi's sarcoma is a vascular neoplastic disorder. Prior to the use of ART, Kaposi's sarcoma was the most common skin disorder seen in men who have sex with men (MSM) with AIDS. The pathogenesis of Kaposi's sarcoma has now been identified as human herpes virus type 8. This virus is transmitted sexually, which explains in part the epidemiology of Kaposi's sarcoma predominantly in MSM.

Clinically, Kaposi's sarcoma skin lesions may be pink, red, brown, or purple macules, patches, plaques, nodules, or tumors and can appear almost anywhere on the body, including the mucous membranes (Fig. 21-1). The appearance of many cutaneous lesions typically predicts visceral organ involvement. When pressure-bearing areas such as the base of the spine are involved, lesions often ulcerate. Marked edema may develop when tumors involve the lymphatics, leading to diffuse swollen areas of the skin and subsequent breaks in the skin.

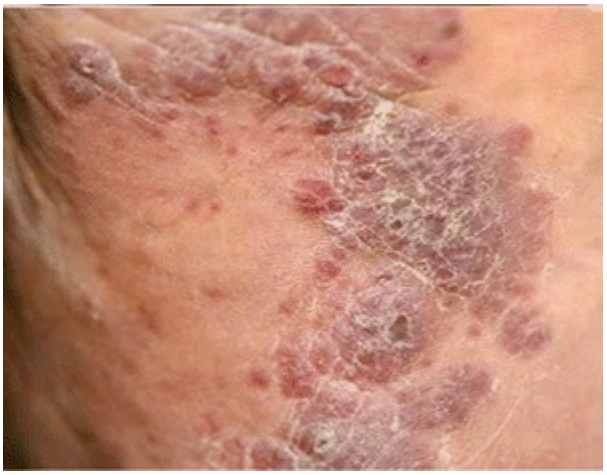

FIGURE 21-1 Kaposi's sarcoma on the sole of the foot.

Diagnosis of Kaposi's sarcoma is usually based on the finding of purplish skin lesions. Biopsy is rarely necessary but may be performed to rule out bacillary angiomatosis. ART is considered the first-line treatment

for Kaposi's sarcoma lesions, and when CD4+ cells improve, lesions tend to regress. Other treatments include radiotherapy, surgical excision, laser cryosurgery intralesioal injections, and topical treatments with cytotoxic drugs.

Squamous Cell Carcinoma of the Anal Mucosa

A growing body of evidence suggests a high prevalence of anal HPV infection and dysplasia in HIV-infected individuals. Among 4,506 HIV-infected males with 37,806 person-years (PY) of follow-up, anal cancer rates (per 100,000 PY) increased fivefold, from 11 in the pre-HAART to 55 in the HAART era. Rates continued to increase, reaching 128 in 2006 to 2008.[19,20] During the ART era, the risk of anal cancer among MSM was nearly 100-fold higher than in the general male population. The risk was about 50-fold higher in other men and 13-fold higher in women relative to the general female population.[20] Receptive anal intercourse may increase the likelihood of anal HPV infection but is not a prerequisite for anal HPV or dysplasia. Patients with lower CD4+ cell counts appear to be at higher risk of developing anal dysplasia. Tumors in the anal area typically present as a mass associated with bleeding and pain. Anal neoplasia must be ruled out in anyone with HIV infection who presents with anorectal complaints. Investigation of any abnormal discharge, bleeding, bowel irregularity, pruritus, dysuria, or pelvic pain should include screening for anal neoplasia. ART and immune reconstitution do not offer protection against anal dysplasia.[19,20]

Anal cancer is an important and increasing cause of morbidity and mortality in HIV-infected persons particularly HIV+ MSM. Screening by anal Paps can identify abnormalities that are probably precursors to anal cancer; however, routine anal cytology screening has not yet been recommended.

Summary

Skin disorders are common in patients with HIV and AIDS. Accurate identification of skin lesions is critical so appropriate treatment can be implemented. Consultation with an HIV or AIDS clinician is helpful in the comprehensive care of these patients.

PATIENT SCENARIO: HIV/AIDS POPULATION

Clinical Data

PK is a 23-year-old black man who was diagnosed with HIV 5 years ago. He is seeing his nurse practitioner (NP) today for his 3-month HIV visit, which includes an assessment of his viral load and CD4+ cell count. At his last visit, PK's viral load was below the limits of detection (<48 copies) and his CD4+ cell count was 653 cell/mm^3. He is 100% adherent with his HIV medications and tells the NP that he is feeling well overall. Since this is also his annual exam, the NP performs a complete physical examination as well as an anal Pap smear. There are no visible lesions around the anus, and PK has no anorectal complaints. Several days later, the NP receives the results of PK's anal Pap, which reveals low-grade squamous intraepithelial lesions (LSIL).

Case Discussion

Anal cancer, like cervical cancer, is a member of a broader group of anogenital cancers known to be associated with sexually transmitted viral HPV infection. HPV is extremely prevalent, particularly in young, sexually active populations. Sexual practices involving receptive anal intercourse lead to a significantly elevated risk for anal dysplasia and cancer, particularly in those with HIV/AIDS. Unlike cervical cancer, there are no universally accepted guidelines or standards of care for anal dysplasia. Expert opinion suggests that annual testing be performed, particularly in high-risk groups. An anal Pap screening involves the blind insertion of a swab into the anal canal and fixing the retrieved cells either on a slide or in fluid for cytological examination.

With the finding of dysplasia, PK will need to be referred for an anoscopic examination and biopsy, the procedure for which is as follows. After an initial application of acetic acid, Lugol's iodine solution is applied. Then, an anoscope, a high-resolution microscope, is used to inspect visually the entire anal canal, particularly the transformation zone, an area of increased risk for dysplastic changes. High-grade lesions do not take up the iodine solution because of the

lack of glycogen in the dysplastic cells; they appear yellow to tan, whereas normal or low-grade lesions appear dark brown or black. Any abnormalities such as acetowhitening (a temporary change to a white color when acetic acid is applied topically), papillation (raised bumps) and ulceration, or irregular surface changes noted in the inspection are biopsied. The classification system for anal cytology similarly includes normal or atypical squamous cells of undetermined significance (ASCUS). Atypical findings are further classified as low-grade (LSIL) or high-grade (HLSL) dysplasia. PK's lesions were LSIL. For PK's small, localized lesions, treatment included application of trichloroacetic acid but could have included various ablative therapies with lasers, infrared coagulation, or cryosurgery. Surgical excision is generally reserved for deeper or more diffusely spread lesions.

BARIATRIC POPULATION

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Sharon Baranoski, MSN, RN, CWCN, APN-CCNS, MAPWCA, FAAN

Objectives

After completing this chapter, you'll be able to:

- discuss bariatric and obesity healthcare facility concerns
- identify skin problems commonly found in the bariatric patient
- discuss unique risk factors and pressure ulcer prevention strategies for the bariatric patient.

An Important Healthcare Concern

The number of larger, heavier individuals in the United States and globally continues to increase, as does the need to provide reasonable accommodation for the bariatric patient population across healthcare practice settings.[1] More than two-third of all individuals living in the United States are overweight or obese, and 3% to 10% (at least eight million) are morbidly obese, many of which are children.[2] Morbid obesity, once a rare occurrence in America, has essentially quadrupled since the

1980s.[3] Research also shows that the heaviest Americans have become even heavier in the past decade.[4] Studies suggest a substantial increase in obesity among all age, racial, ethnic, and socioeconomic groups.[5] Worldwide, the number of individuals who are overweight or obese, which totals nearly two billion, now exceeds the number of those suffering from starvation.[6,7]

Bariatrics is a relatively new and general term derived from the Greek word *baros* and refers to the practice of health care relating to the treatment of obesity and associated conditions.[8] The American Society for Metabolic and Bariatric Surgery (ASMBS) defines the term obesity as the lifelong, progressive, life-threatening, genetically related multifactorial disease of excess fat storage with multiple comorbidities.[9] The National Association to Advance Fat Acceptance (NAAFA) does not consider a person with a high degree of adiposity obese. The association explains that this disapproving term medicalizes a very natural condition. NAAFA reminds us that some people are born tall, some short, some skinny, and ultimately some fat, "We are tired of being labeled in a negative light simply for our God-given habitus."[10] Regardless, obesity, according to the NIH, is simply a diagnostic category that represents a complex and multifactorial condition.[11]

Hospitals and other healthcare organizations best serve obese patients when size-sensitive policies, equipment, training, and support are in place.[12] This level of preplanning for care is designed to better manage the risk associated with the common, predictable, and preventable consequences of caring for this complex patient.

Classifying the Bariatric Patient

There are a number of ways to measure and define the condition of obesity. In the healthcare setting, some recognized methods include the following: body weight, body mass index (BMI), body weight maldistribution, weight-related immobility, or some other factors that interfere with the workers ability to provide care. Body mass index is a mathematical formula that assigns relative risk for morbidity and mortality and is often used in healthcare settings to determine the need for special accommodation simply because of this relative risk. The National Heart, Lung, and Blood Institute (NHLBI) provides the following classifications for obesity based on BMI, which is calculated as body weight in kilograms/body height in meters

squared.[13,14]

- Overweight: BMI 25 to 29.9
- Class I obesity: BMI 30.0 to 34.9
- Class II obesity: BMI 35.0 to 39.9
- Class III (extreme) obesity: BMI over 40

Body weight maldistribution can be a factor in skin assessment, mobility, and risk for pressure ulcer development or other skin impairment. Individuals who carry their excess weight in the hips and legs are often referred to as having a "pear-shaped" body maldistribution. This condition places the individual at risk for pressure formation over the buttocks area. An "apple-shaped" maldistribution can lead to respiratory challenges as this individual carries their weight predominantly over the upper body.[1] Further, the patient with this upper body weight maldistribution may be at risk for developing skin breakdown if a tracheostomy tube is in place, simply because of the excess tissue in and around the submental region (Fig. 21-2).

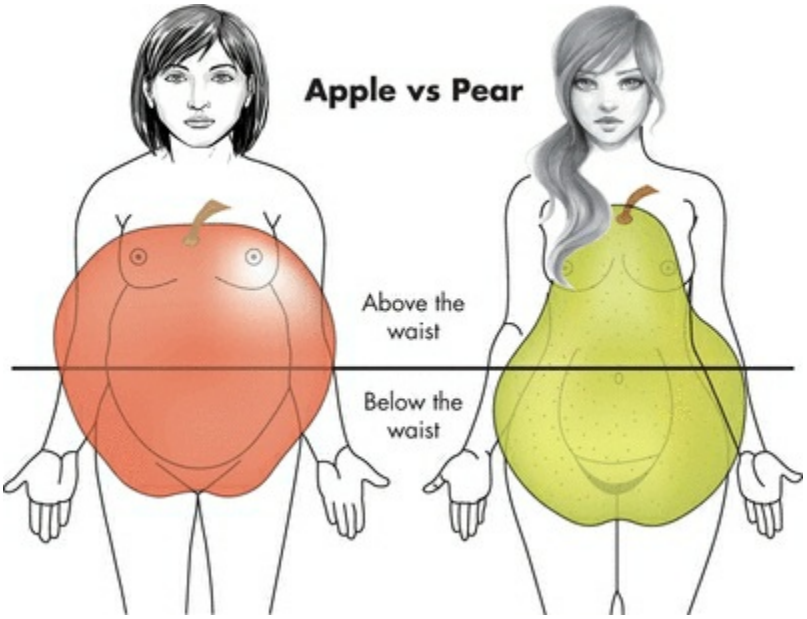


FIGURE 21-2 Identifying body shapes. The waist-to-hip ratio (WHR) is often used to describe the differences between the two types of body shapes: apple shaped and pear shaped (as shown in these illustrations). The apple-shaped physique is associated with hypertension.

The waist-to-hip circumference ratio is often used to describe these differences. The patient with an apple-shaped physique has a greater waist-

to-hip ratio and is at greater risk for cardiovascular disease. The pear-shaped individual has a lower ratio but may need special chairs and commodes to accommodate a larger hip size.[1] Bariatric patients run a high risk of developing pressure ulcers due to their body weight.

Meeting Patient Needs

With ever-increasing obesity rates in the United States, all healthcare facilities need to have bariatric skin care protocols in place and "ready access" to beds, chairs, commodes, walkers, and other equipment appropriate for bariatric patients. Advanced preparation with regard to protocols and equipment is essential to meet the needs of these patients. Bariatric patients frequently have significant healthcare problems, such as hypertension, type 2 diabetes, coronary artery disease, stroke, gallbladder disease, osteoarthritis, sleep apnea, respiratory problems, an increased risk for certain cancers, and numerous skin manifestations.[1] Despite these risks, bariatric patients may delay medical treatment out of fear or embarrassment. Ask yourself these questions as you develop your institutional readiness:

- Is your facility prepared to handle the special needs of bariatric patients as they enter the emergency department?
- Are your staff members prepared to provide safe, respectful patient care while avoiding injuries in both patients and staff members?

Many complex considerations are involved in treating bariatric patients, including multiple comorbid conditions; the potential for nonhealing surgical wounds due to dehiscence or infection; a higher risk of venous disease, pressure ulcer development, and diabetic foot wounds; and nutritional concerns. The focus of our discussion is skin assessment and care of the bariatric patient, with the goal of maintaining skin integrity.

Skin Assessment

A complete skin assessment is the first step in any skin care protocol. A cursory assessment of the skin is not sufficient. Bariatric patients may have changes in skin physiology due to a greater skin-to-weight ratio, redundant tissue, and poorly perfused adipose tissue that can result in compromised wound healing. Skin inspection should be performed on admission with the patient wearing a hospital gown and lying flat, if condition tolerates, in bed. All skin folds should be separated gently and examined for erythematous,

moist denuded areas or pressure ulcers. These areas can be extremely painful, so care and assistance (two or more persons) are needed to complete a thorough exam. Common skin fold areas include behind the neck, under the arms and breasts, under the abdomen or pannus, the flank area, the perineal and rectal area, the upper and lower thighs, and the calf and ankle areas[15] (Fig. 21-3).

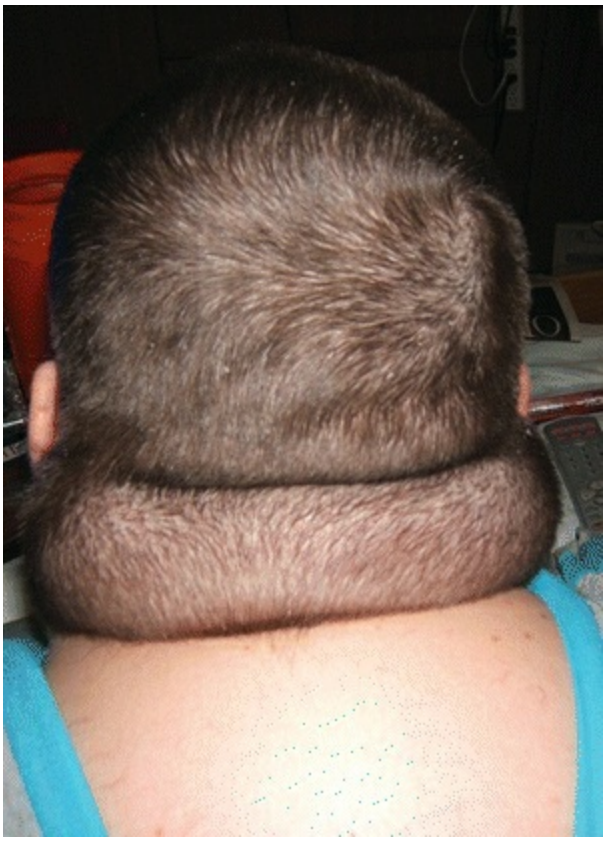


FIGURE 21-3 Skin folds on the back of the neck.

The presence of abdominal fat with an accompanying skin fold is referred to as a panniculus (pannus) (Fig. 21-4). Grading the extent of the redundant has been helpful in assessment, planning for care, and certain reimbursement issues as they relate to targeted intervention. A scale of grades 1 to 5 was cited in the literature as early as 2000 and has been adopted by a number of groups including the American Society of Plastic Surgery.[15,16]

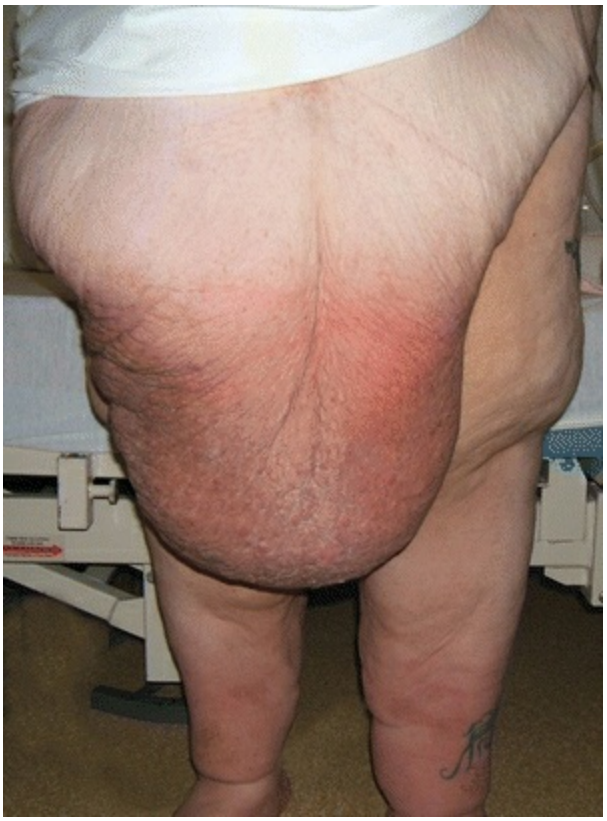


FIGURE 21-4 Grade 5 abdominal pannus.
(Photo copyright 2006 Coloplast Corp. Used with permission. Photographer: K. L. Kennedy-Evans, RN, CS, FNP.)

- Grade 1: Pannus covers the pubic hairline but not the entire mons pubis.
- Grade 2: Pannus extends to cover the entire mons pubis.
- Grade 3: Pannus extends to cover the upper thigh.
- Grade 4: Pannus extends to midthigh.
- Grade 5: Pannus extends to the knee and below.

Forces such as the pressures between skin folds and moisture accumulation, as an aggravating factor, can lead to deep tissue injury, pressure ulcers, or intertrigo (Fig. 21-5). Ongoing skin assessment and appropriate interventions are critical in maintaining and healing any skin manifestations or ulcers. The NPUAP/EPUAP Pressure Ulcer Prevention and Treatment Guideline for Bariatric (Obese) Individuals provides assessment and positioning recommendations.[17] (See *Assessment and positioning recommendations for bariatric patients*.)

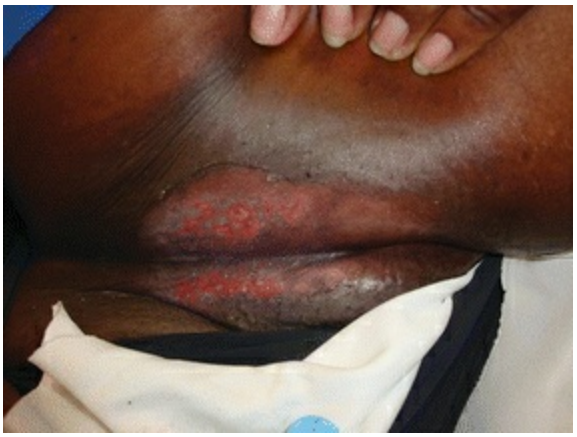


FIGURE 21-5 Grade 5 pannus with intertrigo.
(Photo courtesy of C. Fife, MD.)

When examining the skin, you may find certain benign conditions that more commonly occur in obese individuals. These include the following:

- Acanthosis nigricans: hyperpigmented (often brown) irregular plaques in skin folds (especially the axilla and back of the neck) (Fig. 21-6). The plaques often feel like velvet when touched but may eventually become rougher in texture. This condition is associated with obesity as well as other systemic diseases, such as insulin resistance, diabetes, hormonal changes such as hypothyroidism, cancer, and more. In many situations, treating the underlying problem can help fade the discoloration. Examples may include weight loss, medication, or surgery to address the underlying cause. If the skin surface becomes aesthetically problematic or uncomfortable for the patient, consider prescription creams to lighten the area, antibacterial soaps to reduce odor, or laser therapy to reduce the thickness of the skin surface.[18]
- Plantar hyperkeratosis—excessive weight on the feet leads to thickening of the weight-bearing surfaces on the soles (Fig. 21-7). Treatment includes weight loss and orthotic shoe inserts to protect bony prominences from breakdown.

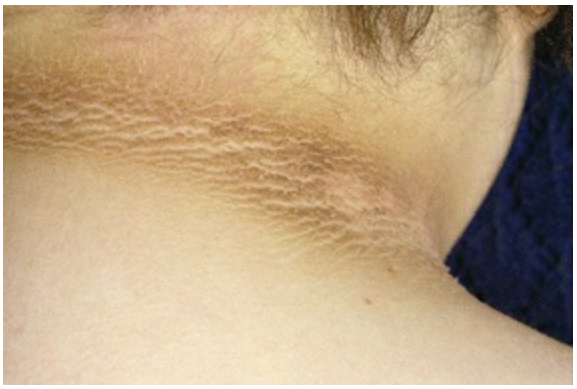


FIGURE 21-6 Acanthosis nigricans. This photo shows a patient with brown velvety hyperpigmentation of the skin often seen in bariatric patients.

Assessment and Positioning Recommendations for Bariatric Patients[1,17]

- Get adequate assistance to fully inspect all skin folds.
- Atypical pressure ulcers may develop over the buttocks, buttocks cleft, or other areas because of maldistribution of weight as well as within skin folds and in locations where tubes and catheters burrow into soft tissue.
- Consider slings and bands used with a ceiling or floor-based lift for patient repositioning; consider limb or pannus slings to safely access hard-to-reach areas.
- Use pillows or other positioning devices to off-load pannus or other large skin folds and prevent skin-on-skin pressure.

More serious skin problems are often found during the initial assessment of obese patients, such as cellulitis; macules, papules, or pustules especially to the neck and back, lymphedema; hemosiderosis and other skin changes associated with venous insufficiency of the lower extremities; venous ulcers; dehiscence of surgical wounds; intertrigo; and pressure ulcers.[19] Many of these conditions are covered in other chapters of this book. Here, we give special consideration to the prevention and treatment of intertrigo and pressure ulcers specifically as they relate to bariatric patients.

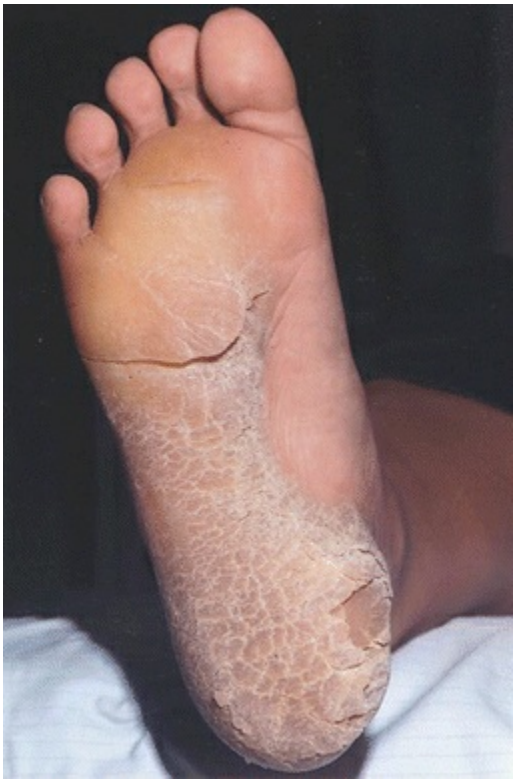


FIGURE 21-7 Plantar hyperkeratosis. Hyperkeratosis results from chronic excessive pressure or friction to the epidermis, as seen here on the plantar aspect of the foot.

Intertrigo

Intertriginous dermatitis is one of four types of moisture-associated skin damage and is commonly referred to as intertrigo and results from moisture trapped within skin folds. Intertrigo may occur in the presence of bacterial, fungal, or viral involvement and is simply inflammation of the skin folds. These inflammatory changes often present as mirror images where one skin surface touches another. Although this condition can be found in any patient, obese patients are at greater risk because of the nature of skin folds that accompany an obese habitus. For example, Brown and colleagues[20] surveyed 100 obese patients for the prevalence of skin problems, including rash, dryness, and "broken skin." Of the respondents, 63% indicated that they had more than one skin problem, the most common being broken skin and itching.[20] Aggravating factors that impact the frequency and severity of intertriginous dermatitis among the obese individual include moisture such as perspiration, urine, feces, mucus, or wound drainage, which becomes trapped under or within skin folds, creating maceration; pressure from large skin folds on underlying skin, creating areas of pressure-induced injury;

friction as one skin surface moves across another; shear with movement resulting in fissures at the base of the skin fold; physical challenges in maintaining hygiene; and the warm, dark, moist conditions that favor microbial factors. Cellulitis becomes a risk if the condition is not treated[21] (Fig. 21-8).

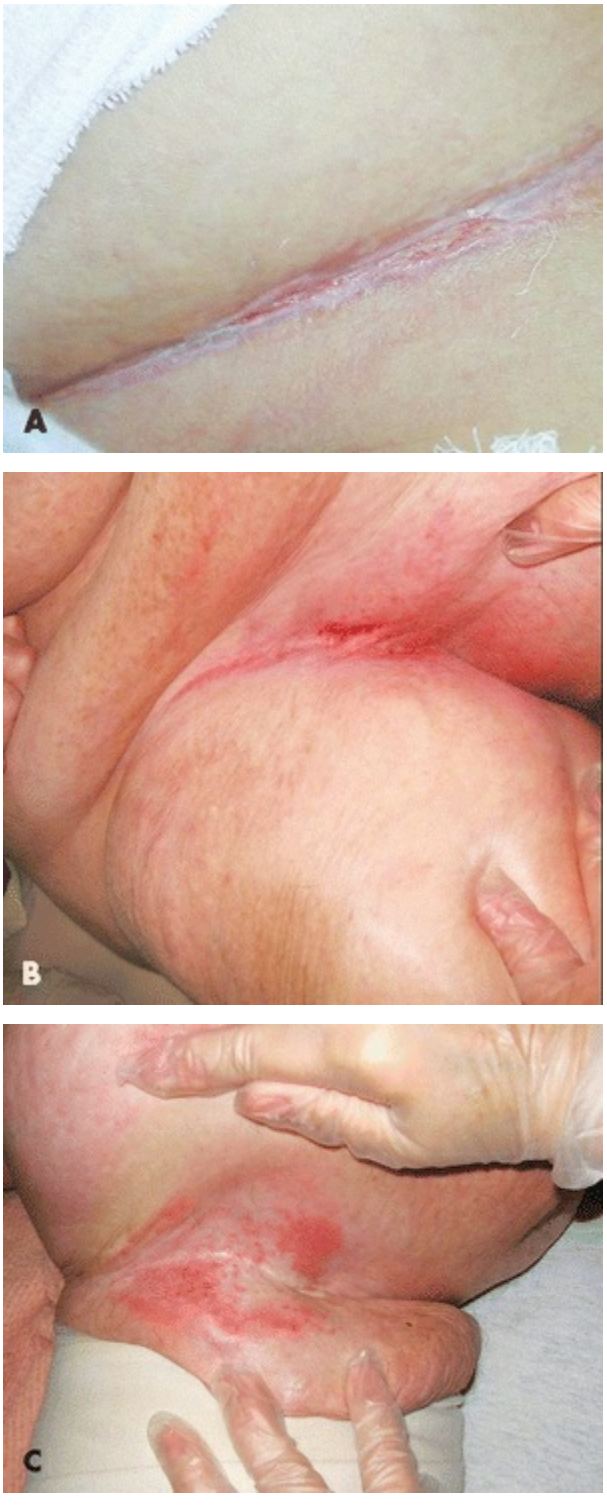


FIGURE 21-8 Intertrigo. **(A)** Intertrigo between the skin folds of the breast of a

female patient with a body mass index of 60. Note the fissure at the base of the skin fold. **(B)** Intertrigo with erythema, erosion, and denudation. (Photo courtesy of K. L. Kennedy.) **(C)** Candida intertrigo in a skin fold. (Photo courtesy of K. L. Kennedy.)

Intertrigo can develop in any skin fold but is most common under the breasts, abdominal skin folds (pannus), and axillae as well as in the submaxillary area and groin or perineum. Obese patients may report a history of skin irritation under skin folds. Confounding factors, such as bed rest, immobility, fever, and the use of such medications as antibiotics and steroids, often increase the risk of recurrence once the patient is hospitalized.

Preventing Intertrigo

Prevention of intertrigo is a key component of any bariatric skin care protocol. Preventive interventions focus on keeping the skin clean, dry, and well supported and minimizing the effects of moisture, pressure, friction, and shear. Use a gentle soap or no-rinse skin cleanser for bathing. Pat skin dry with a soft cloth. Moisture accumulation between skin folds is an ongoing problem between bathing. Soft absorbent pads, such as soft linen or nonocclusive high-air-flow incontinence pads, can be placed between skin folds to off-load pressure, absorb moisture, and lessen friction and shear with movement. Some facilities have had success with a silver impregnated fabric, which is designed to be placed in the skin folds of bariatric patients (Fig. 21-9). A properly fitted nonsynthetic brassiere may also help achieve these goals in large-breasted women. Any material placed between the skin folds should be changed frequently.

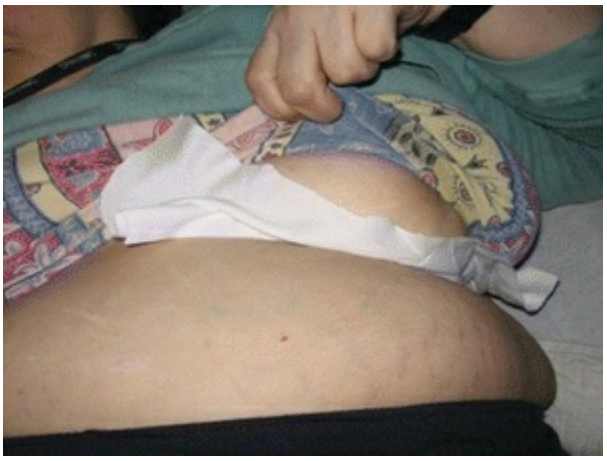


FIGURE 21-9 InterDry, a product used to prevent intertrigo in bariatric patients.

When repositioning bariatric patients in bed, make sure that the pannus is well supported with a pillow and separate the legs by placing a pillow between the knees to make sure there is adequate airflow to skin fold areas. Low-air-loss beds may help dry moist areas.

Treating Intertrigo

A wide variety of products have been used to treat intertrigo; however, the most effective treatments are based on an analysis of the underlying etiology. *Candida* should be treated with topical antifungal products; systemic antifungal agents such as fluconazole may be necessary in severe cases. Likewise, bacterial infections of the skin may be treated with topical antibiotics or, if not improving or if progressing to cellulitis, systemic antibiotics. Topical and systemic steroids may be useful in cases of atopic or contact dermatitis.[22] Consider burrow solution (aluminum acetate) soak applied for 15 to 20 minutes twice per day to soothe and dry affected area.[12]

Unfortunately, intertrigo plagues obese patients especially after weight loss because of the extent of redundant skin. Panniculectomy and other forms of recontouring surgery may be necessary to remove excessive skin folds after weight loss has been achieved.[15]

Pressure Ulcers

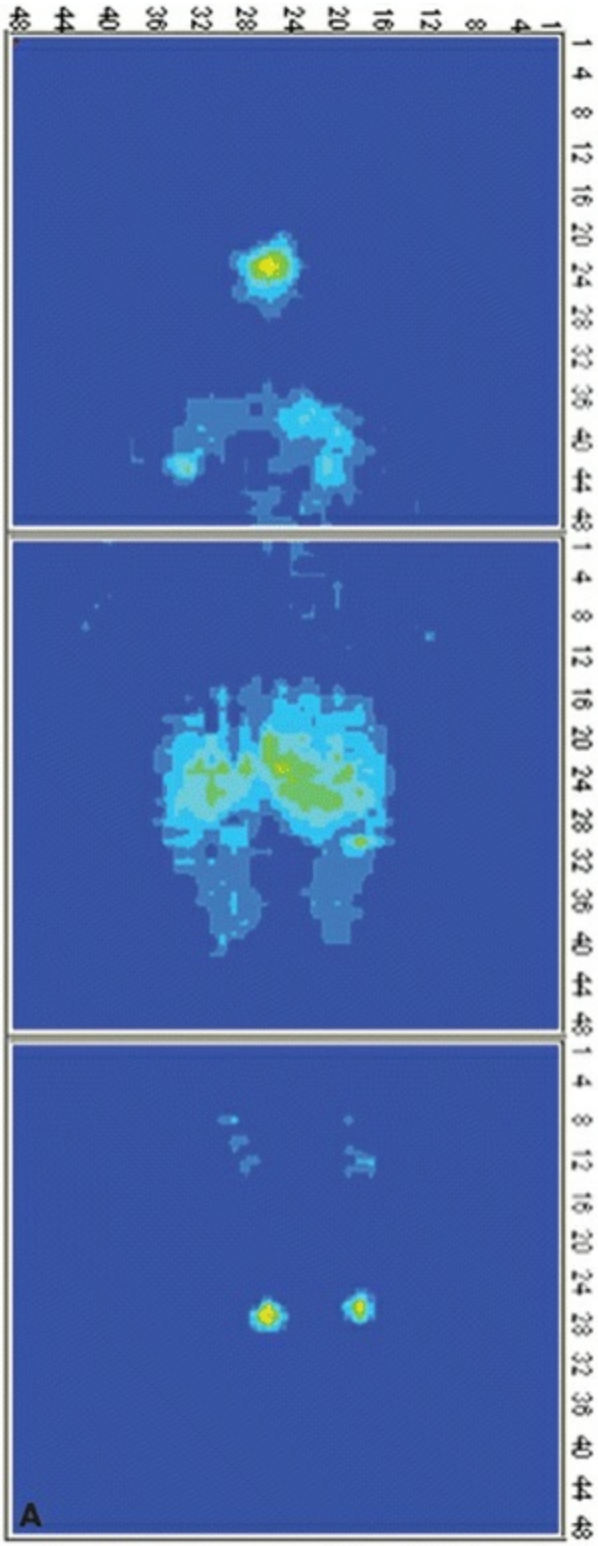
Prevention

Pressure ulcer prevention is discussed in Chapter 13. This discussion focuses on the unique risk factors and needs of the bariatric patient in preventing pressure ulcers.

Unique Risk Factors

Several factors increase pressure ulcer risk in the bariatric patient. Pressure mapping studies indicate that pressure is distributed somewhat differently in obese patients. In patients of normal weight, high-pressure areas in the supine position are predominantly over bony prominences (e.g., the head, sacrum, and heels). In the obese patient, a large amount of weight-induced force is distributed over the entire supine surface. Pressures may be high over bony prominences, but high-pressure areas are also seen in

traditionally soft tissue areas such as the buttocks. Even though the surface area is larger in an obese patient, there is still often greater tissue weight than normal on traditionally soft tissue areas (Fig. 21-10).



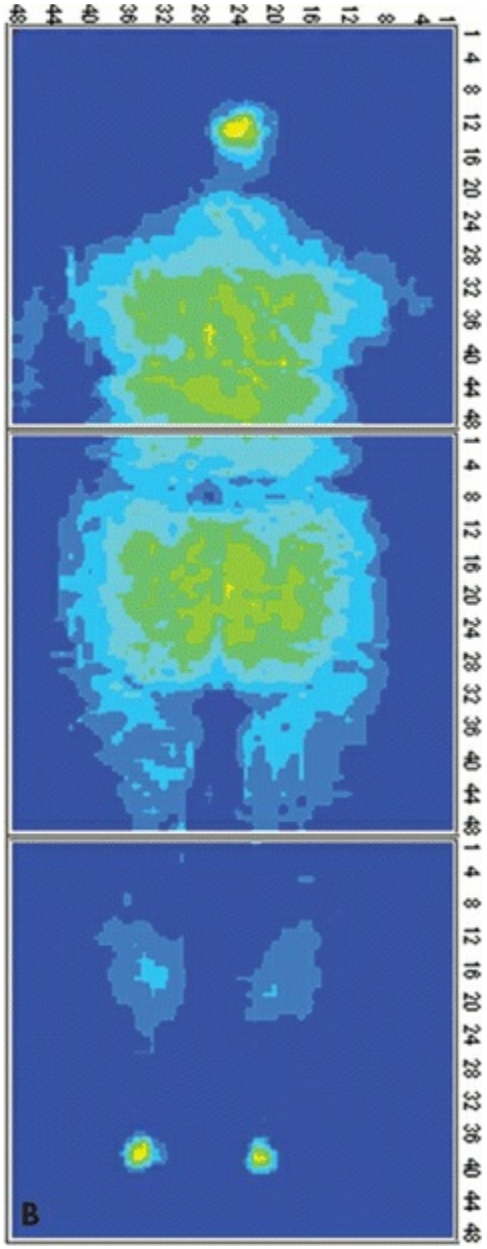


FIGURE 21-10 Pressure distribution. **(A)** High-pressure areas are more focused over bony prominences in patients with a lower body mass index (BMI). **(B)** A more diffuse pattern of high-pressure distribution is evident in the patient with a higher BMI. High-pressure areas occur over soft tissue areas as well as bony prominences. Note enlarged yellow area indicating more pressure in the buttock region.

Assessment

Pressure ulcers that do develop in obese patients often have slightly different characteristics. They develop not only over bony prominences but also over other areas of high tissue pressure, such as the buttocks. (See Fig. 21-10.) Bilateral hip ulcers are commonly seen in patients placed in chairs

that are too narrow for their hip size.[6] Bariatric patients seem to be at higher risk for pressure damage related to medical devices (such as tubes, oxygen tubing, endotracheal tubes, oximetry probes, and tight narrow tracheostomy ties).

Standard risk assessment tools such as the Braden scale are still important as general screening measures for pressure ulcer risk status. However, consider exploring each subscale more fully when caring for an obese patient. Many of these pressure ulcers can be prevented with proper risk assessment, selection of bariatric-sized equipment technology and devices, proper application of same, and frequent inspection of the skin for areas at risk for pressure-related damage or other tissue damage.[23]

Deep Tissue Injury

There is some clinical speculation that suspected deep tissue injury (sDTI) may also be more common in obese patients. Deep pressure may predispose the patient to deep injury. When assessing the skin of bariatric patients, check carefully for subtle signs of sDTI, such as slight discoloration of the skin, and changes in skin temperature and texture. If deep tissue injury is suspected, off-load the affected area in an attempt to reduce pressure and salvage any tissue that is injured but not ischemic.[23]

Consider the following questions when planning care for your bariatric patients:

- For patients on bed rest, what positioning strategies will improve respiration without increasing the risk of shear injury?
- Can pillows be placed under the arms to prevent sliding and shear? Would a trapeze over the bed help with patient mobility?
- Is the bed wide enough for the patient to turn comfortably from side to side?
- What is the patient's nutritional status?
- If incontinence-associated dermatitis is a risk, could it be improved by providing a bariatric bedside commode or walker to ambulate to the bathroom? Is a properly sized lift and sling available to transfer the immobile patient? Is a sit-to-stand or other mobility device available to promote safe transfer to a bedside commode?

Bariatric Equipment

Planning care for the bariatric patient population brings with it many

specific concerns, which need to be addressed in order to provide care that is safe and effective and addresses the unique needs of larger, heavier patients. Areas of particular concern include safety, both for the patient and the caregiver, accessibility for the patient, sensitivity of all caregivers and staff members, use of specialized technology and devices that will work for larger patients, and sufficient staffing.[23]

Ensuring a facility's readiness to provide for patient safety and comfort can be assessed by performing a survey of the facility, paying specific attention to weight limits of equipment, including chairs, beds, commodes, shower seats, physical therapy tables, lift/transfer equipment, carts, exam tables, wheelchairs, walkers, CT scanner, MRI scanner, OR table, and any other equipment needed to provide care for any patient. All equipment should be labeled in a way making it easier for staff to be aware of weight restrictions, while not publicizing that it is "bariatric equipment." Subtle methods include "EC 500" or "U500" for extended capacity to 500 pounds or under 500 pounds. Check to see if toilets are floor or wall mounted. Standard wall-mounted toilets can be adapted to accommodate bariatric patients by using a relatively inexpensive support that is available commercially. Additionally, it is necessary to look at the width of doorways, to assure larger equipment will fit into and out of the room. There should be room for a patient to ambulate through the doorway with a larger walker and with a healthcare professional on either side for stability, since this may be what is needed for safety.[4] Consider the width and length and weight limits of elevators.[24]

Larger sized personal items such as gowns, robes, slippers, identification bands, blood pressure cuffs, antiembolus stockings, sequential compression devices, and even gait belts need to be available for patients so that they can be cared for with dignity and safety. Just as we would not put a standard-sized gown on a pediatric patient, it is not acceptable to snap together two standard gowns to fit around an obese patient.[24]

When selecting a bariatric bed frame, some basic criteria should be considered. Ask yourself these questions[24]:

- Is the bed frame wide enough? Does the bed frame have the capacity to expand and reduce the width to meet the patient's clinical care needs?
- Can the patient turn and reposition himself in the bed? For example, confirm that the bariatric patient's girth does not touch the side rails of the bed when the patient is turned from side to side.
- What is the bed frame's weight capacity? Does the patient have general

obesity or a maldistribution that influences the ability to raise and lower the foot or head feature?
- Is a trapeze (or other supportive structure) available so that the patient can assist with turning?
- Does the bed have features to assist with moisture control if indicated? Consider using features that provide airflow over the surface of the skin to facilitate fluid evaporation if the skin is excessively moist.
- Is the bed designed for easy patient egress to help facilitate early progressive mobility?
- Are wheelchairs and chairs in the room wide enough to accommodate the individual's girth?

Staff Concerns

The American Nurses Association recently published the Safe Patient Handling and Mobility (SPHM) Interprofessional National Standards[25]; shortly thereafter, the Implementation Guide to the Standards was published.[26] The interest in national and state legislation is driving worker safety initiatives. The goal of SPHM is to improve patient safety while preventing caregiver injury. It was not until recently that science helped caregivers and consumers understand the risks of manual handling in the patient care areas.[27] This concern is increasingly more significant when caring for the bariatric individual.[28] To that end, worker safety raises a number of relevant questions. For example, can staff members transport the patient in the bed? Does the bed fit through the doors throughout the entire hospital? Can the bed be steered easily? Does the bed have a power drive feature? Are bariatric-sized lifts and air transfer devices available? Have staff members been properly trained to use bariatric equipment to ensure both staff and patient safety? Eliminating the fear of injury while providing care requires education of the staff with regard to proper lifting and handling techniques as well as available equipment. It should be a part of an organization's educational program to fully prepare all patient care staff on safe handling of the bariatric patient. This will prevent injuries and provide a high level of care that diminishes tissue damage and reduces the inherent risks associated with bariatric patients.[29]

Repositioning and Care

Repositioning the bariatric patient is both an art and a science. Routine turning should be accomplished by a team of trained caregivers in a manner

that preserves the patient's dignity. When possible, enlist the patient's help in turning. Patients and their families often have very useful suggestions for achieving and maintaining mobility despite challenges. The frequency of repositioning should be based on the individual's ability to tolerate turning and repositioning. Bariatric patients who cannot reposition themselves should be assessed frequently and repositioned as often as needed using universally recognized protocols or algorithms.[1]

When repositioning bariatric patients in bed, several strategies are particularly important. Be sure to off-load bony prominences. Rather than repositioning pillows under the patient's shoulders and hips, the patient should be turned sufficiently to avoid areas of potential pressure. Proper technology (equipment and devices) may be required to prevent friction and shearing injuries when repositioning. Grabbing and pulling on a patient may cause skin tears as well as shoulder injuries.[30] As far back as 1995, Oertwhich and colleagues[31] suggested that small shifts in body weight may be effective in promoting skin health. However, small shifts are intended as an adjunct, not a replacement, to regular repositioning in high-risk patients.

Off-Loading Heels

Because of compromised circulation, heels may pose a significant risk to skin health. Heels should be floated off the surface of the bed by placing pillows under the calf or with the use of positioning devices. Examine the unique anatomy of your patient's lower extremities. Patients with large calves may be naturally suspending their heels off the surface of the bed. Feel under the heel and leg to note any high-pressure areas that can be relieved with properly placed pillows that redistribute weight. When using heel positioning devices, make sure they are properly sized for your patient and can be applied without creating high-pressure areas under straps or other areas of the device.

Summary

The prevalence of obesity continues to climb in the United States and worldwide. An increasing number of bariatric patients are admitted to hospitals each day. Caregivers best serve patients when they understand the unique and complex challenges associated with safe, quality, sensitive patient care. Preplanning for care is the first step to success, along with targeted assessment, policies, and procedures, size-appropriate technology,

training, and outcomes measurement.[32]

Show What You Know: Spinal Cord Injury Population

1. All of the following are major risk factors for pressure ulcer development in the SCI population except:

A. preexisting conditions.
B. severity of the SCI.
C. gender.
D. nutrition.

2. The most commonly occurring complications of SCI are:

A. fracture.
B. urinary tract infection.
C. pressure ulcer development.
D. pulmonary/respiratory complications.

3. A pressure ulcer prevention plan for a person with SCI should always include the following:

A. Every 2-hour turning and repositioning schedule
B. Bowel management regimen with padded commode seat
C. Off-loading from the wheelchair every 30 minutes for at least 1 to 2 minutes
D. B and C only

Show What You Know: HIV/AIDS Population

1. Antiretroviral therapy (ART) has impacted skin disorders in patients with human immunodeficiency virus (HIV) in which one of the following ways?

A. Adverse effects of ART have led to an increase in the number of skin

disorders seen in the patient infected with human immunodeficiency virus.

B. There has been a decrease in the incidence of skin disorders seen in the patient with HIV.

C. There has been an increase in the number of noninfectious skin disorders in the patient with HIV.

D. Viral infections are the only skin disorders affected by HAART.

2. Community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) infection can best be prevented by teaching patients to:

A. take antiretroviral drugs every day.

B. wipe surfaces with alcohol-based sanitizers.

C. share only visibly clean items.

D. keep the hands clean.

3. What is the causative agent for dysplastic changes in the anal canal?

A. HIV

B. Hepatitis B virus (HBV)

C. Human papillomavirus (HPV)

D. CA-MRSA

Show What You Know: Bariatric Population

1. Mirror image inflammation in the skin folds of the obese patient is called:

A. a skin tag.

B. stretch marks.

C. intertrigo.

D. hyperkeratosis.

2. Acanthosis nigricans is always a benign condition for which there is no treatment.

A. True

B. False

3. **Proper positioning for the bariatric patient confined to bed includes:**
   A. using small shifts in body weight to replace turning.
   B. keeping the head of the bed flat at all times to prevent shear.
   C. avoiding pillows between the legs.
   D. supporting the pannus and other large skin folds.

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Pressure Ulcers in Neonatal and Pediatric Populations

22

Sandy Quigley, MSN, CPNP-PC, CWOCN

"We'd like to thank Mona Mylene Baharestani, PhD, ANP, CWON, CWS for her past contributions to this chapter."

Objectives

After completing this chapter, you will be able to:

- identify pediatric pressure ulcer risk assessment tools
- describe factors that place neonates and children at risk for pressure ulcers
- discuss disparities between adult patients, neonatal, and pediatric patients that may lead to potential challenges in providing skin and wound care.

Research-based publications related to neonatal and pediatric pressure ulcer development and management have historically been limited. In the past decade, there has been heightened awareness of this challenge in pediatric acute care patient populations. In addition, credentialing bodies such as Joint Commission and the American Nurses Credentialing Center (ANCC) Magnet Recognition Program have further highlighted the importance of identifying patients at risk for pressure ulcers and instituting early interventions to prevent pressure ulcer development. Therefore, clinical guidelines are often extrapolated from adult practice or based upon

consensus-based recommendations to guide best practices in preventing and treating pressure ulcers in neonates and pediatric patients across the age continuum. Pediatric pressure ulcers are considered a quality of care indicator and a largely preventable condition. Since 2011, a national network of children's hospitals called Children's Hospitals' Solutions for Patient Safety (SPS) and the Children's Hospital Association are collaborating to reduce harm by preventing specific hospital-acquired conditions. A goal by year-end 2014 is that the 78 SPS hospitals will achieve a 40% reduction in pressure ulcers. The pressure ulcer work groups[1] have developed strategies consistent with high reliability concepts by creating prevention care bundles to reduce harm and improve patient outcomes. This collaborative is currently collecting data on efficacy of the prevention bundles created, as well as monitoring effects on pressure ulcer prevalence.

Integumentary Development

Skin development is based upon gestational age. The premature neonate, at 24 weeks' gestation, has minimal stratum corneum and rete ridges are very thin and small. Premature neonates are born with red, wrinkled, translucent skin that is almost gelatinous in appearance. Subcutaneous tissue has not yet developed, so the dermal tissue (dermis) is lying over the muscle. As subcutaneous fat deposit begins to form (weeks 26 to 29), skin wrinkling starts to decrease. Maturity of the integumentary system is complete at approximately 33 weeks' gestation. As the infant continues to develop (40 weeks' gestation), the stratum corneum begins to increase in thickness. The skin remains extremely fragile and easily insulted due to developmental immaturity. Given that the skin continues to develop through the first year of life, the use of appropriate, evidenced-based skin care practices is important.[2] Many factors predispose hospitalized infants to skin injuries, including decreased epidermal–dermal cohesion, deficient stratum corneum, relatively alkaline pH of skin surface, impaired nutrition, and presence of multiple medical devices secured on the skin. Epidermal stripping, extravasation injuries, incontinence-associated dermatitis, and pressure ulcers are some of the most common injuries seen in the neonatal and pediatric population. Some authors include epithelial stripping from adhesive-based products as pressure ulcers while others do not, thus further confounding pressure ulcer benchmarking data.[3] This chapter focuses mainly on pressure ulcers that occur in a heterogeneous neonatal and

pediatric population.

Pressure Ulcer Prevalence and Incidence

Published pressure ulcer incidence and prevalence figures in the acute care pediatric population vary widely. Prevalence data are difficult to analyze and utilize as a benchmark due to considerable methodological limitations and insufficient level of reporting detail (i.e., patient ages, critical care vs. noncritical care, surgical vs. nonsurgical, traditional etiology vs. medical device–related, aggregate data are inconsistently reported by stage and body location). For example, a critically ill 9-year-old surgical patient may have significant edema from fluid volume resuscitation, have prolonged sensory impairment, as well as be hemodynamically unstable for the past 12 days impacting the ability to reposition frequently and develop an occipital and coccyx pressure ulcer. This patient's pressure ulcer may be very different from a 13-month-old admitted for 2 days with a respiratory infection who develops a pressure ulcer from a medical device such as a nasal cannula or oxygen mask. Furthermore, there are often incomplete or unclear descriptions of inclusion and exclusion criteria making it difficult to generalize and benchmark findings. Overall, pediatric pressure ulcer prevalence rates during the past decade have been reported as low as 2.3%,[4] 4%,[5] and 27.7%[6] and as high as 35%.[7] Reports of neonatal pressure ulcers vary from a low of 12%[8] and 16%[9] to a high of 31.2%,[10] although authors combined and included skin injuries as a result of pressure, friction, shear, and epithelial stripping injuries as one prevalence rate. Schindler et al.[11] reported on data analyzed from over 5,300 pediatric intensive care unit (PICU) patients with reported 10.2% pressure ulcer prevalence. Noonan et al.[12] reported a 27% in three PICUs and Schlüer et al.[13] reported 44% PICU prevalence. Prevalence rates of 20% have been reported in PICUs and neonatal intensive care units (NICU), respectively, with the majority of these ulcers occurring within 2 days of admission.[14–16] Schindler and colleagues[17] studied 372 pediatric ICU patients between the ages of 0 and 3 months and found an overall pressure ulcer incidence of 18.8%. There are no recent studies that have been published on pressure ulcer prevalence in acute care pediatric settings on patients with neurosensory impairment, spine deformities, and postural challenges such as muscle spasticity or flaccid paralysis as seen with cerebral palsy or myelomeningocele ("spina bifida"). However, it is important that clinicians

pay particular attention to this patient population due to frequent use of orthotics, wheelchairs, and other medical devices. Routine inspection for the proper fit of devices due to the normal trajectory of growth needs to be anticipated during childhood. This is particularly important during hospitalizations when they may have surgical procedures resulting in perioperative body temperature variability, hemodynamic changes, post-op positioning challenges, increased incontinence, and edema from fluid volume resuscitation.

Since 2009, there have been a number of national pediatric collaboratives, which have focused on the prevention and management of hospital-acquired pressure ulcers. Organizations such as Child Health Corporation of America (CHCA) and most recently Children's Hospitals' Solutions for Patient Safety (SPS),[1] which focuses on eliminating 11 hospital-acquired conditions and pressure ulcers, have heightened awareness of the prevalence of pressure ulcers in the acute care pediatric patient population. In addition, since 2005, Hill-Rom Corporation has included pediatrics in their annual International Pressure Ulcer Prevalence Survey. Unfortunately, these collaboratives privately disseminate information among the participating facilities and have not published their findings publicly and therefore are often not utilized as benchmark.

Anatomical Distribution

The occipital area is noted as being the most prevalent area for skin breakdown in children[18] (Figs. 22-1 and 22-2). Multiple authors have identified the occiput as the most common anatomical site for immobility-related pressure ulcer formation in patients from birth to age 3, as the head comprises a disproportionately higher percentage of the total body weight and surface area.[19–21] When lying supine, the occiput is the primary pressure point with the greatest interface pressure.[22] Curley and colleagues[23] report the ear, the occiput, and the nose are the common sites for pressure ulcer development in pediatric patients. The nose is at greatest risk for medical device–related pressure ulcers when noninvasive ventilation masks, such as when used with continuous positive airway pressure (CPAP), are in use.[24,25] Willock and colleagues[26] conducted a multicenter survey in 11 hospitals and report the most frequent site of pressure ulcer development was the sacrum or buttocks, heel, thigh, ear, occipital scalp, malleolus and spine. Kottner and colleagues[27] completed a

pediatric literature review and found the head, including occiput, ears, and neck were most often affected followed by heels and sacral and ischial areas. August et al.[3] studied 247 neonates and reported that the head region had a 35.5% of pressure injuries.

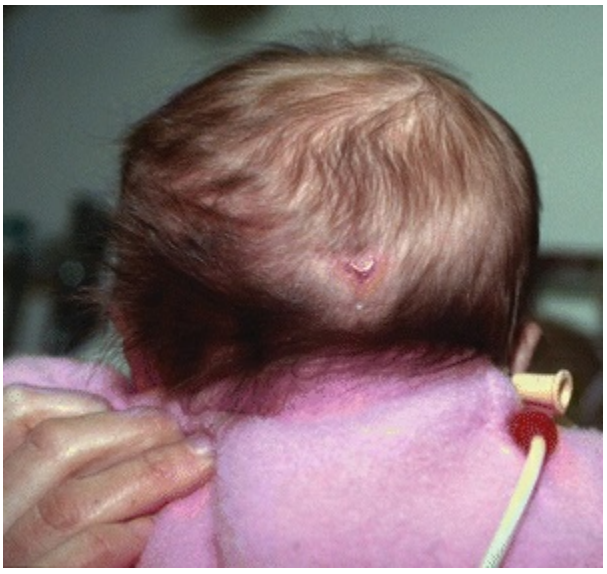

FIGURE 22-1. Stage II pressure ulcer on an infant's occiput due to immobility.

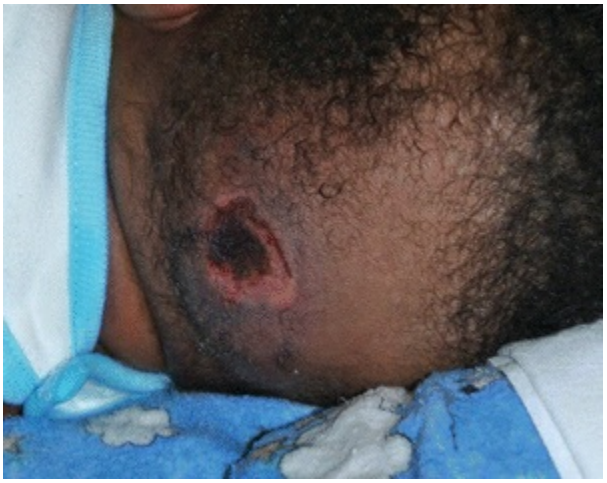

FIGURE 22-2. Unstageable pressure ulcer on an infant's occiput due to immobility.

Practice Point

The occiput is the most common site for immobility-related pressure ulcers in children from birth to age 3.

Risk Factors

Pressure ulcer development has traditionally been viewed as uncommon among neonatal and pediatric populations given the presumed relative ease of repositioning and frequency of movement.[28,29] However, as survival rates among critically and chronically ill premature neonates and children increase through technological advances, so too does the risk for pressure ulcer formation.[30] Infant skin has a higher absorption rate as compared with an adult's skin. This difference in the absorption rates also predisposes infants to a dry, flaky, and impaired skin barrier.[31] Premature neonates also have a higher ratio of skin surface area to weight with decreased or absent subcutaneous fat development based upon gestational age. Analogous to adults, critically ill and injured neonates and children may experience massive edema secondary to fluid resuscitation and hemodynamic, respiratory, renal, and neurologic instabilities that increase the risk for an unavoidable PU.[32] Their anatomically immature skin is further challenged by prolonged intensive care to manage respiratory inefficiency, inadequate tissue perfusion, fluid and electrolytes issues, poor nutrition, and thermal regulatory issues that further increases risk for negative physiological responses to repositioning and pressure ulcer development. Newborns may present with birth injuries such as a fractured clavicle, humerus, femur, skull, or rib(s). They also may be born with musculoskeletal abnormalities such as torticollis (wry neck) and congenital dislocation of the hips that impedes repositioning. Consequently, prolonged periods of immobilization may be maintained, especially among those on extracorporeal membrane oxygenation (ECMO) and high-frequency oscillatory ventilation (HFOV).[33–35]

Furthermore, pediatrics is the branch of medicine caring not only for infants and children but for adolescents as well. Among pediatric hospitals, there exist variable age-limit policies; therefore, a wide variety of patients are cared for, from neonates to middle-aged patients. This poses additional challenges for clinicians to maintain clinical expertise in a diversity of support surfaces and products to manage neonates to bariatric patients seen in their facilities.

Medical Equipment or Devices

Increasing numbers of critical access and monitoring lines/catheters and

other forms of medical equipment often limit safe turning and repositioning options and increase the risk for unavoidable PU.[36,37] In critical care environments, positioning of the head is often limited by internal and external jugular catheters to avoid inadvertent catheter dislodgement, head and neck edema, and air leakage around endotracheal tubes with movement. Medical devices and monitoring equipment secured to the skin or mucosal membranes and compressing tissue are now widely recognized as a risk factor for pressure ulcer development. This is particularly a concern in pediatrics as young active patients or those with developmental impairment do not cognitively understand not to touch or play with equipment; therefore, clinicians tend to very securely adhere devices to the skin to avoid inadvertent dislodgement. In fact, Willock and colleagues[26] found that 50% of neonatal and pediatric pressure ulcers were directly associated with equipment pressing on the skin. Boesch et al.[38] found, while medical devices are a known risk factor in adult populations, up to 75% of hospital acquired pressure ulcers (HAPUs) have been associated with medical device use among infants and children (Table 22-1). Critically ill patients typically require a multitude of invasive and noninvasive equipment anchored securely to their skin placing them at particularly high risk. Specific devices that may result in pressure ulcers are respiratory devices (endotracheal/oral tracheal tubes, tracheostomy tube, noninvasive ventilation mask, and headgear), casts and orthotics, cervical spine immobilizers, pulse oximetry probes, and EEG leads to name a few (Figs. 22-3 to 22-8). CPAP devices place vulnerable neonates and children at risk for pressure ulcers. Robertson et al.[39] first reported a rate of 20% for nasal deformities secondary to nCPAP (nasal CPAP) in very low birth weight infants (seven cases). The high risk for ala (lateral surface of external nose that forms outer wall of each nostril), caudal septal, columnar, and nasal bridge pressure ulceration, which can occur secondary to nasal prongs and/or CPAP mask and headgear, is of concern in all NICUs.[40–42] Fischer et al. reported a 42.5% incidence of nasal trauma rate on 989 patients who were treated with nCPAP in NICU with 90% occurring in first 6 days of nCPAP.[43]

Table 22-1 Common Medical Devices That May Cause Pressure Ulcers in Neonates and Children

- Oxygen saturation (O_2) probes
- Orthotics (casts, splints, braces, cervical collars)
- Respiratory devices (endotracheal/oral tracheal tubes, tracheostomy tube/flange, noninvasive ventilation mask, and headgear)
- EEG leads
- Arm boards
- Endotracheal tubes
- Nasal cannula
- Gastrostomy/jejunostomy abdominal tube flanges or bolsters
- Cables, intravenous tubing
- Blood pressure cuffs
- Sequential compression devices (SCD)
- Nasogastric or orogastric tubes

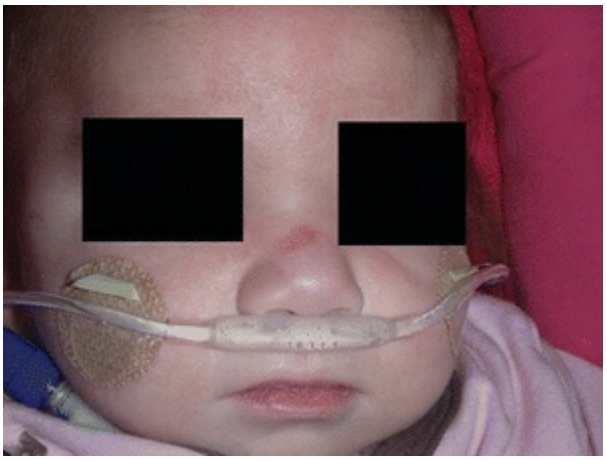

FIGURE 22-3. Stage 1 pressure ulcer on an infant's nose caused by a medical device from BiPAP.

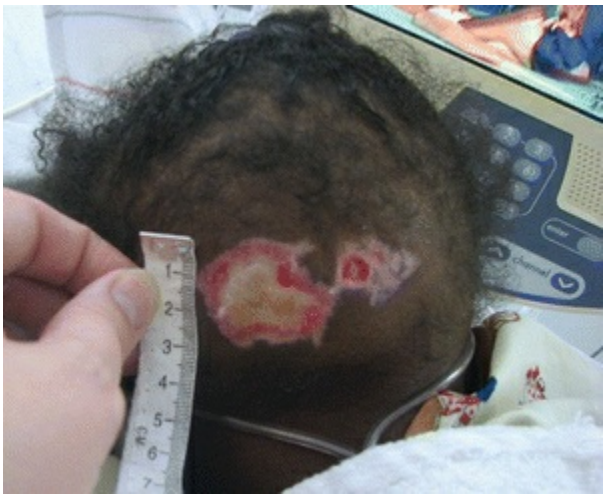

FIGURE 22-4. Unstageable pressure ulcer on an infant's occiput caused by a medical device from oxygen tubing.

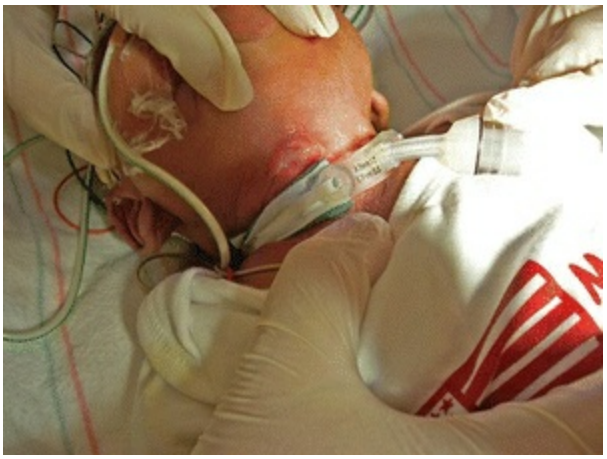

FIGURE 22-5. Unstageable pressure ulcer on an infant's neck caused by a medical device from a trach flange.

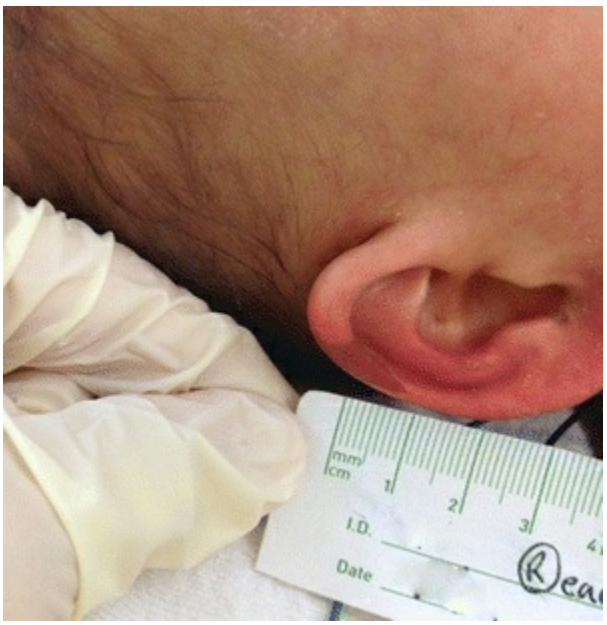

FIGURE 22-6. Suspected deep tissue injury right ear caused by a medical device from oxygen tubing.

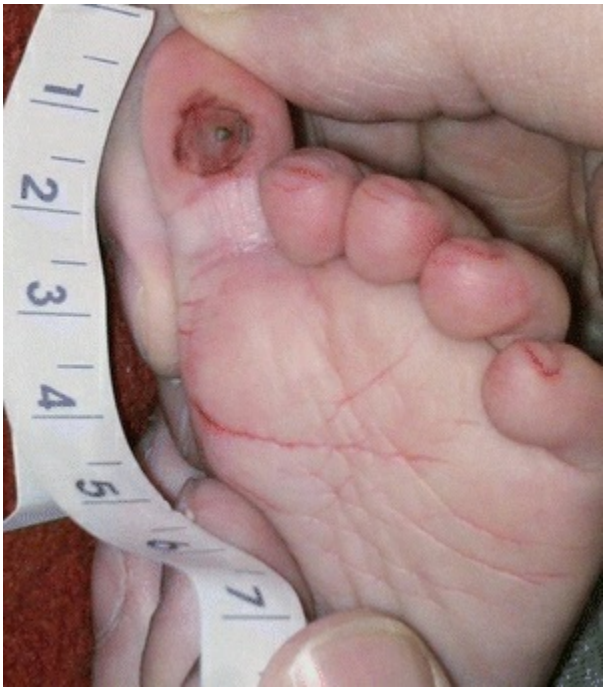

FIGURE 22-7. Suspected deep tissue injury right great toe of an infant caused by a medical device from an oxygen sat probe.

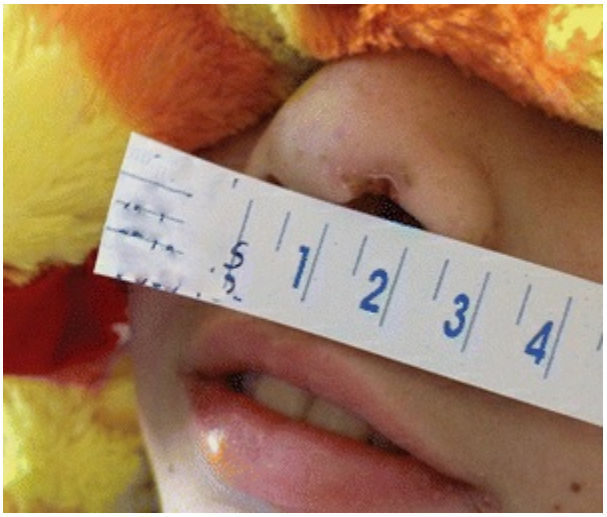

FIGURE 22-8. Unstageable pressure ulcer on a child's left nasal nares caused by a medical device from a nasogastric device.

Preventive measures, such as hourly prong or mask repositioning with skin assessments and the use of protective hydrocolloid or silicone dressings, often prevent the occurrence of these devastating ulcers.[40,42] Murray and colleagues[44] recommend changing oxygen saturation probe locations every 2 to 3 hours, removing orthotics two to three times daily and as needed, and changing dressings under tracheostomy and enteral tube sites

one to two times daily to ensure proper fit and a moisture-free environment, as well as to relieve the pressure. If these actions are medically or clinically contraindicated, this should be documented.

In a case-controlled study of 118 PICU patients by McCord and colleagues,[45] edema, PICU length of stay longer than 96 hours, increased positive end-expiratory pressure (PEEP), weight loss, and not turning the patient or using a specialty bed in turn mode were all identified as risk factors for pressure ulcer development.

Paralysis

Paralysis and insensate skin related to spinal cord injury or myelomeningocele ("spina bifida") have been shown to be risk factors for pressure ulcer development in pediatric patient populations.[46,47] Ekmark[46] reported that shear injuries can occur during wheelchair transfers or transferring out of a car on warm days. Ekmark[46] suggests that new orthotic braces should be checked regularly, particularly 2 and 12 months, and also, whenever children gain or lose weight, wheelchair equipment must be modified. Schottler and colleagues[47] completed a 27-year retrospective chart review of children who had spinal cord injuries at 5 years of age and younger and reported 41% had a history of a pressure ulcer with over 70% located in the foot and ankle. In a retrospective, exploratory study by Samaniego of 69 pediatric outpatients with a primary diagnosis of myelodysplasia, paralysis, insensate areas, high activity, and immobility were all identified as pressure ulcer risk factors.[48]

Risk Assessment Tools

When considering the broad spectrum of skin problems in infants and children, one may regard the phenomenon of pressure ulcers as irrelevant,[49] although over the past decade increasing attention is emerging regarding pressure ulcer risk assessment and prevention in the acutely ill pediatric patient population. To utilize objective criteria to determine patient risk, quantify the severity of risk, and guide clinicians to institute interventions that prevent skin compromise, the use of a pressure ulcer risk assessment scale is indicated. Researchers have emphasized the importance of clinicians recognizing that risk assessment, prevention, and management of immobility-related and medical device–related pressure ulcers are different.[50] It is critically important to differentiate the two phenomena.[44]

There are multiple published neonatal/pediatric pressure ulcer risk assessment scales.[20,51–59] Schumacher and colleagues[60] determined that available tools were too lengthy for clinical use and developed a short trigger tool to identify infants at risk for pressure ulcer development and performed it on 15 NICU patients and determined that although it did not quantify risk, it was an efficient initial screening tool. However, the only pediatric pressure ulcer risk assessment scales for which there are published sensitivity and specificity data are the Braden Q Scale,[51] Neonatal Skin Risk Assessment Scale (NSRAS),[20] and Glamorgan Scale.[57] The three skin assessment tools that have been largely tested and validated are the Braden Q Scale, Glamorgan Q Scale, and the Neonatal Skin Risk Assessment Scale (NSRAS).[61] Both the Braden Q[20,51] and NSRAS were modeled after the adult Braden Scale for predicting pressure ulcer risk.

The NSRAS was created in 1997 for neonates and modeled after the Braden Scale for predicting pressure ulcer risk in adults, although the original Braden Scale has never been psychometrically tested with any age group in the pediatric population.[62] NSRAS measures six subscales pertinent to neonates.[20] The subscales are based on validity testing among a small sample size of 32 NICU patients and demonstrated a sensitivity of 83% and a specificity of 81%.[20] The field of neonatology has changed over the last decade, necessitating a revision of the instrument to reflect evidence-based practice[63]; therefore, recently, the original NSRAS was updated to incorporate the specialized humidification incubator standardly used today to manage insensible water losses of micropreemies with a pilot study to be conducted of the revised tool.

The Braden Q Scale, developed in the early 1990s for predicting pediatric pressure ulcer risk, is a widely used, valid, and reliable pediatric-specific pressure ulcer risk assessment tool that is currently available in nine languages.[58] In developing the Braden Q Scale, Quigley and Curley adapted the six subscale descriptions for the pediatric population and added a tissue perfusion and oxygenation subscale.[36,51] In a multisite prospective study of 322 PICU patients, which excluded children with intracardiac shunting or unrepaired congenital heart disease, the Braden Q Scale was found to be 88% sensitive and 58% specific at a cutoff score of 16.[36] When the Braden Q Scale was first published in 1996, Quigley and Curley used a cutoff of 8 years of age because at the time, the American Heart Association used that cutoff age for pediatric versus adult resuscitation standards. The Braden Q contains all the dimensions of the original Braden Scale and it is

widely used in neonates through adulthood. The Braden Q incorporates the developmental needs of pediatric patients, the prevalence of gastric/transpyloric tube feedings, use of available blood studies, and noninvasive technology. The Braden Q scale[51] has a "tissue perfusion and oxygenation" subscale to quantify risk associated with compromised tissue perfusion. The original study excluded those with complex congenital heart disease so as not to confound the tissue perfusion and oxygenation scale with acutely ill patients who have compromised baseline oxygen saturation levels. Tume et al.[64] conducted a retrospective cohort study on 891 critically ill children to examine how the lowest Braden Q score recorded in first 24 hours of PICU admission related to risk of pressure ulcer development. Tume et al.[64] found that the Braden Q score was found to perform well in children aged 3 weeks to 8 years without congenital heart disease. At a cutoff score of ≤16, it yielded a sensitivity of 100% and specificity of 73.1%.[64] Quigley and Curley have never given permission for the Braden Q Scale to be adapted or modified in any way and discourage use of unvalidated versions such as the Neonatal/Infant Braden Q and Modified Braden Q. Of note, the Braden Q was also never designed to predict medical device–related injuries. The Braden Q, as designed, ***should not be*** used as a comparator in predicting device-related pressure ulcers.[44,50] Quigley, Curley, and Noonan-Caillouette are conducting a multicenter study to re-establish the reliability and validity of the Braden Q for the development of immobility-related pressure ulcers and a new subscale dimension assessing the risk associated with medical devices. The new risk scale is called the Braden Q+D Scale, whereas the letter "D" is for device. Pediatric patients ranging from neonates to 21 years of age, including those born with complex congenital heart disease, are included in the study. They anticipate the study to conclude December 2014 with dissemination of study findings in 2015.

The Glamorgan Scale was developed by identifying risk factors through literature reviews and using the opinions of clinical experts and is based on factors thought to result in the development of pressure ulcers in pediatric patients. Scoring is in two categories mobility and equipment with a weighted risk value score of 15 if there is any equipment pressing on skin. Any patient who scores a 15 or higher is determined to be at "high risk." When used in the assessment of pressure ulcer risk in 336 patients (1 day to 18 years of age), the Glamorgan Scale was found to be 98.4% sensitive and 67.4% specific at a cutoff score of 15.[56,57,65] The higher the Glamorgan total score, the greater the pressure ulcer risk: ≥10 = at risk, ≥15 = high

risk, and ≥20 = very high risk.[56,57,65] In an observational validation study in a pediatric cardiac unit by Kottner and colleagues,[66] they found the Glamorgan scale was unable to make clear distinctions in a low-risk setting; therefore, it is unlikely the tools in this setting provide additional information for clinical decision making.

A further challenge for clinicians is capturing the pressure ulcer risk to patients during procedures such as the operating room, radiology suite, or cardiac catheterization lab. It is imperative for nurses, surgeons, and anesthesiology colleagues to collaborate and assess risk for pressure ulcer development, specific to the positioning needs related to surgical procedures as well. Positioning will vary based upon specific surgical procedures, such as prone position for posterior spinal fusion, and require skin assessment on the frontal surface, which was gravity dependent during surgery. The incidence and prevalence of pressure ulcers for inpatients have been reported, but the effect of surgical procedures on pressure ulcer prevalence and development during admission has not been recognized. Current pressure ulcer risk tools do not address the patient who comes in for surgery, is admitted to the hospital after surgery, and then develops a pressure ulcer.[36,59] Galvin and Curley conducted a comprehensive review of pressure-related skin injuries reported in 24 operative suites and then did a literature review to identify risk assessment scales and intraoperative pressure ulcer prevention strategies for pediatric patients. Consistent with the Braden Q Scale, Galvin and Curley used Braden and Bergstrom's conceptual framework on the etiology of pressure ulcers to organize a pressure ulcer prevention bundle and developed the Braden Q + P Pressure Ulcer Risk Assessment Tool with a yes/no scoring system for each subscale.[59]

Practice Point

Use a pressure ulcer risk assessment scale designed for pediatric patients.

Pressure Redistribution Products

Historically, infants and children were placed on support surfaces (mattresses and overlays) originally designed for adults, the clinical

efficacy of many products with patients of lower weights and smaller stature is unclear. Also, variability of number of layers of linen and continence pads placed by caregivers under patients poses an additional challenge. Many support surface manufactures will disclose upper body weight capacity but not minimal weight requirements. Milbrath and colleagues[67] reported that comparative data demonstrating product effectiveness with persons weighing less than 70 pounds, pressure distribution qualities of construction materials, support for positioning, impact on bed motility, and floatation under load at pressure points were missing. They further reported that patients and their families value comfort and quiet operation and expressed dissatisfaction with powered specialty mattresses due to the operating cyclic on-and-off noise.[67] Premature neonates do not have the muscle tone or strength that full-term infants have and, when placed on their backs, may lie in an extended position causing atypical neuromotor development. To promote flexion patterns of movement, caregivers need to routinely reposition patients using external support aides such as gel or fluidized positioners that facilitate the curled, flexed intrauterine position. Integral to pediatric education is the recognition of each child's uniqueness, the developmental characteristics of each age group, and the psychological and psychosocial factors they face.[26,36]

Today, clinicians have a wide variety of Isolette, crib, incubator, pediatric foam, and foam-/gel-topped mattresses available to choose from as compared to a decade ago. Because children are not "small adults," they should not be placed on adult support surfaces but instead on pressure redistribution products specifically designed for their specialized loading needs. Low–air loss beds designed for adults do not have the numerical option to accommodate for the height and weight of an infant or small child.[14] Children and infants often sink in and between cushions.[14] When adult specialty beds are placed in the turn mode, the occiput of small children pivots on the same pressure point, potentially increasing shear and friction and not redistributing pressure.[45] If a low–air loss bed or alternating overlay is clinically indicated, only those that are age appropriate, clinically efficacious, and safe should be used in accordance with manufacturer's recommendations.

A pilot study of 54 children at Texas Children's Hospital by McLane et al. found the highest interface readings were on the occiput from infancy to age 6 and on the occiput, coccyx, and heels from ages 6 to 18.[68] For children younger than age 2, use of the Delta foam overlay (Span America,

Greenville, SC) resulted in the lowest interface occiput pressures.[68] For children over age 2, use of the Delta foam overlay with a Gel-E Donut head support and positioning aid (Philips Healthcare, Andover, MA) significantly lowered occipital pressure and provided generalized pressure redistribution.[68] Another nonpowered fluidized positioner (Z-Flo, Sundance, White Plains, NY) is designed to function like a fluid but not flow in response to gravity and contour to body surface and redistribute pressure and is widely used in NICUs and in a variety of pediatric environments. More recently, a complete mattress replacement system designed for the pediatric population (Pediatric Pulse, Sizewise) is available for patients weighing between 13 and 55 pounds. It offers a three-zoned support surface and has vented air cells that provide the option of either static low air loss or pulsating low–air loss therapy. A product that simulates the effects of a body floating in a fluid and reduces vertical shear is the Dolphin System (Joerns, Arlington, TX), which applies Fluid Immersion Simulation technology originally developed to transport US Navy dolphins over long distances in a dry environment. Early clinical use was in the perioperative environment as a surgical overlay system for the operating room table, the Dolphin Pad to prevent pressure ulcers from lengthy surgical procedures. Other product lines are now available to replace a standard hospital surface, Dolphin Bed for use with a patient with a minimal weight of 21 pounds that is also available in bariatric sizes, as well as in stretcher and wheelchair pads.

Practice Point

Clinicians must also read all manufacturer disclosures to ensure that product materials, particularly foam cellular pads and mattresses, meet their state- or city-approved flame-retardant fire regulation compliance standards.

Medical Devices

Considering that pressure ulcers occur under a plethora of medical devices, it is imperative that frequent, comprehensive skin inspection occur under all removable devices unless medically contraindicated. To complicate the situation further, materials that might be used to secure medical devices

(e.g., tape, straps, dressings) may prevent assessment of adjacent skin and increase the risk for pressure ulcer development.[44] It is particularly important to assess skin under devices when a patient is developmentally unable to communicate discomfort and has neurosensory impairment such as those with chemical paralysis or spinal cord injury, as well as those with fluid resuscitation that results in localized or systemic edema, which may affect proper fit of medical device. Given that upwards of 50% of pressure ulcers are related to pressure from equipment and devices,[26] it's important to perform frequent skin assessments, rotate blood pressure cuffs and transcutaneous oxygen tension ($tcPO_2$) probes, and provide sufficient padding under tracheostomy plates, nasal prongs, CPAP and BiPAP masks, arm boards, orthotics/casts, and traction boots. All clinicians are responsible for monitoring skin and mucous membranes for alterations in skin integrity. Providers of care including nurses, clinical assistants, physicians, and therapists play an essential role in assessing, preventing, and managing pressure ulcers.[69,70] Collaboration between nursing and respiratory therapy (RT) to move endotracheal tubes and noninvasive ventilation (NIV) bilevel or continuous positive airway pressure (BPAP/CPAP) devices to assess for pressure ulcers of the nose, lips, cheeks, or mucosal injuries of the tongue and buccal mucosa would help prevent or identify concerns early.[44] Manufacturers of NIV equipment today offer variety of masks, headgear, pillows, and chin straps giving clinicians alternative interfaces to avoid skin breakdown in infants and children. Implementing prevention strategies to ensure proper fitting of orthotics, transabdominal feeding tube flanges, wheelchairs, and wheelchair cushions as children grow must be integrated into all clinician practices. Applying interface layers such as foam and gel dressings/products between devices and skin to protect the skin and redistribute pressure should routinely be incorporated into bedside practice.

Beds and cribs should be inspected to ensure that no tubing, cables, leads, hard toys, or syringe caps are inadvertently left under the patient's skin.[26] Tapes securing nasogastric and orogastric tubes, head dressings, and hats should be gently removed and the underlying skin assessed for pressure injuries. Routinely scheduled removal of EEG monitoring pads to assess skin on scalp and forehead should be coordinated with EEG technologist and bedside nursing.

Practice Point

Best Practices for Prevention of Medical Device–Related Pressure Ulcers in Pediatric Populations Poster is available for free download at http://www.npuap.org/wp-content/uploads/2013/04/BestPractices-Pediattric1.pdf.

Practice Point

Because almost half of pressure ulcers in children occur from tubes and devices, routinely assessing the skin beneath them to identify skin compromise and prevent breakdown is critical.

Nutritional Considerations

Nutrition and hydration are a concern to pediatric nurses who must balance their patient's medical condition nutrient needs with the ongoing dietary demands of growth. Potential alterations in nutrition related to metabolic deficiencies, congenital anomalies, swallowing disorders, inadequate intake, and/or increased metabolic need, as well as the additional challenges associated with toddler and school-age food preferences, are routinely encountered in pediatric care environments. The NPUAP has published a White Paper on the role of nutrition in pressure ulcer prevention and treatment although it only addresses adult patient populations. The systemic and immunological ramifications of malnutrition on compromised acute care patients further limit their tissue tolerance to pressure, friction, and shear, especially as third spacing occurs. Nutritional factors such as impaired intake, low birth weight, low body weight or unintentional weight loss, and dehydration may contribute to development of pressure ulcers. It's essential that nutritional screening occurs during the admission assessment and that the protein, caloric, and hydration needs of neonates and children be addressed as part of a comprehensive pressure ulcer prevention and treatment plan. Care providers should attempt to avoid confrontations around food choices to increase cooperation with selections. Offering support for breastfeeding for those expressing an interest in breastfeeding is also paramount. Initiating the use of a breast pump as soon as it is

determined that an infant cannot breast-feed or is made NPO should be standard of care. Furthermore, respect and awareness of vegetarian, gluten-free, peanut-free, lactose-free, and religious diets must be incorporated into dietary choices offered to those who are able to tolerate oral intake in order to maximize nutritional intake.

Topical Management

When selecting a topical agent for use in the neonatal and pediatric populations, it is critical to consider the patient's age and degree of integumentary maturity, skin condition, product adherence, potential for skin sensitization, impact of product absorption, and need for avoidance of products containing dyes, fragrances, and preservatives.[28–30,71] Knowledge of product safety and manufacturer's recommendations in the neonatal and pediatric populations is essential.[28–30]

Normal saline and sterile water are commonly used for wound cleaning in neonatal and pediatric populations. Amorphous and sheet, preservative-free hydrogels and plain and silver hydrofibers are utilized for pressure ulcer treatment, while thin hydrocolloids, foams, thin films, and silicone-based dressings may be used in both prevention and treatment. If the wound bed has devitalized tissue or necrotic eschar and wound closure is the goal of treatment, then an appropriate method of debridement (surgical or autolytic) should be performed. Although anecdotal case reports exist of topical enzymes having been used in the neonatal and pediatric populations, manufacturer recommendations for topical enzymes are only for those over age 18; safety and effectiveness in pediatric patients have not been established.[30]

Practice Point

Before using a topical agent, check with the manufacturer to see if it's safe to use on the skin or wound of a neonate or pediatric patient.

In the treatment of extensive, full-thickness pressure ulcers, negative pressure wound therapy may be utilized to achieve wound closure or as a bridge to surgical closure.[72–74] Stoffan and colleagues[72] reported on 18

infants who had abdominal wounds treated with negative pressure wound therapy (NPWT) and a median duration of treatment of 34.0 days and concluded that NPWT is an important therapeutic tool for the management of abdominal wounds in infants. In the presence of osteomyelitis, the need for appropriate systemic antibiotics must be addressed.

Pressure Ulcer Prevention Bundles

A care bundle incorporates those best practices that incorporate the critical components that if done in combination are likely to lead to better outcomes. The concept of bundling care practices is credited to the Institute for Healthcare Improvement. Defined as a structured way of improving processes of care and patient outcomes, a bundle typically includes three to five evidence-based practices that "when performed collectively and reliably, have been proven to improve patient outcomes."[75] Components of a pressure ulcer prevention program often include a comprehensive head-to-toe skin assessment upon admission and then daily with a focus on bony prominences and skin under medical devices; use of a validated pressure ulcer risk assessment scale and then care planning and protocol implementation to address any areas of risk noted; and an audit tool done daily, weekly, or monthly to check compliance on elements of care. Auditing compliance with a prevention bundle can be done by combination of any of the following to monitor evidence of implementation: paper or electronic medical record documentation, visual observation, and/or skin inspection.

Elements of a pressure ulcer prevention bundle may include any or all of the following components:

- Skin assessment (minimally done once daily or with change in medical condition)
- Repositioning (per facility protocol)
- HOB elevated ≤30 degrees if medically not contraindicated
- Keep heels elevated off bed surface
- Appropriate bed surface/use of positioning aides
- Moisture and incontinence management
- Medical device removal and skin assessment (per facility protocol)

Patient and Family Education

To enhance quality of care, education regarding pressure ulcer risk factors, and prevention and management interventions, is essential for all healthcare

providers, as well as children and their families particularly those at high risk. Empowering families with knowledge will instill confidence and likely decrease risk of pressure ulcer occurrence not only during their hospitalization but at home once they are discharged. Teaching should review the following: avoid dragging or sliding child in and out of the chair or bed; avoid lying in same position and to reposition at least once an hour when sitting and every 2 hours when lying in bed; do not massage any skin that is red, warm or tender; keep skin clean and dry; and apply barrier ointments to protect skin from urine and stool. Parents should be taught to notify their child's nurse if they notice any reddened areas on the skin. When children have darker skin tones, teaching should reinforce to look for any areas of skin discoloration that may have purplish hue or feel warm to touch as compared to surrounding skin, particularly over bony prominences and under medical equipment. At the time of discharge, parents of high-risk infants and children should be taught to perform skin assessments during bathing, diaper changes, and application of orthotics or NIV equipment. Toddlers and preschool-age children can be taught how to perform skin checks on their doll or teddy bear and then on themselves.[76] School-age children with upper extremity abilities should be taught "liftoffs" with the use of mirrors to check the buttocks as well as how to ensure that their wheelchair cushion is functioning properly.[76] Written educational materials, alarm clocks or watches as reminders for liftoffs, and rewards for assuming self-care are beneficial.[76] As parents begin to relinquish control, they should maintain a safety net role in their child's care.[76] Education of teenagers is best provided on a one-on-one basis with respect for their privacy. Educational materials that are concise and focused on their tasks are best received. Watches with automatic alarms to serve as a reminder for liftoffs are of benefit during this developmental stage as well.[76] Graphic images of pressure ulcers and discussion of the possible need for hospitalizations and surgery and time away from friends and social events will assist in emphasizing the importance of prevention.[76]

Practice Point

The pressure ulcer prevention teaching plan should be specific to the child's age, developmental level, and individual characteristics.

Summary

Based on available pressure ulcer prevalence and incidence data, neonates and children are at risk for developing pressure ulcers.[28–30,61] Neonates and children face unique problems from the development of pressure ulcers. Pediatric care environments have an added challenge to maintain competence due to the diverse age population cared for from micropreemies through adults. Support surfaces and topical products manufactured for adults may not be suitable for use in neonates and children because their clinical efficacy and safety in these age groups are unknown. Neonates and children require skin care specific to their needs. The development of much needed clinical practice guidelines and educational programs focused on pressure ulcers in neonates and children must address the immature integument of the premature neonate, including the potential for absorptive toxicity and disparities in weight distribution, and the physiological and psychological uniqueness of these populations while acknowledging the basic tenets found in adult models.[28–30] The use of a risk assessment scale to identify patients at risk, in combination with a comprehensive skin assessment and clinical judgment, is key to a pressure ulcer prevention program. Furthermore, recognizing that any medical device increases a patient's risk for device-related pressure ulcers is the first step in prevention.[44]

Show What You Know

1. What percentage of pressure ulcers in children are caused by medical equipment or devices?

A. None
B. 25%
C. 50%
D. 75%

2. Which one of the following is a pressure ulcer risk assessment scale that can be used for a 5-year-old child?

A. Braden Scale
B. Waterlow Scale
C. Norton Scale

D. Braden Q Scale and Glamorgan Scale

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Palliative Wound Care

23

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Objectives

After completing this chapter, you'll be able to:

- define palliative wound care
- discuss palliative wound treatment
- delineate treatment for a fungating wound
- explain treatment for a radiation wound.

Defining Palliative Wound Care

Palliative care is an approach to enhancing quality of life for patients and their families facing a life-threatening condition and its associated problems. It is focused on preventing and minimizing suffering via early identification, assessment, and treatment of pain and suffering (http://www.WHO.int/cancer/palliative.definition/en/).[1] The 2002 National Consensus Project noted that palliative care is an organized and highly structured system that focuses care on promoting the greatest comfort for and dignity of the patient (http://www.nationalconsensusproject.org)[2] and is best delivered by a multidisciplinary team.[3,4] (See Chapter 1, Quality of Life and Ethical Issues.) In response to these national efforts, many hospitals in the United States and throughout the world have palliative care units or teams. However, in spite of these national-level studies and the growth in palliative care programs, public policies that focus on palliation need to be expanded.

The setting of palliative care, with patients who most often have multiple comorbidities and risk factors and who are at the end of life, is a high risk for pressure ulcer development.[4–6] Although the usual goal and

plan of care is directed toward healing a wound, for individuals at the end of life (with a nonhealing wound), palliative wound care may be desired and the most appropriate goal. While often overlooked as the largest organ of the body, the skin can fail along with the other organs.[7] It is illogical to expect the skin to heal concomitant with the failure of other vital organ systems.[7,8] Most individuals, if not all, at the end of their lives are at risk for developing soft tissue ulceration.[4,9–17] Most professionals agree that pressure ulcers occurring at the end of life are often unavoidable, largely attributable to the individual's frail, compromised condition.[4,5,7,9,13,18–20] In fact, the literature is replete with reports that it is likely impossible to eradicate pressure ulcers in end-of-life patients with their many comorbid conditions and risk factors.[4–6,10,13,17,21–27] However, with education and teaching, which should include a thorough question and answer session between the patient and family and the physician and other healthcare professionals, the goal of care should then be established. The decision to move a patient from a curative to a palliative treatment plan requires that the clinician has determined that the wound is ultimately nonhealing (rather than undertreated) and that palliation is consistent with the patient's goals.[4,28,29]

Extent of the Problem

Today, approximately 300 million individuals, or 3% of the world's population, are in need of end-of-life care every year.[30] Estimates are that by the year 2030, 20% of the US population will be age 65 or older[13] and over 157 million Americans will suffer from chronic illnesses.[31,32] Given this demographic shift, a significant increase must be expected in the number of frail, elderly patients for whom cure may not be the goal. Overall, little information is available on wounds at the end of life.[4,17] Currently, however, wounds affect more than one-third of the nearly 1 million hospice patients in the United States, as well as many more patients at the end of life who are not under hospice care. Over 40 types of wounds have been identified in advanced illness.[17]

Relatively few studies on pressure ulcer prevalence and incidence in end-of-life patients appear in the literature, and, of those that do, most report on subjects with a cancer diagnosis. Little evidence on the prevalence and incidence of end-of-life wounds is available.[33,34] Pressure ulcer prevalence and incidence have been reported in eight studies

worldwide, six in the United States, one in Europe, and one in Canada. Pressure ulcer incidence has varied from 8%[3] to 17%[3,11,20] with a more recent incidence in one study at 10.5% (n = 113/1,081).[35] Pressure ulcer prevalence ranged from a low of 9% to a high of 60.6% in a retrospective study,[36] with a mean of 7.91% in several studies.[8,11,14,36–41] The vast majority were stages I and II. In a 2005 home hospice study, Reifsnyder and Hoplamazian[42] found a 15% to 27% prevalence of pressure ulcers in a population with a 72-year mean age in whom the primary diagnosis was cancer. The primary comorbidity was cognitive-related disorders, with dementia being the primary risk factor.

Tippett[8] conducted a cross-sectional study of 383 hospice patients; 35% had skin wounds, of which 50% were pressure ulcers. The same author did a case series analysis of 192 consecutive patients referred for wound consultation. The mean age was 82 years, 67% were female, and the subjects had multiple comorbidities, with dementia being the primary disorder. The researcher found that 40% of the wounds were pressure ulcers, with the primary location being the sacrum, followed by the heel, foot, and leg.[8] In both populations, pressure ulcers were almost exclusively stages III and IV, with concomitant necrosis and gangrene.[8] Tippett[8] concluded that "wounds at the end of life are a problem of tragic proportion for the nearly 1 million hospice patients and millions of other frail, elderly persons living with chronic disease."

The 2014 International Pressure Ulcer Prevention and Treatment Guidelines[43] present evidence-based recommendations related to palliative care individuals. Unfortunately, but understandably so, a paucity of research evidence exists for this population, leaving the vast majority of the recommendations at the "C" level. International guidelines are also available for venous ulcers[44] as well as arterial and diabetic ulcers.[45,46]

Risk Factors for Skin Breakdown and Pressure Ulcer Development

Several risk factors place individuals at the end of life at risk for the development of skin ulcers as well as impaired healing of skin ulcerations. Advancing age is a well-known risk factor. As one ages, skin becomes drier and more fragile and prone to injury; healing is slowed; the protective function of the epidermis is compromised; and the production of collagen

decreases.[10,47–50] The external forces of pressure and shear deform the skin and underlying tissues, and the tolerance of each individual's tissue to this force is variable. Even with appropriate preventive interventions to avoid the formation of PUs, comorbidities can overwhelm the skin and potentially decrease tissue tolerances.[43,51–53] Many of the known PU risk factors remain nonmodifiable by current treatment.[43,51] There are numerous other risk factors that will be discussed separately.

Impaired Mobility and Repositioning

Any individual at the end of life experiences prolonged periods of inactivity or immobility, which contribute to the occurrence of tissue ischemia from prolonged pressure.[24,35,36,43,47,54–59] The risk of tissue breakdown increases when the individual is older, has more comorbid conditions, or has pain that decreases movement. Immobility is the state of being limited in movement or unable to move independently. Immobility is the most significant risk factor for PU development,[60,61] although at present, there are no methods to measure immobility necessitating reliance on clinical characteristics (e.g., ambulation, bed/chair bound, history of CVA).[61] Immobility was identified as the most common risk factor on risk assessment scale for PU development (on 49 of 56 risk scales).[62] There was a 100% concordance for immobility as a significant risk factor by two consensus panels.[43,51] Immobility contributes to vascular congestion and deep edema from fluid shifts;[63] muscle strength is reduced by 1% to 1.5%/day of bed rest;[64,65] 14 days of bed rest will decrease RBC mass altering oxygen transport, leading to dyspnea and activity intolerance.[63]

Particularly vulnerable to pressure from inactivity are the heels, sacrum, and elbows. Suspending the heels over a pillow while supporting the entire length of the leg or using heel protectors is helpful in decreasing heel pressure.[43] A general guideline is to ensure that the individual with impaired mobility is repositioned at periodic intervals, often occurring every 2 to 3 hours but individualized to the individual's preferences.[43,66] The frequency of repositioning is influenced by the support surface the individual is lying on.[43] Repositioning is challenging for a patient who is hemodynamically unstable, has a great deal of pain, is nauseous or vomiting, or is unable to lie on one side or on the back.[20,43] Honor the individual's choices after explaining the rationale for these interventions. (See Chapter

11, Pressure Redistribution: Seating, Positioning, and Support Surfaces.)

Friction and Shear

Along with immobility, friction and shear are risk factors for pressure ulcer development. Shear stress is the "force per unit area exerted parallel to the plane of interest," and shear strain is the "distortion or deformation of tissue as a result of shear stress."[67] Friction is the "resistance to motion in a parallel direction relative to the common boundary of two surfaces."[67] When combined with pressure, these forces damage and distort tissue and can accelerate tissue damage. To protect the buttocks and sacral areas, use a lift sheet or an overhead trapeze.[43,47,68–71] Protect the sacral area or other bony prominences with a transparent film, silicone, foam, or hydrocolloid to minimize friction.[43] While maintaining the head of the bed at the lowest elevation possible (ideally 30 degrees or less) is recommended to minimize friction and shear to the sacrum and buttocks[43,72,73]; this may not be consistent with the patient's condition or goals. Furthermore, even the 30-degree HOB elevation increased interface pressures.[74] Many individuals at the end of life have impaired ventilation and require the head of the bed to be raised greater than 30 degrees, which would be inconsistent with the pressure ulcer guidelines.[43] A pressure-redistributing mattress overlay or a specialty bed may also be helpful.[43,72] (See Chapter 11, Pressure Redistribution: Seating, Positioning, and Support Surfaces.)

Nutrition and Hydration

As the body systems are shutting down at the end of life, food and fluid requirements generally decrease as well. Individuals at the end of life have a diminished hunger and thirst mechanism, leading to dehydration, decreased oral intake, and impaired metabolism.[71] Lessening of oral intake can occur weeks to months before death.[73] Poor hydration impairs skin turgor, leaving tissue vulnerable to new breakdown. A decrease in protein intake leads to protein wasting and malnutrition. Albumin, a component of protein, provides colloid oncotic pressure to hold liquid in the vascular system. When oncotic pressure is decreased, fluid leaves the vascular system and enters interstitial spaces, causing tissue edema, lowered blood pressure, and impaired blood flow. Nutrition and hydration status can be further impaired if a draining wound causes the loss of large amounts of

fluids and proteins in the exudate. At the end of life, the swallow reflex is decreased as well, impairing food and fluid intake and leaving the individual vulnerable to aspiration. Helping the family and loved ones to understand this end-of-life process can relieve their anxiety and stress. (See Chapter 10, Nutrition and Wound Care.)

That protein–calorie malnutrition is associated with pressure ulcer development in individuals at the end of life has been demonstrated in several studies.[3,36,54,55,74–76] In fact, in a large retrospective cohort study of more than 2,400 adult nursing home residents, Horn et al.[54] documented that an unintentional weight loss at any body mass index increased the chance of developing a pressure ulcer by 147%. Cytokines, stress response proteins, are produced following tissue injury and contribute to the occurrence of malaise, anorexia, and consequent malnutrition, muscle wasting, and decreased albumin synthesis.[77] (See Chapter 10, Nutrition and Wound Care.)

It is important to maintain adequate nutrition and hydration that is compatible with the condition and wishes of the individual. Maintaining adequate nutritional support may not be attainable. Liberalize food choices, offer several small meals and/or snacks, and offer supplements.[43]

Moisture

Moisture is another risk factor that can arise from perspiration, wound exudate, urine, and/or feces.[5,8,12,14,78] Moisture increases the tissue's susceptibility to damage. Incontinence is of particular importance with palliative care patients because of the risk for skin injury and breakdown. When incontinence is present, the primary goals are to prevent and manage skin breakdown, enhance comfort, and control odor. Feces are a chemical irritant to the skin, and their removal adds the element of a mechanical irritant, so gentleness is important. Skin protectants/barriers are also helpful.[43]

Palliative Wound Treatment

The goals of palliative care are to prevent wounds if possible, stabilize and aim to close existing wounds, and manage related symptoms to enhance individual comfort, well-being, and quality of life.[4,5] Palliative wound care differs little from those of curative wound care, aside from the goal of healing. Palliative care is the primary focus when it becomes apparent that

the wound has failed to progress or when the patient's clinical condition deteriorates to a point at which aggressive measures are no longer appropriate. This can be particularly true in "nursing home residents with flexion contractures, cognitive impairment, and limited quality of life."[79]

While the exact mechanisms that lead to poor wound healing are not well elucidated, it is reasonable to assume that the cause is an interrelation between local and systemic factors.[80] When it becomes apparent that wound healing would not render the patient's quality of life significantly better, wound stabilization and palliation become the focus of care.[4,5,81] Palliative wound care focuses on controlling pain, choosing appropriate dressings, managing infection, and protecting the periwound area.

Maintenance of Skin Integrity

The goal of palliative care is to optimize quality of life by controlling physical symptoms and monitoring the patient's psychosocial needs. Patients at the end of life are particularly at risk for skin breakdown. They experience a failure of the homeostatic mechanisms, including the skin's ability to counter insults such as pressure, friction, and shear. This includes decreased cutaneous perfusion and local tissue hypoxia, which ultimately reduce oxygen and vital nutrient utilization and removal.[82] The immune system also becomes impaired with skin breakdown. Thus, not all skin breakdown and pressure ulcers are avoidable.[52,82] When breakdown occurs, impaired oxygenation slows healing due to decreased hemoglobin levels, impaired gas exchange, and decreased blood pressure.[83]

It is essential to maintain skin integrity to prevent pressure ulcers. Maintaining healthy skin requires both comprehensive assessment and care planning. Nutrition and hydration play a key role in skin health. Appropriately managing other skin conditions, (e.g., rashes, incontinence-associated dermatitis) is necessary to maintain the skin's integrity and ability to protect underlying tissues. Preventing and treating skin breakdown go a long way toward meeting palliative care goals. Skin breakdown can be prevented and treated through risk appraisal and assessment, meticulous skin care, positioning, reducing friction and shear, using support surfaces, providing nutrition and hydration, and managing moisture. Careful attention to skin care in the end-of-life patient is exceedingly important, as a feeling of cleanliness can enhance an overall sense of comfort and well-being and can address any odor problems that might be present. A low pH skin cleanser is useful along with a moisture barrier to minimize the effects of

excess moisture. Excess moisture can cause skin maceration, which interferes with the ability of the skin to withstand friction, shear, and pressure, thus making it vulnerable to injury.[72] Therefore, gently cleanse the skin, paying attention to the sacrum, elbows, and heels, which are prone to friction and pressure injury.[68,69] Avoid massage over a reddened area as it can further damage tissue with already impaired perfusion.[72] Unless contraindicated, a gentle overall body massage is often appreciated in an individual at the end of life. Emollients and skin protectants are helpful in protecting the skin.[43] Skin emollients applied according to manufacturer's directions are helpful in maintaining adequate skin moisture and preventing dryness.[43] When incontinence is present, it is important to minimize its harmful effects with skin barrier products.

Practice Point

End-of-life wound care focuses on:

- pain management
- odor control
- management of exudates
- bleeding control
- self-image
- dignity
- quality of life

Patient and Wound Assessment

It is essential to perform a complete head-to-toe assessment of the end-of-life patient, including physical and psychosocial health and overall quality of life. A thorough assessment will assist the healthcare professional in realistically evaluating the need for preventive interventions as well as efficacy and cost-effectiveness of achieving closure, should there be tissue breakdown. The assessment should establish both the risk for and the presence or absence of skin breakdown, including the presence of a pressure ulcer and the risk for developing additional ulcers. The following areas should be included in the patient assessment:[43]

- comorbid health problems
- medications the patient is taking
- risk factors present in the patient
- nutritional status
- diagnostic test results
- psychosocial implications
- environmental resources
- patient/family goals.

The Pressure Sore Risk Assessment Scale for Palliative Care[68] was developed for use in individuals at or near the end of life. Its seven subscales include sensation, mobility, moisture, activity in bed or chair, nutrition and weight change, skin condition, and friction and shear. Scores can range from 7 to 28, with a score of 12 or lower indicating low risk; 13 to 17, medium risk; 18 to 21, high risk; and 22 or over, very high risk. Risk assessment can be done weekly or with significant changes in condition. (See Box 23-1, *Hunters Hill Marie Curie Centre pressure ulcer risk assessment*.) Risk reassessments are important given the likelihood of the patient's deteriorating condition. As the end of life approaches, assessments may be performed less frequently in order to enhance comfort.[43]

Box 23-1 Hunters Hill Marie Curie Centre Pressure Ulcer Risk Assessment

Patient's Name _____ Patient ID No. _____

SENSORY PERCEPTION Ability to respond meaningfully to discomfort related to pressure	**1 No impairment** Communicates discomfort clearly	**2 Slightly Limited** Responds to verbal commands but cannot always communicate discomfort or has sensory impairment in one or two extremities
MOISTURE Degree of skin exposure to moisture/fecal matter	**1 Rarely Moist** Skin rarely moist	**2 Occasionally moist** Skin occasionally moist, extra linen required once per day approximately
MOBILITY Ability to mobilize when out of bed	**1 Walks frequently** Walks around bed at least once every 2 h and outside room at least twice a day	**2 Walks Occasionally** Walks occasionally during day for short distances but may require assistance
ACTIVITY Ability to change body and limb position when in bed or chair	**1 No limitation** Able to change position frequently and unaided	**2 Slightly Limited** Makes slight but frequent changes in body or extremity position
SKIN CONDITION Observed condition of skin in areas exposed to pressure	**1 Skin condition good** Skin appearance good, no evidence of edema, discoloration, etc.	**2 Fragile skin** Skin thin, fragile, dry, flaky, or edematous (e.g., due to age, steroids, edema, inflammation, or lymphedema)
NUTR/T/ON/WE/GHT Food intake or weight change pattern	**1 Satisfactory** Food intake very good OR no significant weight change in last 6 mo	**2 Marginally adequate** Weight appears normal. Food intake slightly restricted
FRICTION/SHEAR Presence of friction/shear	**1 No apparent friction/shear** Can lift body or limb completely without sliding when moving in bed or chair	**2 Occasional friction/shear** Occasionally slides down bed or chair or drags body or limbs—due to position and poor muscle strength or fatigue

Low: 12 and under; Medium: 13 to 17; High: 18 to 21; Very High: 22 and over

Wounds or ulcers in end-of-life patients are often chronic in nature, as healing is significantly impaired due to physical condition and existing comorbidities. A chronic pressure ulcer has a well-defined border with surrounding nonblanchable erythema. When induration is present, it can

extend outward from the wound edges. Many chronic wounds have rolled-under edges that impede wound healing and closure. Wound edges roll under when the wound bed is dry. In response, the wound attempts to preserve what little moisture is present and epithelialization is slowed, leaving the wound bed unepithelialized. Most wounds have drainage, and chronic wound drainage contains destructive enzymes as well as fibroblasts that are less effective at producing collagen to heal the wound.[84] Assess the wound initially and with each dressing change, but at a minimum weekly unless the individual is actively dying.[43] (See Chapter 5, Acute and Chronic Wound Healing.)

Evidence-Based PracticeWound and Patient Assessment in Palliative Care

1.0. Set treatment goals consistent with the values and goals of the individual, while considering the family input. (Strength of Evidence = C)

1.1. Set a goal to enhance quality of life, even if the pressure ulcer cannot be healed or treatment does not lead to closure/healing. (Strength of Evidence = C)

1.2. Assess the impact of the pressure ulcer on quality of life of the individual and his or her family. (Strength of Evidence = C)

1.3. Assess the individual initially and with any significant change in condition to reevaluate the plan of care. (Strength of Evidence = C)

Brown, G. "Long-term Outcomes of Full-thickness Pressure Ulcers: Healing and Mortality," *Ostomy/Wound Management* 49:42-50, 2003.

Evidence-Based PracticeMaintaining Skin Integrity in Palliative Care Patients

1.0. Ensure that a complete skin assessment is part of the risk assessment screening policy in place in all healthcare settings.

(Strength of Evidence = C)

Brown, G. "Long-term Outcomes of Full-thickness Pressure Ulcers: Healing and Mortality," *Ostomy/Wound Management* 49:42-50, 2003.

Support Surfaces

Support surfaces are "specialized devices for pressure redistribution designed for management of tissue loads, micro-climate, and/or other therapeutic functions (i.e., any mattress, integrated bed system, mattress replacement, overlay, or seat cushion, or seat cushion overlay)."[67] Some support surfaces provide the additional capability of turning or rotation assistance. It is important to match the support surface to the needs of the individual such that the surface redistributes weight over a larger area, thereby minimizing tissue pressures, particularly over bony prominences.[67] These surfaces are helpful for both the bed and the chair. Refer to the NPUAP/EPUAP/PPPIA guideline[43] for more specific recommendations regarding pressure redistribution in palliative care patients.

Practice Point

Individualize the patient's turning and positioning schedule based on his or her pain tolerance and comfort level.

Evidence-Based PracticePressure Redistribution in Palliative Care Patients

1.0. Strive to reposition an individual receiving palliative care at least every 4 hours on a pressure-redistributing mattress such as viscoelastic foam or every 2 hours on a regular mattress. (Strength of Evidence = B)

Brown, G. "Long-term Outcomes of Full-thickness Pressure Ulcers: Healing and Mortality," *Ostomy/Wound Management* 49:42-50, 2003.

Evidence-Based PracticeNutrition and Hydration in Palliative Care Patients

1.0. Allow the individual to ingest fluids and foods of choice. (Strength of Evidence = C)

1.1. Offer several small meals per day. (Strength of Evidence = C)

Brown, G. "Long-term Outcomes of Full-thickness Pressure Ulcers: Healing and Mortality," *Ostomy/Wound Management* 49:42-50, 2003.

Pain Management

Both prevention and treatment of skin breakdown can be uncomfortable for individuals at the end of life. The majority of patients with a pressure ulcer experience moderate to severe pain,[85–88] especially during dressing changes and wound bed treatments, and the pain can be acute or chronic.[88,89] In fact, 21 of 23 patients (91%) in one study reported a pressure ulcer as painful.[90] Likewise, in a systematic review of 15 studies addressing the impact of pressure ulcer pain, the authors concluded that "pain was the most significant consequence of having a pressure ulcer and affected every aspect of patients' lives."[73] In fact, pain from a pressure ulcer can be the most distressing symptom an individual might report.[73] It is important to keep in mind that "pain is anything the patient says it is."[91]

Wound pain can be caused by the tumor pressing on nerves and blood vessels or by exposure of dermis.[92] Pain in wounds often arises from painful procedures, including cleansing and dressing removal, particularly if the dressing is dry and adherent.[93] Given that an individual at or near the end of life has a wound that likely will not heal, the wound and wound pain are chronic in nature. Chronic pain is pain that is persistent and can occur even when the wound is not being manipulated.[94] Concomitant acute pain can occur with dressing changes, treatments, and additional trauma to the area.

One of the most significant goals in palliative care is to alleviate, or at the very least manage, pain. It is therefore essential to perform regular pain

assessments.[29,81] Pain assessment tools reported to be valid and reliable include the visual analog scale for pain (VAS pain); the visual rating scale (VRS); the numeric rating scale; the FACES rating scale (FRS); the FLACC scale (face, legs, activity, cry, consolability); and the McGill pain questionnaire (MPQ). A 2005 study[95] identified the VAS pain, the FRS, and the MPQ as valid and reliable to identify pressure ulcer pain.

A mild to moderate opioid, such as 1 mg morphine or diamorphine mixed with 1 mg of a hydrogel,[96,97] or nonopioid medication can be used to relieve wound pain, as can a topical agent that contains a local anesthetic (e.g., one with lidocaine, such as EMLA cream, Lidoderm, or Regenecare)[97–99] or a foam dressing containing ibuprofen (not available everywhere).[100,101] One hospital solely dedicated to caring for palliative patients has developed a unique mixture of Balmex and lidocaine 2.75%, which may be applied topically to painful and odorous wounds. They have reported success with this strategy.[102] Wound treatment pain can also be minimized by using minimal mechanical force for cleansing (4 to 15 psi irrigation force); using warmed products, such as normal saline or gauze pads[103]; and avoiding antiseptic and cytotoxic agents.[43,72] Nonpharmacological strategies such as music, relaxation, position changes, meditation, guided imagery, healing touch, distraction, etc., are also available.

Practice Point

Premedicating the patient with pain medication 30 to 60 minutes before changing the dressing is an important part of palliative wound care management.

Wound Dressings

When possible, select a dressing that can remain in place for several days; however, this isn't always possible when a large amount of exudate is present. A dressing that protects periwound skin is also desirable, as is one that protects the wound from moisture and feces. As a rule, nonadherent, foam, polymeric membrane, and/or silicone dressings are best. Maintain a moist wound care environment to prevent exposure of delicate nerve

endings,[104] as dry, desiccated wound beds and dressings are nearly always painful.[105] (See Wound Treatment Options, Chapter 9.)

Evidence-Based PracticeTreating Open Pressure Ulcers in Palliative Care Patients

1.0. Diamorphine HDG is an effective treatment for open pressure ulcers [wounds] in the palliative care setting. (Strength of Evidence = B)

Brown, G. "Long-term Outcomes of Full-thickness Pressure Ulcers: Healing and Mortality," *Ostomy/Wound Management* 49:42-50, 2003.

Wound Infection, Odor, and Exudate

Tissue deprived of oxygen and nutrients becomes devitalized and nonviable,[106] and bacteria thrive on this moist, devitalized tissue.[107] As the bacteria colonize, necrotic material appears in the wound and creates an odor, which varies depending on the bacteria present. Nonviable tissue eventually serves as a culture medium to support bacterial growth and inhibit leukocyte phagocytosis of bacteria.[106] Odor is generally associated with anaerobic and some gram-negative (e.g., *Pseudomonas*) microorganisms.[83,108] Anaerobic bacteria are usually present in necrotic material, thrive in the absence of oxygen, and can become buried deeper within the wound. Anaerobic bacteria[105] also have a stronger, more offensive odor that can be particularly distressing to the patient. The wound appearance becomes black and leathery with exposure to air or yellow-gray when exposed to moisture, which occurs over varying lengths of time depending on the underlying disorder.[105]

It is the odor, drainage, and pain arising from the infection that are often the most distressing to the individual and therefore should be treated. Wound odor can be embarrassing to the individual and lead to isolation, depression, and poor quality of life.[2,22,108] Treatment is aimed at removing the cause of the odor and the odor itself. Because saturated dressings can hold odor, more frequent changing may help control the odor as well as the pain from the weight of the dressing. A secondary dressing may help.

Frequent irrigation also helps remove exudate and odor. Nonviable tissue can be debrided, and autolytic debridement is often the least painful for the individual. Sharp debridement is more or less a last resort, and caution must be exercised to prevent excessive bleeding and/or pain.[108,109] Topical metronidazole (gel, cream, crushed tablets, oral) has been shown to be effective for anaerobic bacteria as has silver sulfadiazine.[4,5,43,110–118] Activated charcoal dressings have also been shown to be helpful.[80,84,109,114,118–122]

Nonsurgical (autolytic or enzymatic) debridement is recommended due to the tendency for bleeding and "seeding" of malignant cells in fungating and radiation wounds.[105] Topical metronidazole has also been used successfully to control odor.[2,78] Activated charcoal dressings are effective in controlling odor quickly,[22,109,111,115,120,123,124] as are occlusive dressings and frequent dressing changes.[22] Cadexomer iodine is an effective antiseptic,[116] as is povidone–iodine.[117] Silver dressings are effective for treating infections and thus in controlling odor. Dakin's solution (0.25% sodium hypochlorite) is another effective odor controller; it is saturated into gauze and placed in the wound for a limited time and may cause some discomfort.[117] Placement of larvae is another effective method of eliminating infection and controlling odor from wounds with extensive necrotic tissue.[118] Room deodorizers are also helpful. Sugar paste and honey are once again being used for their antibacterial and debriding properties.[121,122] The high sugar content produces a hyperosmotic wound environment to inhibit bacterial growth and assist in debridement.[9,121,122]

Periwound skin protection is crucial because exudate, which is liquid and sometimes caustic, can exacerbate skin damage[123] by causing maceration, breakdown, and itching.[43] Dressings that appropriately control exudate without unnecessarily increasing wetness or dryness are recommended, such as an alginate, Hydrofiber, foam, or a nonadherent dressing (silicone, polymeric foam, foam), often with a secondary absorbent pad.[105,124,125] Be sure to change dressings when they become saturated, as heavy or overly saturated dressings can cause wound bed pain and irritate periwound skin. Alternatively, if exudate is minimal, a low-absorbency dressing, such as a hydrocolloid or semipermeable film, is recommended.[126] A barrier film around the periwound area is helpful in controlling damage from moisture. See Chapter 9, Wound Treatment Options, for further information on dressings.

Evidence-Based PracticeWound Dressing and Odor Control Options for Palliative Care Patients

1.0. Manage the pressure ulcer [wound] and periwound area on a regular basis as consistent with the individual's wishes. (Strength of Evidence = C)

1.1. Use antimicrobial agents as appropriate to control known infection and suspected critical colonization.[2,102,108,112,127] (Strength of Evidence = C)

1.2. Consider the use of properly diluted antiseptic solutions for limited periods of time to control odor. (Strength of Evidence = C)

1.3. Consider the use of topical metronidazole to effectively control pressure ulcer [wound] odor associated with anaerobic bacteria and protozoal infections.[2,102,108,128–131] (Strength of Evidence = C)

1.4. Consider the use of dressings impregnated with antimicrobial agents (e.g., silver, cadexomer iodine, medical grade honey) to help control bacterial burden and odor.[122] (Strength of Evidence = C)

1.5. Consider the use of charcoal or activated charcoal dressings to help control odor.[22,108,132,133] (Strength of Evidence = C)

1.6. Consider the use of external odor absorbers for the room (e.g., activated charcoal, kitty litter, vinegar, vanilla, coffee beans, burning candle, potpourri).[2,128,132] (Strength of Evidence = C)

1.7. Cleanse the wound with each dressing change using potable water (i.e., water suitable for drinking),[134] normal saline, or a noncytotoxic cleanser to minimize trauma to the wound and help control the odor.[72,135] (Strength of Evidence = C)

1.8. Debride the ulcer [wound] of devitalized tissue to control

infection and odor.[43,72] (Strength of Evidence = C)

1.9. Avoid sharp debridement with fragile tissue that bleeds easily.[72,136–138] (Strength of Evidence = C)

Brown, G. "Long-term Outcomes of Full-thickness Pressure Ulcers: Healing and Mortality," *Ostomy/Wound Management* 49:42-50, 2003.

Practice Point

Odor control is vital for enhancing quality of life for wound patients receiving palliative care.

The inflammatory fluid that seeps from the extracellular spaces is what is known as exudate. All bacteria produce exudate, the color and odor of which vary according to the causative organism.[105] For example, green exudate generally indicates gram-negative, aerobic bacteria, which respond well to silver found in many dressings now on the market.[105] The more persistent the inflammation or infection is, the more exudate that will be produced. Exudate frequently contains proteins; when combined with inadequate oral intake of protein commonly seen in the end-of-life individual, the degree of hypoproteinemia can increase. Managing exudates can be a major challenge.[127]

Fungating Wounds

Fungating wounds occur when the skin and its supporting blood and lymph vessels are infiltrated by a local tumor or by metastatic spread from a primary tumor, resulting in oxygen starvation to the tissue and eventual necrosis.[12,120,136] It's reported that approximately 5% to 19% of patients with metastatic cancer will develop a fungating wound.[11,137] The incidence in elderly individuals over age 70 is higher.[125,134] Although these wounds often develop during the last months of life, they can be present for years.[22] The most common site for development of a fungating wound is the breast, but they can also be found on the head and neck as well as in an area of melanoma. The anatomical location and the delicacy of the surrounding

tissue make it challenging to address these wounds.

The term fungating refers to a malignant process of both ulcerating and proliferative growth through direct invasion.[139–142] An ulcerating wound will produce a craterlike wound, whereas a lesion with a predominantly proliferative growth pattern often develops into a nodular "fungus"- or "cauliflower"- appearing lesion.[137,139] Mixed-appearing lesions can also develop.[137,139] Skin tumors tend to become ulcerated because the skin is a bacterially contaminated surface.[140] Common symptoms of fungating wounds include exudate, pain, odor, pruritus, and bleeding as well as psychosocial issues.[120,136]

Practice Point

Families, support persons, and caregivers may need emotional support when viewing patients with fungating wounds.

Fungating wounds rarely heal;[139] thus, management is centered on symptom control, promotion of comfort, and maintenance or improvement of quality of life.[137,141] Assessment and management by healthcare providers, especially the nurse and physician, are most challenging.[137] Therefore, excellent interdisciplinary care and ongoing patient–caregiver communication are essential.

Care of Fungating Wounds

Woo and Sibbald[80] proposed an acronym for treatment of fungating wounds. HOPES provides a systematized approach and includes **H**emorrhage, **O**dor, Pain, **E**xudate, and **S**uperficial infection.[80] Hemorrhage due to erosion of blood vessels is the most common emergency seen in fungating wounds and can also be related to the decreased platelet function within the tumor.[142] Blood vessels can become eroded from the tumor cells themselves or secondary to necrosis or sloughing of tissues after radiotherapy.[143] Nonsurgical (autolytic or enzymatic) debridement is recommended due to the tendency for bleeding and "seeding" of malignant cells.[105,124] Fungating lesions are friable and predisposed to bleeding. To minimize

bleeding, use a nonadherent or soft silicone dressing, maintain a moist wound bed, and clean by gentle irrigation rather than swabbing.[140] Dry dressings can cause bleeding when they adhere to the wound bed and should be avoided.[105] Alginate dressings have a high seaweed content and exchange sodium ions for calcium ions in the wound bed, thus encouraging the clotting cascade. Alginates must be used with caution in fragile tumors, however, because these dressings can also cause bleeding.[96] Other natural hemostats are collagen and oxidized cellulose; silver nitrate is a sclerosing agent; epinephrine is a vasoconstrictor; and sucralfate is an astringent.[105,120,134] Hemostatic surgical sponges can also be used and left in place for a time.[22] Alginates, foam, silicone, and polymeric foam dressings are helpful for odor.[144,145]

Radiation Wounds

Radiation therapy targets a high-energy x-ray beam to an area of treatment. The target area is usually a tumor, the area surrounding a tumor, or an area where a tumor has been surgically removed. While each treatment is designed to target tissue at a particular depth, the tissues overlying the site can be affected as well.[146,147]

Radiation-related skin changes or ulcerations can occur in soft tissues during the course of therapy, immediately after therapy, or a long time following therapy.[146,147] Skin problems can also be noted in individuals who underwent treatments years ago before technological improvements in radiation machines were implemented. The skin reactions seen are generally specific to the area that was irradiated, and the inflammation can occur almost immediately.[147,148] Acute erythematous wounds result from the dilated blood vessels in the irradiated area. The ulceration may be large and may present initially as a draining sinus.[149]

The more common skin reactions associated with radiation therapy include flaking or peeling, redness, changes in pigmentation, loss of hair, decreased or absent perspiration, superficial blood vessel changes, edema, ulceration, and scarring.[140,147] (See Box 23-2, *Common radiation wound skin reactions*.)

Box 23-2 Common Radiation Wound Skin

Reactions

- Flaking or peeling (dry desquamation)
- Erythema
- Alteration in pigmentation
- Hair loss
- Loss of perspiration or sebaceous excretion
- Changes in superficial blood vessels
- Edema
- Ulceration (moist desquamation)
- Scarring

From Smith, S. Skin Care Following Radiation Therapy: The Clinician's Notebook. *Carrington Laboratories, Inc. Newsletter*; 1(3):1-3. Available at: http://www.woundcare.org/newsvol2n2./ar3.htm. Accessed June 4, 2014.

Changes at the cellular level can be reflected by poor healing at the site. Healing is impeded related to atrophy of the epidermis and epidermal accessory structures, microvascular occlusions, exuberant connective tissue, decreased fibroblast reproduction, and significant amounts of cellular damage.[140,149]

Most radiation-related lesions are superficial. In 1994, the Oncology Nursing Society created a classification system for radiation ulcers. This system was refined in 2002. The five-level classification system ranges from "0" or no skin problem within a radiation field to "4" or skin necrosis or ulceration of full-thickness dermis[150] (Table 23-1).

Table 23-1 Oncology Nursing Society Classification for Skin Reactions

0	None
1	Faint erythema or dry desquamation
2	Moderate to brisk erythema or patchy moist desquamation, mostly confined to skin folds and creases; or moderate edema
3	Confluent moist desquamation ≥1.5 cm in diameter and not confined to skin folds; pitting edema
4	Skin necrosis or ulceration of full-thickness dermis; may include bleeding not induced by minor trauma or abrasion

Adapted from Oncology Nursing Society. *Radiation Therapy Patient Care Record: A Tool for Documenting Nursing Care*. Pittsburgh, PA: Author, 2002, with permission of the publisher.

Treatment of a radiation-induced skin lesion is essentially like treatment for other types of wounds. Any tissue within a radiation field must be considered at high risk for potential breakdown and should be kept clean, appropriately moistened, and protected from potential injury. Skin can also be protected by avoiding restrictive clothing, adhesives, harsh chemicals, heat or sunlight, and trauma. Should a minor skin reaction such as erythema or dry desquamation occur, the same guidelines apply, along with use of a topical hydrogel or a steroid cream.[140] Moist desquamation is also treated in the same manner as described above, with the addition of a nonadherent or foam dressing to manage the wound environment. It's important to cover the wound to prevent evaporation of fluid, control pain, and reduce risk of infection.[147,148]

Severe ulceration or necrosis needs to be treated as an open wound, using moist wound healing principles.[140,147] However, it's important to first rule out a new malignancy in the area.[148] Skin grafting or growth factor application may be required.[136] As a consequence of the vascular changes and resultant hypoxia, irradiated tissues have a decreased ability to fight infection. Avoiding or controlling infection is important, and antibiotics are best delivered topically.[149] The vascular changes and hypoxia also are responsible for pain being present in these ulcerations.[149] These wounds are typically difficult to manage and slow to heal. In all instances, systemic support is necessary to enhance the patient's healing potential in order to minimize further trauma to the wound site.[147]

Summary

While cure and/or healing of a wound is not always realistic, it's possible to provide compassionate and symptom-relieving treatment for palliative care patients who have wounds. This includes balancing the management of local wound symptoms, such as pain, odor, exudate, and bleeding, while preserving patient dignity and self-esteem and maximizing quality of life. Few randomized clinical trials or other research studies exist in the area of palliative wound care. However, there is a consensus document from the International Palliative Wound Care Initiative that looks at managing these wounds across the life continuum.[151] Continued study is needed to more clearly understand when a palliative care goal is appropriate. A comprehensive palliative wound care program needs to be developed in clinical agencies that work with these patients. The interdisciplinary team would include the physician, nurses, wound care specialists, dietitian, chaplain services, social services, and pain and hospice consultants.[69] Palliative care units are increasing in number, particularly for patients who are chronically, but not terminally, ill.[152] Wounds treated appropriately, even when the goal is not healing, can markedly improve in 50% of the cases, even on a hospice unit.[7,153,154]

PATIENT SCENARIO

Clinical Data

Mr. M is an 86-year-old widower who has end-stage cancer with bone metastasis. Because he is quite coherent, refuses to be hospitalized, and insists on staying in his home, his family is caring for him with the assistance of hospice. While Mr. M is consuming liquids fairly well, his food intake is very minimal; he eats very little, even with encouragement. With his small frame, his current weight is 109 lbs, down from his usual weight of about 165 lbs. He remains continent and uses a bedside commode with assistance. Upon assessment, the hospice nurse identifies a stage III pressure ulcer over the left greater trochanter measuring 2.5 × 2.3 × 0.3 cm, and there is a moderate amount of tan/green exudate with a slightly foul odor emanating from it. He also has a stage IV pressure ulcer measuring 0.25 × 0.25 cm on the left ear.

The family has tried both a sheepskin and an air overlay on his bed, but Mr. M insisted they be removed because he didn't like them. Mr. M informs the hospice nurse that he is "ready to go, and doesn't wish for any fancy or heroic treatments. Just let me be."

Case Discussion

Based on the initial assessment, the hospice nurse identified a number of problems that needed to be addressed. The assessment included ascertaining both Mr. M's goals of care and those of his family. Mr. M expressed his desire to just be kept comfortable. His wish was to remain in his own bed with a "pillow top" mattress rather than in a "hospital" bed, and he was adamant that he did not wish to have any other pressure-redistributing devices on his bed. While a plan of care would include regular repositioning, Mr. M verbalized that "it hurt less to lie on my left side on the ulcer than in any other position." The nurse educated him on the risk for further breakdown of the ulcer and ear as well as other areas of his skin. With encouragement from his family, Mr. M agreed to lie on his back and right side for 30 minutes each and on his left side for 2 hours at a time, on a rotating basis. Bilateral heel protectors were used, and his legs were elevated with pillows placed lengthwise in the bed.

Mr. M's ulcer was cleansed at each dressing change with an antiseptic solution to assist with odor management and covered with a composite dressing. A foam dressing was used on the ear to cushion and protect it. A pie tin with charcoal was placed under his bed to absorb room odor. A skin protectant was used, particularly over bony prominences, to help prevent further breakdown and protect his fragile skin.

Mr. M's dietary likes and dislikes were also assessed. After being educated on the need for protein for strength and energy to get up to the bedside commode, he agreed to consume three high-protein liquid drinks per day, along with popsicles, ice cream, and occasionally oatmeal and a piece of cold meat rolled with a slice of cheese, as tolerated.

After pain assessment as well as 30 minutes prior to dressing changes, Mr. M was medicated using a nonopioid medication. Mr. M was encouraged to request a "time-out" during dressing changes if needed.

While the pressure ulcer over the left greater trochanter did not

heal, it only increased in size to 3.5 × 3.5 × 0.3 cm prior to his death. This outcome was within the goal of the patient and family. The ulcer on the left ear did not heal either, nor did it increase in size. Taking care to position Mr. M's head when he was on his left side proved helpful in preventing deterioration. Mr. M remained coherent up until his death. Two days prior to his death, Mr. M expressed his appreciation for allowing him to do things "his way."

Show What You Know

1. Which of the following defines palliative care?

A. An organized and highly structured system to deliver care focused on promoting the greatest comfort and dignity of the patient

B. Care that affirms life yet strives to deliver highly organized care to an individual who is focused on regaining a former health state

C. A care delivery system focused on wound healing and elimination of symptoms

D. Care that is delivered at home by loved ones without the involvement of healthcare providers

2. Which of the following situations would constitute palliative wound care?

A. Wet-to-dry dressing changes every 4 hours around the clock

B. Calcium alginate dressings used on a necrotic-appearing wound with minimal exudate

C. Silver-impregnated dressings used on a wound with little evidence of inflammation and essentially no evidence of infection

D. A hydrogel dressing placed on a wound every 3 days and as needed

3. Which of the following orders should the clinician question in caring for a fungating wound?

A. Using nonadherent dressings

B. Using cold saline when irrigating the wound

C. Using a mixture of morphine with amorphous hydrogel

D. Using music and other relaxation techniques when providing care

4. A patient with a grade 3 (Oncology Nursing Society classification system) skin reaction would require which the following interventions?

A. None, skin is normal
B. Frequent application of skin moisturizer
C. Use of a protective skin barrier
D. Enzymatic debridement ointment three times per day

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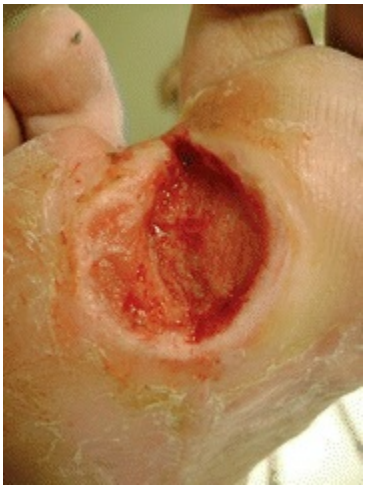
Wound Gallery

24

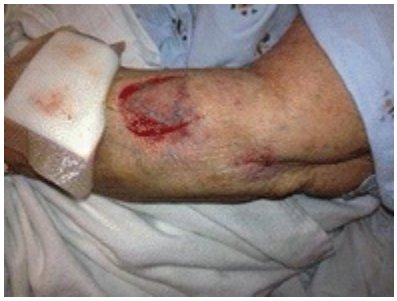
Sharon Baranoski, MSN, RN, CWCN, MAPWCA, APN, FAAN
Elizabeth A. Ayello, PhD, RN, ACNS-BC, CWON, MAPWCA, FAAN

1. What is the most likely diagnosis of this recently debrided ulcer on the plantar surface of the foot?



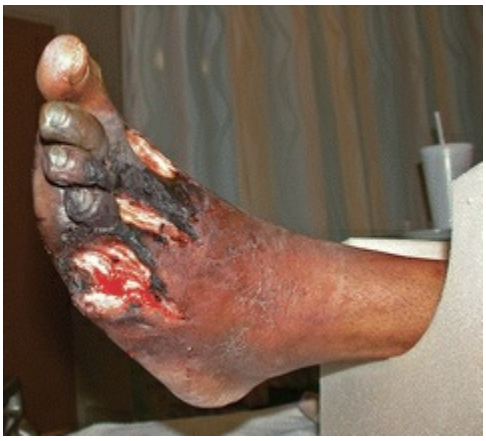
A. Pressure ulcer
B. Arterial ulcer
C. Diabetic ulcer
D. Venous ulcer

2. Using the International Skin Tear Advisory Panel (ISTAP) classification system, what type of skin tear is seen here on the upper extremity?



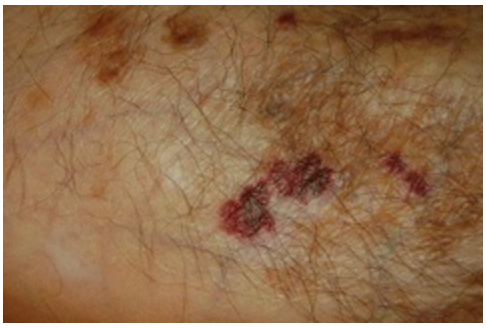
A. Type I
B. Type 2
C. Type 3
D. Type 4

3. Given that this patient has gangrene of his forefoot, what is the most likely etiology?



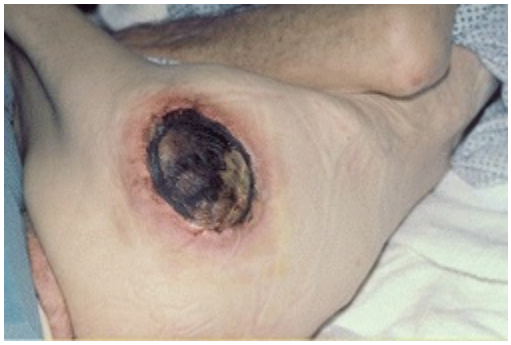
A. Arterial disease
B. Venous disease
C. Necrotizing fasciitis
D. Pyoderma gangrenosum

4. During a skin assessment, you discover the brownish-purplish dry area on the upper extremities; you suspect that this is:



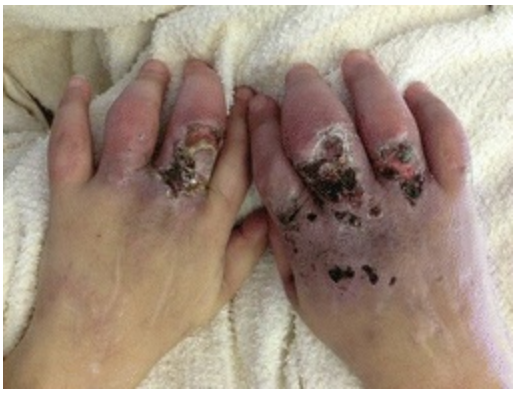
A. melanoma.
B. pyoderma.
C. basal cell carcinoma.
D. senile purpura.

5. Using the National Pressure Ulcer Advisory Panel (NPUAP), this ulcer is classified as:



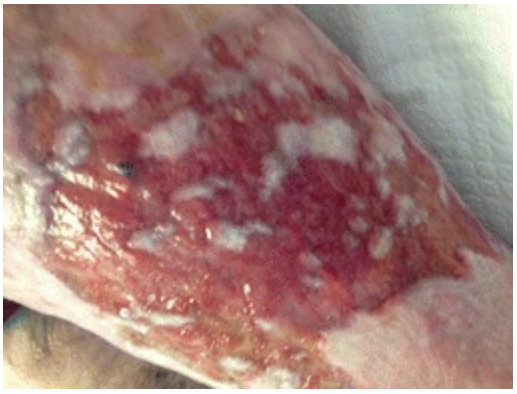
A. stage I.
B. stage III.
C. unstageable.
D. SDTI.

6. What do you think is the etiology of these multiple wounds on the fingers?



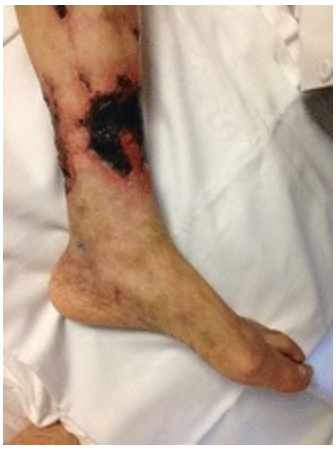
A. Frostbite
B. Eczema
C. Drug injection injury
D. Necrotizing fasciitis

7. What are these white area of tissue called?



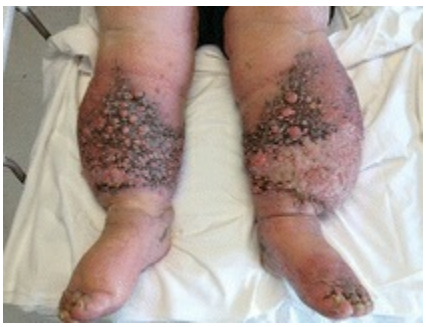
A. Biofilm
B. Granulation island buds
C. Slough
D. Eschar

8. What do you think is the underlying etiology of this wound?



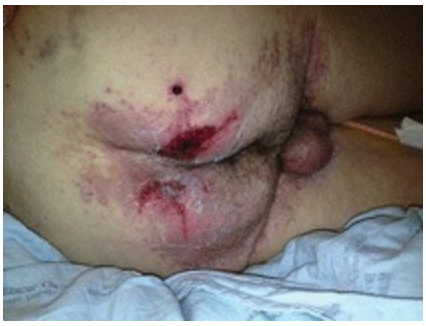
A. Pressure
B. Renal failure
C. Sickle cell disease
D. Ischemia

9. Which of the following best describes this patient's legs?



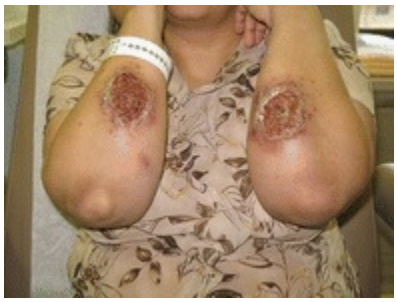
A. Lymphedema with fibrosis
B. Lipedema
C. Melanoma
D. Scleroderma

10. What is the *primary* etiology of this skin injury?



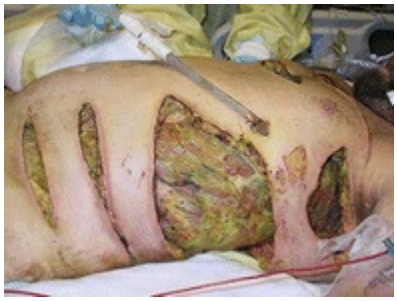
A. Pressure ulcer
B. Skin tear
C. Gluteal cleft injury
D. Moisture-associated skin damage

11. This patient presents with ulcerative lesions and provides a history of working as an itinerant field laborer. What is the most likely diagnosis?



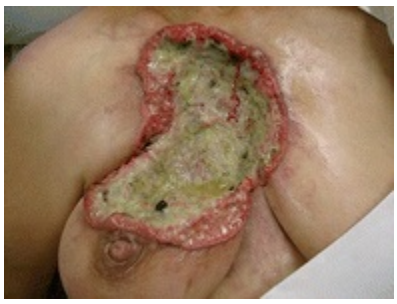
A. Buruli ulcer
B. Blastomycosis
C. Montezuma ulcer
D. Traumatic abrasions

12. Based upon the tissue appearance and clinical findings, the BEST diagnosis is:



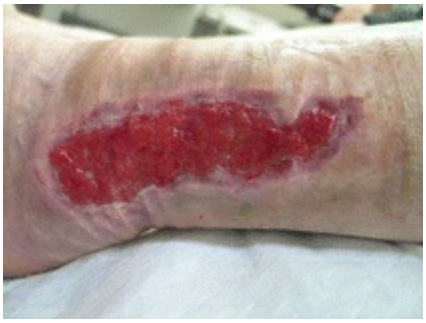
A. gas gangrene.
B. infected postsurgical wound.
C. necrotizing cellulitis.
D. necrotizing fasciitis.

13. Despite the clinical findings that suggest carcinoma, tissue biopsy is indicated to establish a definitive diagnosis. Based on histopathology, which location would be the best choice to perform tissue biopsy?



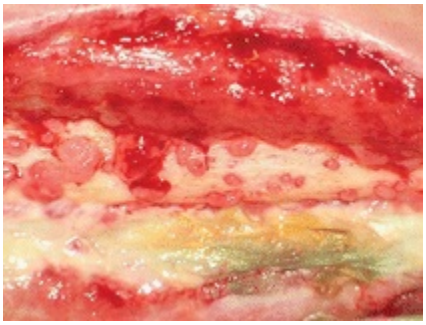
A. Site 1
B. Site 2
C. Site 3
D. Site 4

14. Which of the following is the best description of the translucent tissue indicated by the arrow?



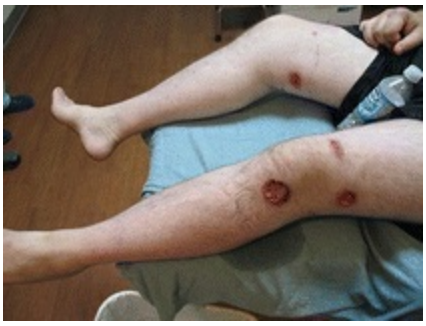
A. Fibrin
B. Granulation
C. Neoepithelium
D. Proliferative

15. Granulation tissue that forms on exposed bone is unusual and suggests:



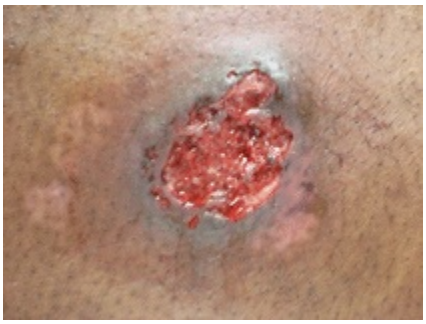
A. intact and viable periosteum.
B. recent revascularization.
C. use of hyperbaric oxygen.
D. application of negative pressure wound therapy.

16. Based on the wound presentation, which diagnosis is most likely correct for this patient?



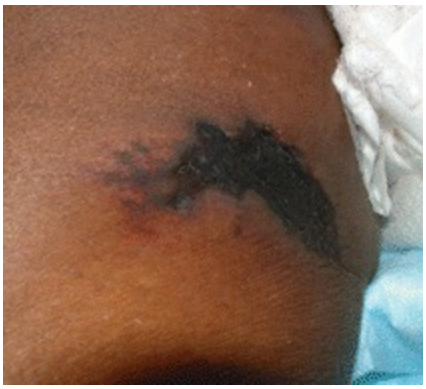
A. Venous insufficiency ulcer
B. Arterial insufficiency ulcer
C. Marjolin's ulcer
D. Kaposi's sarcoma

17. This rapidly progressive and painful ulcer is highly suspicious for:



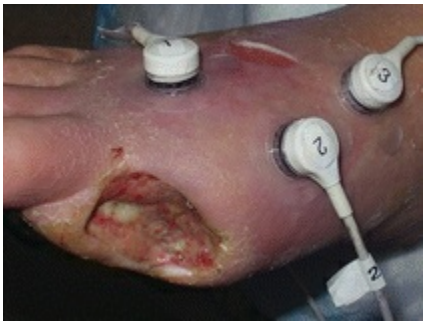
A. pyoderma gangrenosum.
B. basal cell carcinoma.
C. calciphylaxis.
D. vasculitis.

18. Which wound diagnosis is most accurate for this end-stage renal dialysis patient?



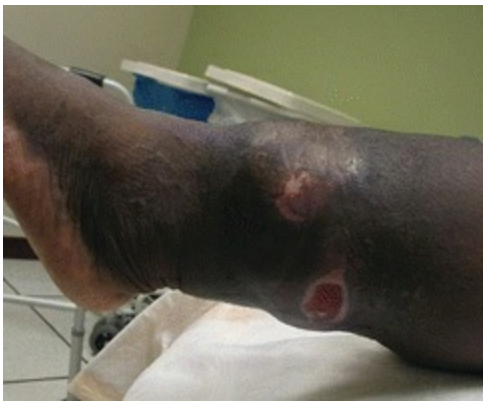
A. Calciphylaxis
B. Calcific uremic arteriolopathy
C. Uremic ulcers
D. Both A and B

19. Which noninvasive test is demonstrated in this photo?



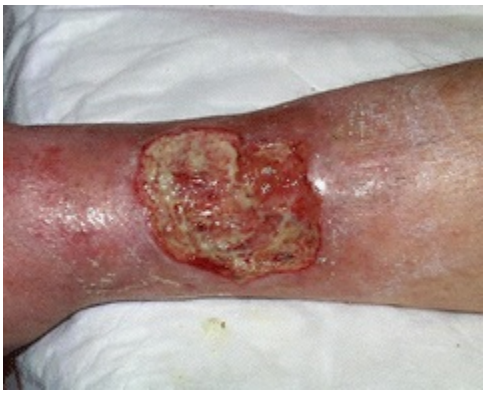
A. Dermal thermography
B. Pulse oximetry
C. Transcutaneous oximetry
D. Skin perfusion pressure

20. This patient presents with a wound that is BEST classified as which of the following?



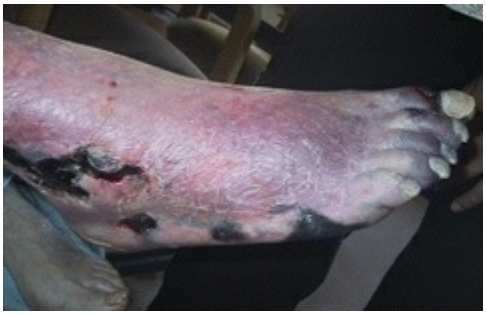
A. Arterial ulcer
B. Venous ulcer
C. Pressure ulcer
D. Atypical ulcer

21. How would you document the percentage of slough tissue in this pressure ulcer?



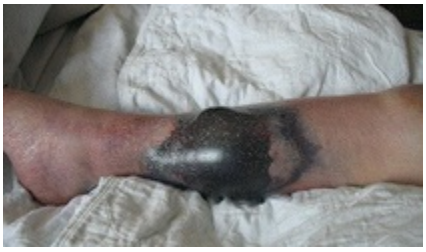
A. 10%
B. 25%
C. 30%
D. 90%

22. Would compression therapy be indicted for this patient?



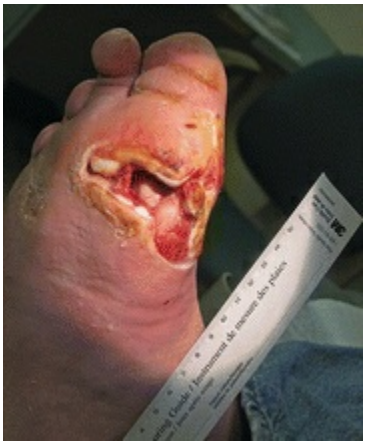
A. Yes
B. No

23. How would you document this wound type?



A. Suspected DTI
B. Hematoma
C. Necrotizing fasciitis
D. Unstageable pressure ulcer

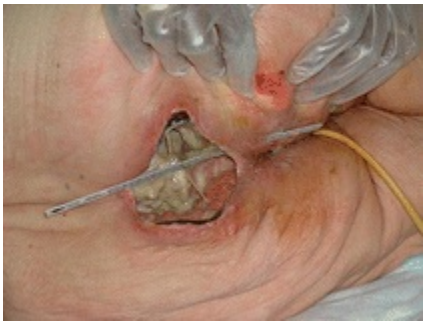
24. What should be included in the plan of care for this patient?



A. Antibiotics

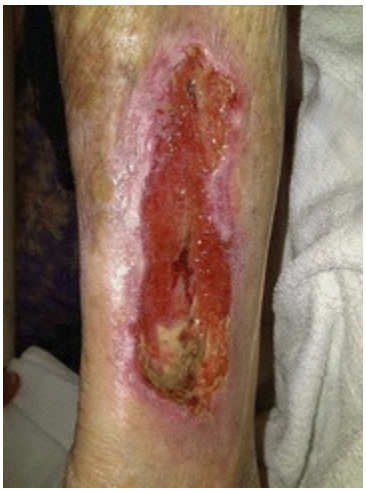
B. Maintenance debridement
C. Pressure off-loading
D. All of the above

25. This wound has a tunnel that should be documented. What position should be used to document the tunneling in this wound?



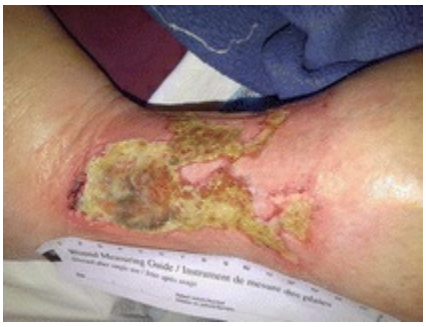
A. 1:00
B. 3:00
C. 5:00
D. 8:00

26. How would you describe the tissue at the wound edges?



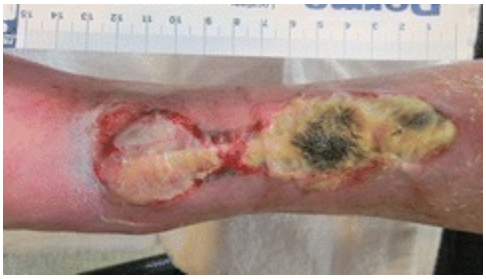
A. Epithelialized wound edges
B. Rolled wound edges
C. Stalled wound edges
D. Necrotic wound edges

27. A priority for initiating treatment of this wound would be:



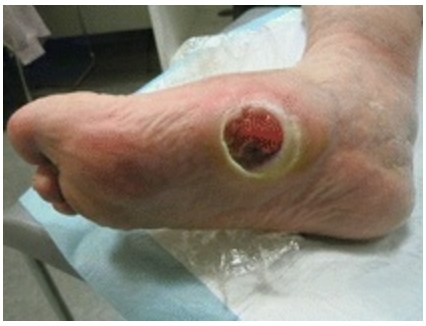
A. applying a dry dressing.
B. applying an emollient.
C. debriding of slough.
D. pending amputation.

28. What structure do you see in this wound?



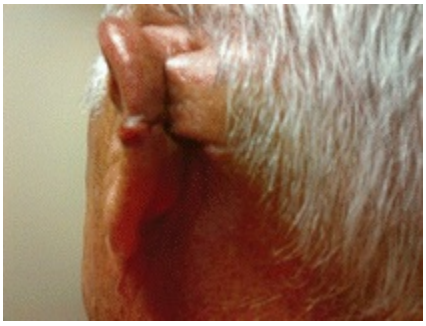
A. Bone
B. Tendon
C. Muscle
D. Cartilage

29. Progressive degeneration of a weight-bearing joint, as depicted in this photo, is called:



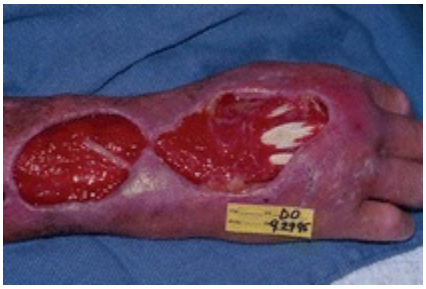
A. Charcot foot.
B. hemosiderin deposit.
C. senile purpura.
D. pressure ulcer.

30. What is the correct documentation for this flap reconstruction called?



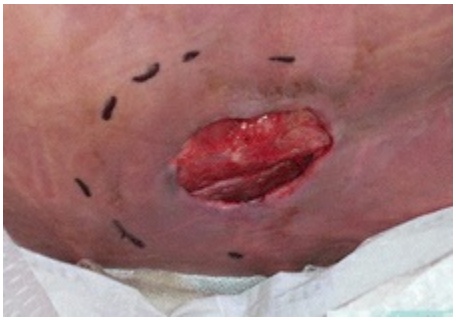
A. Pedicle flap
B. Bone flap
C. Split-thickness flap
D. Express flap

31. Extravasation caused by a chemotherapy agent resulted in this wound. What structure do you see that could impact the healing process?



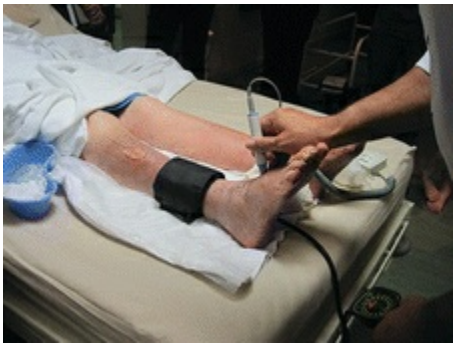
A. Slough
B. Tendon exposure
C. Eschar
D. Cartilage

32. This wound is marked up to show areas of destruction surrounding the wound. This destruction is called:



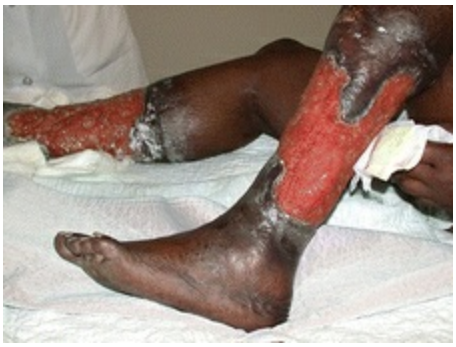
A. slough.
B. tunneling.
C. hypergranulation.
D. undermining.

33. What diagnostic test does this photo represent?



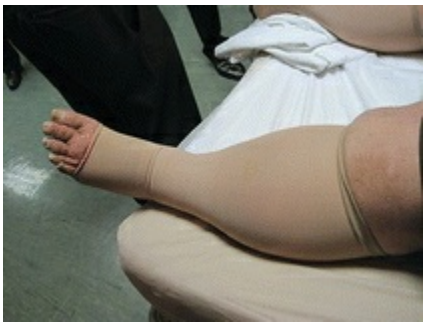
A. Stemmer's testing
B. Monofilament testing
C. Ankle–brachial index testing
D. Waveform testing

34. What type of tissue is primarily depicted in this wound bed?



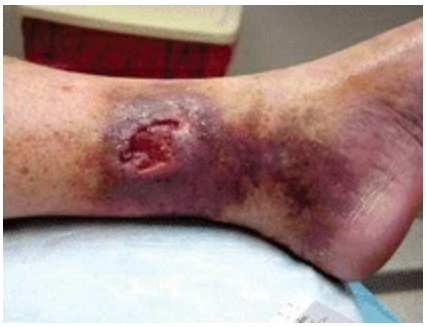
A. Necrosis
B. Eschar
C. Slough
D. Granulation

35. What's wrong with this picture?



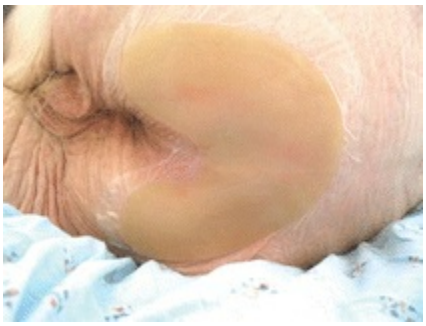
A. Nothing
B. Inappropriate sizing of compression stocking

36. How would you describe the discoloration around this wound?



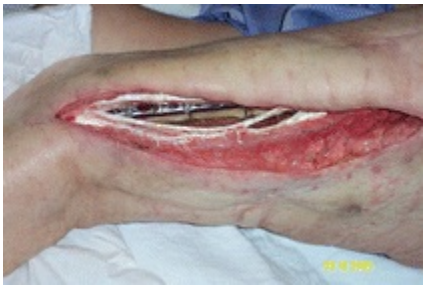
A. Hemosiderin deposits
B. Contact dermatitis
C. Xerosis
D. Gangrene

37. The type of dressing seen in this photo is called:



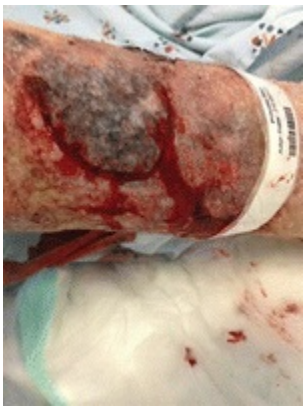
A. Collagen dressing
B. Hydrocolloid dressing
C. Hydrogel dressing
D. Foam dressing

38. A rare but severe surgical complication where the incision opens and the internal muscle/organs and/or hardware can be seen in the incision is called:



A. Deep tissue Injury
B. Dehiscence
C. Evisceration
D. Necrotic tissue

39. What type of dressing would be appropriate for this wound?



A. Hydrogel
B. Foam
C. Dry Gauze
D. Betadine mesh

Thank you to our colleagues for their contributions of photos for this chapter. Photo acknowledgments for this chapter:

Photo 1: Javier La Fontaine, MD
Photo 2: Mary Gloeckner, RN
Photo 3, 34: Mary Sieggreen, RN
Photo 4: Beth Beck, RN
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Photos 11–20: Jeffrey Niezgoda, MD
Photos 21–28, 32: KDS Professional consulting
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Photo 30, 31, 36: Sharon Baranoski, RN
Photo 37: Sharon Baranoski
Photo 38: K.L. Kennedy, RN

Photo 39: M. Gloeckner

Answers Appendix

Chapter 1

1. C. Those with wounds are often viewed as appalling and repulsive.
2. D. Assessment of pain is commonly lacking in wound assessment; size, odor, and drainage are usually assessed.
3. A. The patient's perceptions of well-being should direct quality-of-life treatment decisions.
4. D. All of the above.

Chapter 2

1. C. A is incorrect because it refers to the Medicaid program, which is a collaboration between the federal and state governments to deliver care. B is incorrect because Medicare Part A is for inpatient hospital costs. D is incorrect because Medicare Part B requires the beneficiary to pay a 20% copayment.
2. B. OASIS-C is only used by home health agencies to assess patients and determine reimbursement.
3. A. B is incorrect because the beneficiary must spend 3 consecutive days in the hospital. C is incorrect because skilled nursing services must be needed on a daily basis. D is incorrect because the beneficiary must be admitted within 30 days of hospitalization.
4. C. There are currently no quality initiatives by CMS on turning patients. A, B, and D are all true with regard to CMS. A is true because data on the performance of hospitals, nursing homes, and home health agencies are readily available on the CMS website. B is true because there is tremendous interest at CMS to reward providers of high-quality care, known as pay for performance. D is true because QIOs are emphasizing working with providers to improve care rather than just detecting episodes of poor care.

Chapter 3

1. C. The jury determines who, what, when, why, where, and how—in other words, what happened. The judge interprets the law by instructing the jury about the law to be applied to the facts as it has determined them. The other options are incorrect.
2. D. The plaintiff must prove all four elements in order to prevail at trial.
3. A. Opinions related to patient care aren't proper entries in a medical record. The medical record is a communication tool among practitioners and is best used for the transmittal of factual information. The other options are incorrect.
4. A. Standards must be reviewed and amended to reflect the latest research and practice experience in a treatment area. Standards based on practice experience only and not supported by research may not survive judicial scrutiny at trial and don't offer the patient the best care.
5. C. Suspected DTI damage may occur days before it is visibly evident on the patient. The other options are incorrect.
6. D. An individualized turning schedule based on patient assessment q 4-hour turning is the best way to document the frequency of turning in the medical record. The other options are incorrect because they are rigid and inflexible and do not allow for individualized patient care. Too many variables can impact repositioning to mandate an absolute time frame for all patients.
7. A. Normative policies and procedures describe care that can be realistically and consistently provided. The other options are incorrect. Answer B is the definition of positive policies and procedures; C, policies are not based on staffing ratios; and D has nothing to do with normative policies.

Chapter 4

1. D. The cells of the stratum corneum can shed and look like flakes during routine cleaning activities such as bathing.
2. D. Upon stimulus from the hypothalamus, skin blood vessels will either vasoconstrict (heat needs to be conserved to elevate temperature) or vasodilate (heat needs to be eliminated to lower temperature) depending upon specific needs. Skin can synthesize vitamin D, not vitamin K. Carbon dioxide is eliminated via the lungs. Glucose levels are regulated by the islets of Langerhans in the pancreas, not the Langerhans cells in the skin.
3. D. The entire biology of keratinocytes is dedicated to barrier formation and its maintenance.

4. **D.** ABI is a test used for peripheral vascular disease; it does not tell you about skin assessment. Answers A, B, and C should all be part of a skin assessment.

5. **D.** A 72-year-old female is the oldest, least mobile, and receiving steroids, which are known to further cause thinning of the skin, so her skin is at highest risk.

6. **B**. Answer A is incorrect because there is no tissue loss in type I. Type 3 is incorrect because in category III, there is complete loss of the tissue flap. D is incorrect because type 4 is not part of the classification system.

7. **A.** Nonemollient soaps should be used instead of detergent, which dries the skin. The literature suggests that routine every-other-day bathing for elderly people is adequate (unless the skin is soiled) and can reduce skin tear injury. B, C, and D are all interventions to consider as part of a skin tear protocol.

8. **D.** Emollient moisturizers are a cornerstone in the treatment of xerosis. Answer A is incorrect because daily cleaning of the skin by either showering or bathing is not recommended as it further dries the skin. Answer B is incorrect because a low-pH soap needs to be used as deodorant soaps have a high pH that makes the skin alkaline. Answer C is incorrect because rubbing can irritate dry skin.

9. **C.** Pruritus is the medical term for itchy skin.

10. **B** is the correct definition; A, C, and D refer to other skin injury or conditions. A. Xerosis is dry skin, and D is itchy skin. C. Skin failure is a new concept where the skin dies due to hypoperfusion.

11. **D**. Bulla. A is incorrect because a papule is an elevated, palpable, firm, circumscribed lesion up to 1 cm. B is incorrect. Although a vesicle is a fluid-filled blister, it is only up to 1 cm. C is incorrect as although it is elevated and similar to a vesicle, it is filled with pus.

12. **D** is correct. While all four choices are types of moisture-associated skin damage (MASD), the etiology of all four is different. It is important to differentiate between them. Intertriginous dermatitis (ITD) is the type of MASD caused by trapped perspiration and frictional forces from opposing skin surfaces. While various microorganisms can proliferate, of those which are fungal, Candida can be found in ITD. A is incorrect because peristomal skin damage occurs around an ostomy, and there was no information that this patient had a stoma on their abdomen. B is incorrect, because there was no information that the patient had an abdominal wound. C is incorrect because there is no information that the patient was incontinent of urine or stool.

Chapter 5

1. **B.** Rapid hemostasis is essential as it preserves the integrity of the closed and high-pressure circulatory system to limit blood loss.
2. **D.** Because chronic wounds contain abnormally high levels of proteinases and proinflammatory cytokines, prolonged inflammation is believed to be the most significant factor in delayed healing.
3. **A.** New **TISSUE**, or granulation tissue, grows into the extracellular matrix, which is composed of neovascular tissue, collagens, fibronectin, and proteoglycans.

Chapter 6

1. **D.** Wound assessment involves observation, data collection, and an ongoing evaluation process. Surgical debridement is an intervention for the management of wound care.
2. **B.** A superficial wound involves only the epidermis and the dermis is still intact. A full-thickness wound extends through the dermis. A subdermal wound extends into underlying structures below the skin such as bone, muscle, or tendon.
3. **D.** A sinus tract/tunnel is a channel that involves an area larger than the visible surface of the wound. Maceration is the softening of the surrounding skin usually from exposure to or excess wound drainage. A fistula is an opening between two organs or between an organ and the skin.
4. **C.** Holding the camera at a 90-degree angle rather than perpendicular to the wound plane can cause distortion of the wound size in the photo. A, B, and D are correct techniques that should be part of a wound photo protocol.
5. **A.** Electronic health records and electronic medical records are different. The electronic medical record is a legal record within a special institution, such as a hospital, while electronic health record uses the electronic medical record as a source of information to share patient data across care settings. Options B, C, are D are true.

Chapter 7

1. **B.** The NERDS analysis differentiates infected and critically colonized/biofilm categories based on whether wound size has changed. This makes accurate and consistent measurement of the wound an important aspect of assessment. Purulent exudate as well as necrotic tissue may be

observed in both types of wounds. Purulent exudate is the result of WBCs phagocytosing debris and dead WBCs that have phagocytosed microorganisms. Necrotic tissue supports the growth of bacteria but in itself is not infected because it is dead tissue. Infection can only be present in living tissue. Wound odor is related to the organism present. Malodorous fumes can be present in either type of wound.

2. C. A culture that tells only the organism present is not useful to planning treatment, as the number of organisms must be identified to know whether infection is present (and therefore treatment indicated). Semiquantitative cultures are most often used in clinical practice, although they suffer from limitations, most specifically that they measure surface contaminants and not necessarily organisms deep within the tissues. Quantitative cultures provide a count of the actual number of organisms present in a standard gram of tissue taken from beneath the wound surface in the wound tissue but the recommended level for infection is 10^6.

3. B. Antibiotics target specific intracellular sites. Antiseptics have a broad mechanism of action and so may both kill microorganisms and be toxic to cells essential to wound healing. Resistance to antiseptics is expected; more commonly, organisms develop resistance to antibiotics as they adapt to the antibiotic-specific mechanism of action. Antiseptics can be harmful to cells essential to healing, for example, fibroblasts, and thus interfere with wound repair.

4. C. Weekly sharp debridement is recommended for removal of biofilm to expose the wound bed and any biofilm organisms to treatment as well as to prohibit planktonic organisms from attaching to the wound surface. Data are not sufficient to recommend enzymatic or autolytic debridement. Debridement must be followed by a topical antibiofilm agent, for example, silver and iodine product.

Chapter 8

1. B. Debridement is the removal of debris so that cell movement can be enhanced. A is incorrect because most wounds need debridement to heal; C is incorrect because removing necrotic tissue decreases not increases the wound's biological burden; and D is incorrect because healable wounds need moist healing.

2. A. Exudate is a sign of infection, which signals the need for debridement. Other signs are erythema and fluid wave. The other options are incorrect.

3. D. Pulsed lavage is a method of mechanical debridement. A is incorrect

because collagenases are enzymes; B is incorrect because maggots secrete natural collagenase; and C is incorrect because film dressings are a method of autolytic debridement.

4. A. Because the resident is on Coumadin and bleeding can occur, surgical debridement would be least indicated. Also, the appropriate personnel and equipment may not be available in the patient's long-term care facility. The other options are correct and should be considered within the context of other patient characteristics.

5. A. Time is of the essence, and surgical debridement is the quickest method that can be used with infected wounds. Because the client is hospitalized, the appropriate personnel and equipment to perform this method of debridement are available. The other options are incorrect.

Chapter 9

1. B. A, C, and D are all old concepts of wound management.

2. C. Gauze is a form of dry dressing therapy. A, B, and D are moist wound therapy dressings.

3. B. Nurse preference shouldn't be a parameter of dressing selection. A, C, and D are appropriate dressing parameters.

4. A. Transparent film doesn't absorb fluid. B, C, and D are all advantages of transparent film use.

5. A.

6. D. Apligraf is a bilayered skin substitute. A, B, and C are all moist wound therapy choices.

7. D. Hydrogel amorphous gel has minimal ability to absorb drainage, so it is not indicated for use in heavily draining wounds. A, B, and C are all specifically used for heavily draining wounds.

8. A. Tissue-engineered skin substitutes must be applied to a wound bed that is free of necrotic tissue and infection. B and C are incorrect as rejection of these products is not an issue. Although the patient's weight may be part of the total care plan, it is not essential to evaluate for use of this product.

9. B. Tissue oxygenation is increased with the use of electrical stimulation. A, C, and D are all correct.

10. C. Hyperbaric oxygen therapy causes vasoconstriction not vasodilation in both arterial and venous vessels. A, B, and D are all ways in which hyperbaric oxygen therapy enhances wound healing.

Chapter 10

1. D. *125 ÷ 2.2 = 57 kg × 1.2 – 1.5 = 68.4 g to 85.5 g.*
2. B. The RDN should complete a nutrition assessment and request regular diet based on his individualized needs.
3. C. Spreading out the calories by offering a high-protein, high-calorie snack between meals will ease his feeling of fullness and early satiety.
4. C. *A multivitamin with minerals is recommended if the diet is inadequate or a deficiency is confirmed.*
5. A. *Arginine and glutamine are conditionally indispensable.*

Chapter 11

1. D. Lateral rotation moves side to side to aid pulmonary function, but unless a second feature is present such as low air loss, it's not appropriate for pressure ulcer prevention.
2. D. All are important considerations. A patient at home may have different needs than one in an acute care setting.
3. C. Support surfaces redistribute pressure.
4. D. This is the National Pressure Ulcer Advisory Panel definition of pressure redistribution. Immersion is the depth of penetration into a support surface. Envelopment is the ability of a support surface to conform to the body irregularities. Pressure gradient is a change in pressure over a distance.

Chapter 12

1. D. McCaffery's classic definition of pain is that it's whatever the experiencing person says it is. A is incorrect as research has shown that sudden, severe pain may elevate vital signs, but this only occurs for a short time. B is incorrect as research has shown that patients can sleep even though they have moderate or severe pain. C is incorrect because even though pain may be a consequence of wound care, this isn't a definition of pain.
2. A. The Numeric Pain Intensity Scale is a 10-cm line with the words "no pain" at one end and "worst possible pain" at the other end. B refers to the Faces Pain Rating Scale. C doesn't describe a pain scale. D refers to the WHO analgesic ladder.
3. B. A nonopioid is the drug recommended initially for mild pain. An

adjuvant can be added if there is neuropathic and/or nociceptive pain. A is wrong as drugs are part of the WHO analgesic ladder. C and D are incorrect as they are part of step 2 in the ladder.

Chapter 13

1. B. Unrelieved pressure is the cause of tissue death in pressure ulcers. Incontinence and diabetes mellitus are patient characteristics that may put a patient at risk for pressure ulcers but in and of themselves don't cause pressure ulcers. Heat causes burns, not pressure ulcers.
2. C. Friction has occurred. Electrical stimulation is an adjunct therapy used to heal pressure ulcers. Shear is a type of mechanical trauma caused by tissue layers sliding against each other. Maceration is not a mechanical force caused by dragging the skin across a surface.
3. C. In this ulcer, the tissue destroyed is into the subcutaneous tissue. The newly revised NPUAP classification system now includes that category/stage III pressure ulcers are full-thickness wounds that may have some slough and undermining/tunneling, but bone or muscle is not visible or palpable. The ulcer isn't a stage I because the epidermis is no longer intact. It isn't a stage II because the tissue destroyed is deeper than superficial level (partial thickness) and is well into the subcutaneous tissue and it has slough, there is no slough in a stage II pressure ulcer. It isn't a stage IV because in this ulcer, muscle or bone is not palpable or visible.
4. D. The research-based cut score for onset of pressure ulcer risk for older patients and blacks is 18. With the Braden Scale, scores at or lower than the cutoff score indicate risk for pressure ulcer development. Answers A, B, and C are all wrong answers because they're higher than the cutoff score of 18. With the Braden Scale, low numerical scores indicate a risk for pressure ulcers.
5. C. It is recommended that pressure lifts be done every hour for chair-bound patients. Answer A is incorrect. The exact frequency of turning a patient is not yet known but needs to be individualized. Turn the patient at least every 2 to 4 hours as his or her condition warrants. B is incorrect. Don't use hot water, but rather warm water, and avoid soaps that dry the skin. D is incorrect as there's no need to limit the patient's fluids.
6. A. Depth is not on the PUSH tool to measure pressure ulcer healing. Exudate, tissue type, and length × width are the three variables measured.

Chapter 14

Venous Disease

1. **B.** Venous stasis was thought to cause venous ulcers because of pooled blood in the veins. However, current literature reports that venous hypertension is responsible for increased pressure along the vein wall and in the subcutaneous tissue.
2. **A.** Perfusion of the lower extremity is indirectly measured by the ABI.
3. **C.** Compression is the most important component—the edema must be managed in order for venous and lymphatic ulcers to heal.
4. **C.** Nicotine shouldn't be used in any form. It constricts vessels and contributes to atherosclerosis and venous disease.

Lymphedema

1. **B.** A, C, D are correct statements; B is false. A broad definition of lymphedema is persistent edema lasting longer than 3 months, little or no response to overnight elevation or diuretics, and the presence of skin changes (primarily thickened skin, hyperkeratosis, and papillomatosis). A. is a true statement because in the Third World, the most common cause is infection by the nematode *Wuchereria bancrofti*, which is carried by mosquitoes. In the developed world, damage to lymphatics—usually from cancer treatment—is the most common cause. C is a true statement because there is no cure. D is a true statement because the differential diagnosis of leg enlargement can be challenging since lipedema, a pathological deposition of fat, usually below the waist, may be confused with lymphedema. Although there may be orthostatic edema, the feet are spared. However, lipedema can develop into lymphedema.
2. **C.** Lymphatic flow is determined by several factors, including capillary blood pressure, osmotic pressure, and interstitial fluid pressure (hydrostatic pressure). Anything that increases capillary filtration means that more fluid is in the interstitial space (because of an increase in either the rate of filtration or the total amount of ultrafiltrate, perhaps due to an increase in blood pressure or volume overload). This will cause the interstitial fluid pressure to increase. If the oncotic pressure inside the blood vessels decreases (due, e.g., to decreased albumin), then more fluid will leave the blood vessels for the interstitial space and edema will increase.
3. **C.** Diuretics may cause dehydration and thus increase interstitial oncotic pressure, thereby increasing edema. The lynchpin of therapy is compression with short-stretch garments, which have a low resting pressure (and thus

cannot cause ischemia) but a high working pressure, able to work with the muscle pump as the patient ambulates.

Chapter 15

1. **D.** Varicose veins of the lower legs are an early sign of venous insufficiency, and the presence of venous disease is not a known risk factor for the development of arterial disease. Smoking, hypercholesterolemia, diabetes mellitus, and hypertension are risk factors for arterial disease.
2. **C.** In patients without the ability to heal, antimicrobials that will work with moisture reduction, such as povidone–iodine or chlorhexidine, are a necessary treatment. Other treatments include conservative debridement of the slough, moisture reduction, and antibacterials that work in a dry environment. For silver to be effective in a wound bed, it requires moisture to be converted to the ionized form, and this is contraindicated in ulcers without the ability to heal.
3. **B.** An arterial ulcer by definition is always associated with arterial insufficiency or reduced blood flow (100%). Lipodermatosclerosis and edema are associated with venous ulcers. There is a less common but significant association of arterial ulcers with diabetes (30%) and hypertension in 29% to 39% of patients.
4. **A.** Perfusion of the lower extremity is indirectly measured by the ABI.
5. **C.** Arterial ulcers are associated with arterial insufficiency, and a bypass graft is meant to restore the arterial circulation to the ischemic tissues. The other options are incorrect.

Chapter 16

1. **B.** One-third of people with diabetes are unaware of their condition.
2. **A.** Diabetes mellitus is the single leading cause of lower extremity amputation. B, C, and D are not the single leading cause of amputation in diabetic patients. While these conditions can contribute to complications in the diabetic, they aren't listed as the leading cause for amputation.
3. **B.** Moisturizers shouldn't be applied between the toes—fungal infections can occur. All others are ADA recommendations for good skin care.
4. **A.** Off-loading must be tailored to the individual.
5. **D.** Studies by researchers have found that making walking boots irremovable (nonremovable) that patients had same healing as with the gold standard of off-loading, the total contact cast. The other options are all

removable shoes and research has not demonstrated healing rates comparable to TCC.

Chapter 17

1. C. Persons with heterozygous sickle cell disease receive only one gene for abnormal hemoglobin from one of their parents and are at less risk of ulceration than persons who have abnormal hemoglobin genes from both parents (homozygous). A, B, and D are true statements.
2. A.
3. A. Edema is a key indicator that a person with sickle cell disease may get an ulcer. B is incorrect as hemoglobin A1C is not related to sickle cell disease but to diabetes mellitus. C is incorrect as patients with sickle cell disease often have fever of unknown origin. D. Pain is frequently a major problem for patients with sickle cell ulcers.
4. D. A bone scan is virtually useless in diagnosing osteomyelitis in the sickle cell ulcer patient while an MRI is preferred. A, B, and C are all correct and should be part of the assessment of a patient with a sickle cell ulcer.
5. E. All of the above are all important parts of the holistic plan of care of patients with a sickle cell ulcer.

Chapter 18

1. B. Secondary healing is the simplest method for any wound, but in many deep wounds, secondary healing may be lengthy and result in excessive scarring. Venous stasis ulcers and sternal dehiscence can sometimes heal secondarily, so skin grafting would be more complex. A stage III ulcer should be able to heal with other methods than free flaps, which are the "top of the line" for surgical options.
2. C. Only the radiation of the chest wall is a wound with muscle involvement.
3. D. The loss of arterial inflow will render a flap pulseless (often only by Doppler), so delays in capillary refill are seen first and then pallor. If the early signs are not recognized, the flap can become cyanotic and eventually lose tissue.
4. B. A hospitalized patient often has pressure reduction via beds and chair cushions. In addition, the nurses remind the patient to off-load. Once discharged, the lack of these devices and cues can quickly lead to

deterioration. The other factors can contribute and should be considered as well.

Chapter 19

1. **C.** Effluent from a low-output fistula is less than 200 mL/d, whereas high-output fistulae produce more than 500 mL/d. B is incorrect because external fistulae empty to the environment; internal fistulae empty into an organ. A is incorrect because NPWT can now be used to treat patients with fistulae. D is incorrect.
2. **C.** If the tube is stabilized, the skin is less likely to break down; if adequate hydration is maintained, there will be fewer problems with the tube; nutritional support is the primary goal.
3. **D.** Crohn's disease is an inflammatory bowel disease that affects all layers of the bowel wall, predisposing the patient to fistula formation. A, B, and C are all within normal range.
4. **B.** Using a bedside gravity drainage system will prevent overfilling the pouch and reduce leakage. Answers A and C expose the skin to corrosive fistula drainage. Answer D runs the risk of dehydrating the patient and reducing caloric intake.
5. **D.** Fistulae are named for the anatomy at which the start and end point is located. Fistulae that begin in the small intestine and exit at skin level are termed "enterocutaneous."
6. **A.** Since the pancreas secretes digestive enzymes into the digestive tract, the pH will be alkaline and the effluent will be clear, watery in nature.

Chapter 20

1. **B.** It's critical to recognize when a wound is caused by an etiology other than prolonged pressure, neuropathy, or abnormal vascular supply so that appropriate measures may be undertaken to make a correct diagnosis and provide appropriate therapy. Although an infectious agent that's contagious may be a cause of an atypical ulcer, this isn't common. Although oftentimes specific therapies for atypical wounds exist, these are usually coupled with principles of good wound care, such as compression, off-loading, moist wound healing, and others. Billing for medical therapies is based on Evaluation and Management Codes as opposed to CPT codes.
2. **D.** As a variety of etiologies cause atypical wounds, a variety of techniques are used to confirm these etiologies. Histology is critical for

diagnosing inflammatory, malignant, and infectious causes. Biopsies for tissue culture aid in diagnosing infectious causes, and biopsies for immunofluorescence will aid in diagnosing some inflammatory and autoimmune diseases.

3. **C.** In susceptible people, even minimal trauma to the skin can result in the production of pyoderma gangrenosum lesions, such as pustules and ulcers. This phenomenon is called pathergy. Therefore, debridement of an ulcer secondary to pyoderma gangrenosum may lead to severe worsening of the ulcer.

4. **A.** *Sporothrix schenckii* causes sporotrichosis. *Mycobacterium ulcerans* and *M. marinum* are species of mycobacteria. *Fonsecaea pedrosoi* is a pigment fungi related to chromoblastomycosis.

5. **C.** Cryofibrinogenemia is a painful cutaneous ulceration classified as a vasculopathy. Mycobacterium is a bacterium. Pyoderma gangrenosum is an inflammatory ulcer, and metabolic disease is an uncommon cause of chronic wounds.

6. **A.** Calciphylaxis is the correct answer. Vasculopathy, radiation dermatitis, and chemical burns are all other types of atypical wounds.

7. **B.** Factitial dermatitis is a self-imposed injury usually found in easily accessible areas such as the extremities, abdomen, and anterior chest. The other options are incorrect.

Chapter 21

Spinal Cord Injury Population

1. **C.** Although more males have SCI, gender isn't a major risk factor for pressure ulcer development. Pre-existing conditions, severity of the SCI, and nutrition are all major risk factors for pressure ulcer development.

2. **B.** Urinary tract infection is the most commonly occurring complication of SCI. Pressure ulcer development is second. Fractures and osteoporosis are also other complications that may occur.

3. **D.** A is incorrect as the exact number of hours for a turning and repositioning schedule must be individualized for the person based on the type of support redistribution surface being used and the patient's conditions and comorbidities. B and C are correct and should be part of a prevention plan.

HIV/AIDS Population

1. B. ART-based regimens have contributed to a significant decrease in HIV-associated morbidity and mortality, including many of the cutaneous manifestations of HIV infection. The other options are incorrect.
2. D. Frequent hand washing or using an alcohol- based hand sanitizer is the best method of preventing transmission of community-acquired methicillin-resistant Staphylococcus aureus (CA-MRSA).
3. C. HPV is the causative agent for anal dysplasia and anal carcinoma.

Bariatric Population

1. C. Intertrigo is an inflammation in the skin folds and often presents as a mirror image from the skin in one area touching the other. A, B, and D are skin manifestations seen in bariatric patients.
2. B. Acanthosis nigricans is usually benign but occasionally undergoes malignant changes. Symptomatic treatment is available to promote patient comfort.
3. D. The pannus and other skin folds should be supported. "Off-load" turns are necessary; small shifts may be an adjunct but do not replace turning. The head of the bed may need to be elevated for proper respiration; supporting the arms with pillows will lessen the chance of sliding and sacral shear. Pillows should be placed between the legs to provide air and pressure redistribution and avoid compression and pressure damage from a Foley catheter.

Chapter 22

1. C. Studies support that about 50% of pressure ulcers in children occur under medical devices.
2. D. The Braden Q Scale and Glamorgan Scale are both correct. The Braden Scale was originally designed and used for adults. The Braden Scale is not the same as the Braden Q Scale, which can be used for pediatric patients. The Waterlow Scale and Norton are for adults; however, the Waterlow Pediatric Scale could be used for a 5-year-old child. Remember: Skin assessment is not the same as pressure ulcer risk assessment.

Chapter 23

1. **A.** Palliative care is focused on holistically supporting an individual for comfort rather than care or healing of a wound while improving quality of both living and dying. Palliative care affirms life and views death as part of a normal process and is implemented to neither delay nor speed death. Palliative care provides relief from pain and other symptoms yet is not focused on the complete elimination of these symptoms. Answers B, C, and D do not define palliative care.

2. **D.** A hydrogel dressing that is placed on a wound every 3 days and as needed is recommended in a palliative care patient with a wound, as the hydrogel provides for a moist wound environment, which is soothing and comforting to the patient. It assists in protecting the periwound skin from maceration and is nonadherent, which is desirable. A silver-impregnated or calcium alginate dressing is not necessary unless there is heavy exudate or signs of infection or moderate to severe inflammation. Wet-to-dry dressings that are changed every 4 hours could likely contribute to periwound maceration and cause pain.

3. **B.** Warm rather than cold saline is recommended when irrigating wounds in palliative care patients. Answers A, C, and D are appropriate management strategies.

4. **C.** The skin is moist and desquamated, so it needs protection from injury caused by the edema and wetness. Answer A is incorrect because the skin is compromised and needs care. Answer B is incorrect as the skin is too moist already. Answer D is incorrect because debridement is not indicated. In addition, debridement agents do not need to be applied as frequently as three times per day.

Chapter 24

1. **C.** Diabetic ulcer
2. **B.** Type 2
3. **A.** Arterial disease
4. **D.** Senile purpura
5. **C.** Unstageable
6. **C.** Drug injection injury
7. **B.** Granulation island buds
8. **D.** Ischemia
9. **A.** Lymphedema with fibrosis

10. **D.** Moisture-associated skin damage
11. **B.** Blastomycosis**D.**
12. **D.** Necrotizing fasciitis
13. **C.** Site 3
14. **C.** Neoepithelium
15. **A.** Intact and viable periosteum
16. **D.** Kaposi's sarcoma
17. **A.** Pyoderma gangrenosum
18. **D.** Both A and B
19. **C.** Transcutaneous oximetry
20. **B.** Venous ulcer
21. **D.** 90%
22. **B.** No
23. **B.** Hematoma
24. **D.** All of the above
25. **C.** 5:00
26. **A.** Epithelialized
27. **C.** Debridement of slough
28. **B.** Tendon
29. **A.** Charcot foot
30. **A.** Pedicle flap
31. **B.** Tendon exposure
32. **D.** Undermining
33. **C.** Ankle–brachial index
34. **D.** Granulation
35. **B.** Inappropriate sizing of compression stocking
36. **A.** Hemosiderin deposits
37. **B.** Hydrocolloid dressing
38. **C.** Evisceration
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