

National Athletic Trainers' Association Position Statement: Management of Acute Skin Trauma

Joel W. Beam, EdD, LAT, ATC*;
Bernadette Buckley, PhD, LAT, ATC*;
William R. Holcomb, PhD, ATC, FNATA, FNSCA†;
Mario Ciocca, MD‡

*Clinical and Applied Movement Sciences, Brooks College of Health, University of North Florida, Jacksonville; †School of Kinesiology, University of Southern Mississippi, Hattiesburg; ‡Department of Sports Medicine, University of North Carolina at Chapel Hill



Objective: To present recommendations for the cleansing, debridement, dressing, and monitoring of acute skin trauma in patients.

Background: Acute skin trauma is common during participation in athletic and recreational activities. Clinical decisions and intervention protocols after injury vary among athletic trainers and are often based on ritualistic practices. An understanding of cleansing, debridement, and dressing techniques; clinical features of infection and adverse reactions; and monitoring of acute skin trauma is critical for certified athletic trainers and other allied health and medical professionals to

create a local wound environment that promotes healing and lessens the risk of complications.

Recommendations: These guidelines are intended to provide the certified athletic trainer and others participating in athletic health care with specific knowledge about and recommendations for the management of acute skin trauma.

Key Words: abrasions, avulsions, blisters, incisions, lacerations, punctures, cleansing, debridement, nonocclusive dressings, occlusive dressings, infection, adverse reactions

Traumatic injury to the skin is common among athletes participating in all sports.¹ The exact frequencies of abrasions, avulsions, blisters, incisions, lacerations, and punctures are difficult to calculate because many patients do not seek medical attention after injury; for others, their activity level is initially unaffected, and the injury is not recorded on surveillance reports. Unreported skin trauma and inappropriate wound management can result in delayed healing, cross-contamination, bacterial colonization, and infection, adversely affecting the overall health and playing status of the patient. Managing acute skin trauma through appropriate cleansing, debridement, and dressing techniques can create an environment conducive to healing and lessen the risk of complications.^{2,3} Wound-management techniques have undergone drastic changes over the last 50 years, and other allied health care professions, organizations, and facilities have developed guidelines that serve as standards of care.⁴⁻⁶ However, guidelines for the management of acute skin trauma by athletic trainers (ATs) are limited in the literature.^{1,7} The development and implementation of cleansing, debridement, and dressing techniques for acute skin trauma are

critical for ATs to successfully deliver health care services to patients. The following review and recommendations provide information on the management of acute skin trauma and guidelines for ATs and other allied health and medical professionals who care for patients.

RECOMMENDATIONS

This position statement is based on current research and literature with regard to the cleansing, debridement, dressing, and monitoring of acute skin trauma. We independently categorized the studies and literature using the Strength of Recommendation Taxonomy (SORT) developed by the American Academy of Family Physicians.⁸ The taxonomy grades the quality of the data from the literature (level of evidence) and provides strength ratings for the suggested recommendations with the letter *A*, *B*, or *C* (Table 1).

The recommendations have been organized into the following categories: wound cleansing, debridement, dressings, identification of infection and adverse reactions,

Table 1. Strength of Recommendation Taxonomy^{a,b}

Strength Rating	Definition
A	Recommendation based on consistent and good quality experimental evidence (morbidity, mortality, exercise and cognitive performance, physiologic responses).
B	Recommendation based on inconsistent or limited quality experimental evidence.
C	Recommendation based on consensus; usual practice; opinion; disease-oriented evidence ^b ; case series or studies of diagnosis, treatment, prevention, or screening; or extrapolations from quasi-experimental research.

^a Reprinted with permission from Ebell MH, Siwek J, Weiss BD, et al, "Strength of Recommendation Taxonomy (SORT): A Patient-Centered Approach to Grading Evidence in the Medical Literature," February 1, 2004, Vol 69, No 3, American Family Physician Copyright ©2004 American Academy of Family Physicians. All Rights Reserved.

^b Patient-oriented evidence measures outcomes that matter to patients: morbidity, mortality, symptoms improvement, cost reduction, and quality of life. Disease-oriented evidence measures intermediate, physiologic, or surrogate end points that may or may not reflect improvements in patient outcomes (eg, blood pressure, blood chemistry, physiologic function, pathologic finding).

follow-up, and supplies for athletic training facilities and kits.

Cleansing

1. The wound bed and adjacent periwound tissues should be thoroughly cleansed as soon as possible after acute skin trauma.^{9–11} *Strength of recommendation: C*
2. After initial cleansing, the wound should be cleansed only when clinically necessary (eg, visibly contaminated, clinically infected).^{9,12–18} *Strength of recommendation: C*
3. Irrigation can be used to cleanse superficial- to full-thickness abrasions, incisions, and lacerations.^{19,20} *Strength of recommendation: B*
 - a. To effectively remove wound debris, irrigation pressure should range between 4 and 15 pounds per square inch (psi; 27.58–103.42 kPa).^{11,19,21–23} *Strength of recommendation: B*
 - b. A range of 7 to 11 psi (48.26–75.84 kPa) can be achieved with a 35-mL syringe and an 18- to 20-gauge needle, needle hub, or plastic cannula.^{11,19,23,24} *Strength of recommendation: B*
4. Showering can be used to cleanse postoperative incisions.^{19,20,25} *Strength of recommendation: A*
5. Hydrotherapy (eg, whirlpool baths and soaks) may be used during cleansing to hydrate a wound.^{11,24,26} *Strength of recommendation: C*
6. Scrubbing or swabbing of the wound bed should be avoided because it may be ineffective in reducing bacterial counts and can further contaminate and damage the granulation tissue.^{9,13,24,26–29} *Strength of recommendation: C*
7. Normal saline and potable tap water should be used as cleansing agents with superficial- to full-thickness abrasions, incisions, and lacerations.^{19,20,25,30–33} *Strength of recommendation: A*
8. Tap water should be avoided in the presence of exposed bone or tendon, although the basis for this guideline is unclear.³⁴ *Strength of recommendation: C*

9. Antiseptics (eg, povidone-iodine, hydrogen peroxide) should be used with caution as cleansing agents because some may be toxic to tissues.^{19,35–43} *Strength of recommendation: B*
10. Irrigation with 1% povidone-iodine can be used to cleanse acute, traumatic, contaminated wounds.¹⁹ *Strength of recommendation: B*
11. The cleansing solution should be delivered to the wound at a temperature between 98.6°F and 107.6°F (37°C and 42°C).^{13,14,44,45} *Strength of recommendation: C*

Debridement

12. The area of acute skin trauma should be thoroughly cleansed and debrided before dressings are applied.^{4,46–53} *Strength of recommendation: B*
13. Debridement should continue until well-vascularized, healthy granulation or epithelial tissue is exposed.⁵⁴ *Strength of recommendation: C*
14. The method of debridement should be based on the type of wound, amount and type of debris, training and expertise of the health care provider, and cost-effectiveness and time-effectiveness of the technique.^{55,56} *Strength of recommendation: C*
15. Irrigation can serve as an extension of cleansing for debridement of superficial- to full-thickness abrasions, avulsions, blisters, incisions, lacerations, and punctures.^{3,57–59} *Strength of recommendation: C*
 - a. Irrigation pressure should range between 4 and 15 psi (27.58–103.4 kPa).^{3,57,59–61} *Strength of recommendation: C*
 - b. To avoid driving debris and contaminants deeper into puncture wounds, an irrigation pressure of 2 to 4 psi (13.79–27.58 kPa) should be used.^{3,57,59–61} *Strength of recommendation: C*
16. Hydrotherapy debridement (eg, whirlpool baths and soaks) should be avoided because it presents a risk of cross-contamination and is neither cost-effective nor time-effective.^{62,63} *Strength of recommendation: C*
17. Wet-to-dry debridement should be avoided because tissue removal is nonselective and painful.^{54,55,57,59,64–70} *Strength of recommendation: C*
18. Wet-to-moist debridement may be used for superficial- to full-thickness abrasions, avulsions, blisters, incisions, and lacerations and with the formation of eschar. However, care should be taken to protect healthy granulation tissue.⁵⁹ *Strength of recommendation: C*
19. Scrubbing should be used only for superficial- to partial-thickness abrasions, avulsions, blisters, incisions, and lacerations contaminated with large quantities of small debris (eg, sand, grass, clay, asphalt).⁶⁶ *Strength of recommendation: C*
20. Conservative sharp debridement can be used after cleansing to remove loosely adhering, devitalized tissue from superficial- to full-thickness abrasions, avulsions, blisters, incisions, and lacerations.^{71,72} *Strength of recommendation: C*
21. Chemical debridement (eg, sodium hypochlorite, hydrogen peroxide, silver) should be avoided because some elements and compounds may damage viable tissue.^{71,73,74} *Strength of recommendation: C*

Table 2. Sample Dressings

Brand Name	Type	Manufacturer
Allewyn	Foam	Smith & Nephew Pty, Limited, Mount Waverley, Victoria, Australia
Aquaflor	Hydrogel	Covidien, Mansfield, MA
Aquaheal	Hydrogel	Spenco Medical Corporation, Waco, TX
Bioclusive	Film	Johnson & Johnson, Medical LTD, Ascot, United Kingdom
Dermabond	Dermal adhesive	Ethicon, Somerville, NJ
Duoderm	Hydrocolloid	ConvaTec, Princeton, NJ
Histoacryl	Dermal adhesive	Aesculap AG, Tuttlingen, Germany
Leukostrips	Wound-closure strip	Smith & Nephew Pty
PolyMem	Foam	Ferris Mfg, Burr Ridge, IL
Polyskin II	Film	Covidien
SteriStrips	Wound-closure strip	3M Company, St Paul, MN
Tegaderm	Alginate, film, foam, hydrocolloid, and hydrogel	3M Company
Ultec Pro	Alginate/hydrocolloid	Covidien

22. Autolytic debridement should be used for selective proteolytic digestion of necrotic tissue.^{51,55,57,58,64,71,75–85} *Strength of recommendation: B*
- Autolytic debridement can be used with the following wounds:
 - Postoperative incisions.⁷⁹ *Strength of recommendation: B*
 - Superficial- to full-thickness abrasions, avulsions, and lacerations. *Strength of recommendation: C*
 - Superficial- to full-thickness blisters after removal of the necrotic roof with conservative sharp debridement.⁷¹ *Strength of recommendation: C*
 - Superficial- to partial-thickness punctures. *Strength of recommendation: C*
 - Autolytic debridement should be avoided with infected wounds.^{57,66,75} *Strength of recommendation: C*

Dressings

23. After thorough cleansing and debridement, an area of acute skin trauma should be covered with an appropriate dressing until fully healed (Table 2).^{2,86–90} *Strength of recommendation: B*
24. Nonocclusive dressings can be used as primary dressings with the following wounds (Table 3):
- Woven and nonwoven gauze for clinically infected abrasions, avulsions, blisters, incisions, lacerations, or punctures.^{91,92} *Strength of recommendation: C*
 - Woven, nonwoven, and impregnated gauze for puncture wounds that have cavities.^{93–96} *Strength of recommendation: C*
 - Wound-closure strips with superficial, linear lacerations and postoperative incisions under minimal static and dynamic tension.^{3,97,98} *Strength of recommendation: A*
 - Woven gauze with superficial- to full-thickness abrasions, avulsions, blisters, incisions, and lacerations to achieve wet-to-moist debridement.^{55,68,91,94} *Strength of recommendation: C*

- Woven and nonwoven gauze, nonadherent pads, and adhesive strips or patches for superficial- to partial-thickness abrasions, avulsions, and blisters and superficial-thickness incisions, lacerations, and punctures as a temporary dressing and on irregular body surfaces.^{91,95,99} *Strength of recommendation: C*
25. Woven or nonwoven gauze, nonadherent pads, and adhesive strips or patches can be used as secondary dressings to absorb moderate-to-heavy exudate and provide additional protection for superficial- to full-thickness abrasions, avulsions, blisters, lacerations, punctures, and traumatic and postoperative incisions.^{91,94,100,101} *Strength of recommendation: C*
26. Occlusive dressings should be used as primary dressings with the following wounds (Table 4):
- Alginates, films, foams, hydrocolloids, and hydrogels for superficial- to partial-thickness abrasions, avulsions, blisters, incisions, lacerations, and punctures.^{2,90,91,95,102–112} *Strength of recommendation: A*
 - Alginates, foams, and hydrocolloids for partial- to full-thickness abrasions, avulsions, blisters, lacerations, and traumatic and postoperative incisions.^{2,90,91,102,106,109–117} *Strength of recommendation: A*
 - Dermal adhesives for partial- to full-thickness lacerations and traumatic and postoperative incisions in areas of low skin tension.^{97,98,118,119} *Strength of recommendation: A*
 - Alginates and foams (nonsilver or silver impregnated) or silver-impregnated films and hydrocolloids for contaminated and clinically infected abrasions, avulsions, blisters, incisions, and lacerations.^{91,94,120,121} *Strength of recommendation: C*
27. Films and hydrocolloids can be used as secondary dressings to provide impermeability to microorganisms, improve adherence, and promote a moist wound environment for superficial- to full-thickness abrasions, avulsions, blisters, lacerations, punctures, and traumatic and postoperative incisions.* *Strength of recommendation: C*

Identification of Infection and Adverse Reactions

28. The athletic training staff should monitor the patient and wound area for clinical features of wound infection, including fever, pain, edema, erythema, warmth, wound dehiscence, and delayed wound healing.^{124–128} *Strength of recommendation: C*
29. For treatment of bacterial infections, refer to the National Athletic Trainers' Association position statement on skin diseases.¹²⁹ *Strength of recommendation: B*
30. Infection rates in patients with acute skin trauma can be reduced with appropriate cleansing, debridement, and dressing techniques.^{39,125,130–132} *Strength of recommendation: B*
31. Skin antisepsis is effective in reducing the incidence of surgical-site infections in incisions and lacerations.¹³³ *Strength of recommendation: A*

*References 91, 101, 105, 108, 109, 113, 114, 116, 122, 123.

Table 3. Nonocclusive Dressing Indications

Indications	Primary Dressing	Secondary Dressing	Other Considerations
Clinically infected abrasions, avulsions, blisters, incisions, lacerations, and punctures	Woven and nonwoven gauze	Woven or nonwoven gauze; nonadherent pads; adhesive strips or patches; adhesive gauze; nonadherent, self-adherent, and adherent tapes and wraps	Daily dressing changes required
Punctures with cavities	Woven, nonwoven, and impregnated gauze	Woven or nonwoven gauze; nonadherent pads; adhesive strips or patches; adhesive gauze; nonadherent, self-adherent, and adherent tapes and wraps	Daily dressing changes required
Superficial, linear lacerations and postoperative incisions	Wound-closure strips	Woven or nonwoven gauze; nonadherent pads; adhesive strips or patches; nonadherent, self-adherent, and adherent tapes and wraps	Use only on wounds with minimal static and dynamic tension
Superficial- to full-thickness abrasions, avulsions, blisters, incisions, and lacerations for wet-to-moist debridement	Woven gauze	Woven or nonwoven gauze; nonadherent pads; nonadherent, self-adherent, and adherent tapes and wraps	Use care to protect healthy granulation tissues
Superficial- to partial-thickness abrasions, avulsions, blisters, superficial-thickness incisions, lacerations, and punctures as temporary dressings	Premoistened woven and nonwoven gauze; nonadherent pads; adhesive strips or patches	Woven or nonwoven gauze; nonadherent pads; adhesive strips or patches; adhesive gauze; nonadherent, self-adherent, and adherent tapes and wraps	Follow-up in athletic training facility for comprehensive management
Superficial- to partial-thickness abrasions, avulsions, blisters, superficial-thickness incisions, lacerations, and punctures on irregular body areas	Woven and nonwoven gauze; nonadherent pads; adhesive strips or patches	Woven or nonwoven gauze; nonadherent pads; adhesive strips or patches; adhesive gauze; nonadherent, self-adherent, and adherent tapes and wraps	Daily dressing changes required

32. Oral antibiotics should not be used prophylactically unless the abrasion, avulsion, blister, incision, laceration, or puncture is heavily contaminated.^{134,135} *Strength of recommendation: A*
33. Topical antimicrobial agents can reduce infection rates in acute skin trauma but should be used judiciously given the emergence of resistant bacterial strains.^{135–142} *Strength of recommendation: A*
34. The patient should be monitored for the development of adverse reactions stemming from the use of some cleansing solutions, topical antimicrobial agents, and

- nonocclusive and occlusive dressings.^{143–147} *Strength of recommendation: C*
35. The patient's health status (eg, malnutrition, smoking, diabetes) may contribute to the development of adverse reactions and should be considered.^{130,148–161} *Strength of recommendation: B*
36. The athletic training staff should monitor the patient and wound area for clinical features of adverse reactions, including erythematous rash, eczematous reaction, vesicles, white discoloration, tenderness, nodularity, burning, pruritus, or systemic reactions such as urticaria and anaphylaxis.^{143–146,162–165} *Strength of recommendation: C*

Table 4. Occlusive Dressing Indications

Indications	Primary Dressing	Secondary Dressing	Other Considerations
Superficial- to partial-thickness abrasions, avulsions, blisters, incisions, lacerations, and punctures	Alginates, films, foams, hydrocolloids, and hydrogels	Films; woven or nonwoven gauze; adhesive gauze; nonadherent, self-adherent, and adherent tapes and wraps	Minimal exudate: films and hydrogels; moderate exudate: hydrogels and hydrocolloids; heavy exudate: alginates and foams
Partial- to full-thickness abrasions, avulsions, blisters, lacerations, traumatic and postoperative incisions	Alginates, foams, and hydrocolloids	Films; woven or nonwoven gauze; adhesive gauze; nonadherent, self-adherent, and adherent tapes and wraps	Moderate exudate: hydrocolloids; heavy exudate: alginates and foams
Partial- to full-thickness lacerations and traumatic and postoperative incisions	Dermal adhesives	Films; hydrocolloids; woven or nonwoven gauze; nonadherent pads; nonadherent, self-adherent, and adherent tapes and wraps	Use only in areas of low skin tension
Clinically infected abrasions, avulsions, blisters, incisions, and lacerations	Alginates and foams, silver-impregnated alginates, films, foams, and hydrocolloids	Films; woven or nonwoven gauze; adhesive gauze; nonadherent, self-adherent, and adherent tapes and wraps	Minimal exudate: films; moderate exudate: hydrocolloids; heavy exudate: alginates and foams; daily dressing changes required

Table 5. Recommended Duration of Wound Dressing Usage^a

Dressing Type	Duration, d
Woven, nonwoven, and impregnated sterile gauze; nonadherent pads; and adhesive strips and patches ^{87,91,93,94,105,113,168}	1
Wound-closure strips ¹⁷⁴	5–10
Alginates ¹⁰⁵	≤7
Films and foams ^{91,94,105,122,170}	3–7
Hydrogels ^{94,105,122}	1–7
Hydrocolloids ^{94,105,122,170}	5–7
Dermal adhesives ^{171,172}	5–10

^a Wounds without dressing-integrity problems (eg, wrinkling or bunching, channel formation, separation from periwound tissues, strike-through, leakage of exudate) or clinical features of adverse reactions or infection.^{89,91,94}

37. Treatment of the adverse reaction should consist of identifying the reaction, removing the causative agent, and directing appropriate measures at the reaction.^{143–146,162–165}
Strength of recommendation: C
38. Criteria for physician referral include
- Any evidence of deeper injury that may require repair, evaluation for nerve or tendon damage, or concern for heavy contamination. *Strength of recommendation: C*
 - Wounds that develop erythema, warmth, edema, drainage, pain, or rash or demonstrate delayed healing. *Strength of recommendation: C*

Follow-Up

39. The athletic training staff should visually inspect the patient, wound area, and dressing daily throughout the healing process.
- The visual inspection should include the patient, wound bed, and periwound tissues for the presence of adverse reactions and infection. If any signs or symptoms are present, the patient should be referred to a physician. *Strength of recommendation: C*
 - Frequency of dressing changes varies based on the type of dressing (Table 5). A dressing change is warranted with evidence of dressing channel formation, separation from periwound tissues, significant exudate accumulation, strike-through, leakage, or wound desiccation.^{89,91,94,166,167} *Strength of recommendation: C*
 - Dressing transitions are required as reductions in exudate and new tissue growth occur during healing.[†] *Strength of recommendation: C*
40. Patients should be educated on dressing guidelines to increase compliance.
- Patients should follow instructions from the athletic training staff on dressing changes. Occlusive dressings can stay in place over wounds for longer periods than nonocclusive dressings.^{91,94,105,122,170–172} *Strength of recommendation: C*
 - Patients will notice changes in the dressing over time. An accumulation of moisture beneath occlusive dressings may be visually detected or demonstrated by expansion of the dressing over the wound bed and

should not be confused with infection.^{91,103,105,173}

Strength of recommendation: C

- c. Patients must immediately report dressing channel formation, separation from periwound tissues, seal breach, strike-through, or leakage or signs or symptoms of adverse reactions and infection.^{89,91,94,166,167}

Strength of recommendation: C

Supplies for Athletic Training Facilities and Kits

41. Supplies to manage acute skin trauma should be available in the athletic training facility.
- Cleansing: normal saline, potable tap water, 35-mL syringe, 18- to 20-gauge needle hub or plastic cannula, antiseptic skin cleanser, and clean basin or cup.
 - Debridement: normal saline, potable tap water, 35-mL syringe, 18- to 20-gauge needle hub or plastic cannula, woven gauze, high-porosity sponge or surgical scrub brush, and sterile scissors and tweezers.
 - Dressings: nonocclusive dressings (woven, nonwoven, and impregnated sterile gauze, nonadherent pads, adhesive strips and patches, and wound-closure strips), occlusive dressings (alginates, films, foams, hydrogels, hydrocolloids, and dermal adhesives), adhesive gauze, and nonadherent, self-adherent, and adherent tapes and wraps.
 - Miscellaneous: sterile or clean (or both) drapes or towels, biohazard container, boxed gloves, face or eye shields (or both), and topical antibiotics.
42. Supplies to manage acute skin trauma should be available in athletic training kits.
- Cleansing: normal saline, potable tap water, 35-mL syringe, 18- to 20-gauge needle hub or plastic cannula, and clean basin or cup.
 - Debridement: same as cleansing.
 - Dressing: nonocclusive dressings (woven and nonwoven sterile gauze, nonadherent pads, and adhesive strips and patches), adhesive gauze, and nonadherent, self-adherent, and adherent tapes and wraps.
 - Miscellaneous: boxed gloves, face or eye shields (or both), and biohazard container.

BACKGROUND

Acute skin trauma is a disruption of the integrity of the epidermis, dermis, or subcutaneous tissues (or a combination of these). Acute wounds are characterized based on the mechanism of injury and resultant tissue damage and include traumatic abrasions, avulsions, blisters, lacerations, punctures, and traumatic and postoperative incisions. The mechanisms of injury of acute skin trauma are shear and tensile forces and tensile loads.¹⁷⁴ Acute wounds can also be characterized by the amount of tissue damage.¹⁷⁵ *Superficial-thickness wounds* involve damage to the superficial epidermis. *Partial-thickness wounds* extend through the epidermis and into the superficial dermis. *Full-thickness wounds* extend through the epidermis and dermis and into the subcutaneous adipose tissue. Acute wounds proceed through an orderly and timely reparative process of inflammation, proliferation, and remodeling that results in restoration of anatomic and functional integrity and wound appearance.¹⁷⁶ Some have characterized acute

†References 91, 94, 100, 104, 108, 168, 169.

wounds as those that heal themselves within 4 to 6 weeks.¹²⁴

THE EVIDENCE AND LITERATURE REVIEW

Cleansing

Wound cleansing, considered a critical part of the management of acute skin trauma, is the process of applying a nontoxic solution to aid in the removal of exudate, bacteria, foreign debris, and dressing residue to create an environment conducive for healing.^{39,177} Acute wounds are initially considered to be contaminated; cleansing is necessary to remove any debris and facilitate healing.^{9,11,17} After the initial cleansing, it may not be necessary to cleanse wounds at every dressing change but rather to rehydrate a wound to create a moist environment, visualize and assess the wound, minimize trauma during adherent dressing removal, or promote patient comfort.^{9,12–18,178} If signs of clinical infection are present, then continued wound cleansing is necessary.

Technique. The selection of an appropriate cleansing technique is necessary to create an optimal environment for wound healing (ie, a moist, clean, warm environment). The more commonly used techniques include irrigation, showering, hydrotherapy (eg, whirlpool baths and soaks), and scrubbing and swabbing.

Irrigation. *Irrigation*, the steady flow of solution across the wound surface, is the preferred method of cleansing.^{11,39,179} The purpose of irrigation is to remove loose debris and excess wound secretions to create an optimal healing environment. This method allows for newly granulating tissue to be preserved, bacteria and debris to be effectively removed, and the comfort and convenience of the patient to be ensured. The potential risks associated with this technique include splash back, additional trauma, and bacteria driven into deeper tissues if the pressure is excessive.¹¹

Although many irrigation recommendations are based on trials involving chronic wounds, investigators^{11,19–25,29,39} agree that high-pressure irrigation (eg, 4–15 psi [27.58–103.42 kPa]) is the best practice for cleansing superficial- to full-thickness abrasions, incisions, and lacerations. Authors¹⁹ of an evidence-based medicine review found that a pressure of 13 psi (89.63 kPa) was effective in reducing infection and inflammation in lacerations and chronic wounds among children and adults, although no consensus exists as to the optimal pressure that should be used. Researchers²⁴ agree that low-pressure irrigation (eg, <4 psi [27.58 kPa]) is not as effective in cleansing and serves only to moisten the wound bed. Pressures greater than 25 psi (172.4 kPa) may be necessary to debride a wound but are not recommended for routine cleansing, as they may damage healthy granulation tissue.^{13,24}

The equipment commonly used for irrigation includes bulb syringes, pressurized canisters, and syringes with an attached needle or catheter. A 35-mL syringe with an 18- to 20-gauge needle, needle hub, or plastic cannula exerts pressures in the range of 7 to 11 psi (48.26–75.84 kPa), delivering a solution without damage to the tissue.^{11,19,23,24}

Showering. *Showering* is effective for larger traumatic wounds, although the pressure is rarely controlled. Authors of a Cochrane review²⁰ examining the effects of postop-

erative showering versus no showering found strong evidence of no difference in the infection and healing rates of incisions. However, investigators^{19,20} reported that patients derived a sense of health and well-being from showering.

Hydrotherapy. Whirlpool baths can be used as an aggressive form of cleansing; the turbulence of the water dislodges debris from the wound bed. More often used for chronic wounds, bathing in lukewarm tap water has become an increasingly common practice, although its role may be considered more for debridement than cleansing.^{11,24,26} Researchers¹⁹ suggested that whirlpool therapy may reduce pain and inflammation in surgical incisions during the first 72 postoperative hours. Clinically, this method of cleansing may be used to hydrate a wound, even though concerns and potential risks include disrupting the moisture balance of the wound bed, macerating periwound tissues, and impairing healing by introducing microorganisms from the immersion fluid.¹¹

Scrubbing and Swabbing. *Scrubbing and swabbing* involve the use of a material (eg, gauze, foam) to wipe the surface of the wound in a systematic manner. This method lacks the evidence necessary to show that it creates the optimal wound environment: scrubbing and swabbing redistribute bacteria over the wound as opposed to removing them.^{9,13,24,26–29} Cotton wool fiber remnants from woven gauze often contaminate the wound and have resulted in nonwoven swabs being the material of choice for swabbing.^{9,13,29} Swabbing can be used to cleanse the periwound tissues or wounds with loose necrotic tissue or slough (moist necrotic tissue).^{26,27} However, others^{9,13,24,26–29} caution against using the swabbing technique on the wound bed because of residual damage to the granulating tissue. Overall, no evidence supports or refutes scrubbing and swabbing to cleanse wounds.¹⁹

Solutions. The selection of an appropriate nontoxic solution to remove debris and create an environment to promote healing is a critical component of wound cleansing. Various cleansing solutions are recommended for their therapeutic value.¹⁸⁰ The characteristics of an ideal solution are nontoxicity to human tissue, effectiveness in the presence of organic material, ability to reduce the number of microorganisms, low likelihood of causing sensitivity reactions, cost-effectiveness, availability, and shelf-life stability.^{41,178,181} Based on these characteristics, many authors^{7,12,20,182} consider normal saline the most appropriate cleansing solution for acute skin trauma, although the use of potable tap water has become more widely accepted.

Normal Saline and Potable Tap Water. In a number of evidence-based medicine reviews, researchers^{19,20,25,30–33} have examined the effectiveness of normal saline and potable tap water on rates of infection and healing in acute wounds. Good evidence shows that effective cleansing can be achieved with normal saline or potable tap water. In a Cochrane review²⁰ comparing normal saline and tap water as cleansers, the incidence of infections among various acute wounds and sutured lacerations in adults was reduced with the use of tap water. It is notable that the solutions were at different temperatures in the trials: normal saline at room temperature and tap water at 98.6°F (37°C). However, water from a properly treated supply and run from the tap for a few minutes before use does not increase the risk of

contamination and infection.¹⁷⁸ Tap water has the advantages of being efficient, cost-effective, and accessible.²⁰ Yet tap water should not be used to cleanse wounds in which tendon or bone is exposed. Normal saline is the recommended solution for these wounds.³⁴

Antiseptics. Topical antiseptics are antimicrobial agents that kill or reduce the number of microorganisms that may impede wound healing. Commonly used solutions include povidone-iodine (eg, Betadine; Purdue Products LP, Stamford, CT) and hydrogen peroxide.^{36,38–41,183,184} However, the use of antiseptics as prophylactic antimicrobial agents for acute skin trauma has been controversial for many years.^{35–37,40,184}

Several investigators^{19,37} have examined the effects of povidone-iodine and the product Betadine on rates of infection and healing. In 1 evidence-based medicine review,¹⁹ infection rates were compared between acute wounds that were cleansed with 1% povidone-iodine or with normal saline. Among contaminated postoperative incisions and traumatic lacerations, 1% povidone-iodine was favored over normal saline. An individual trial in this review¹⁹ examining lacerations demonstrated no differences in infection rates between 1% povidone-iodine and normal saline. Authors³⁷ of a more recent evidence-based medicine review examined the use of iodine and other local wound care methods (eg, honey, silver-impregnated dressings) on postoperative incisions and various acute wounds and noted no differences in rates of infection or healing. An additional narrative review³⁵ examining the effects of povidone-iodine on healing in animal and human wound models has raised questions about the benefits of the solution; delayed wound healing, reduced wound strength, and increased rates of infection were noted when acute skin trauma was treated with the antiseptic. In a small investigation, researchers⁴¹ demonstrated the safe use of antimicrobials in diluted concentrations. A 1:10 diluted solution of Betadine was effective against bacteria and not harmful to human fibroblasts.⁴¹ A 1:5 diluted solution of Betadine was toxic to fibroblasts and, therefore, one may conclude that the commercially produced solution of Betadine would be toxic as well.⁴¹

Other investigations have examined the efficacy of hydrogen peroxide as an antiseptic in animal and human wound models. In a narrative review,³⁸ hydrogen peroxide demonstrated minimal effects in reducing the bacterial bioburden (quantity of microorganisms), but the findings were inconclusive regarding cytotoxic effects on tissues and rates of healing. Small experimental investigations^{41,42} support the finding that hydrogen peroxide is ineffective in reducing microorganisms in the wound bed. However, these trials demonstrated a greater cytotoxic effect of hydrogen peroxide on tissues,^{42,43} which perhaps delayed healing.

Temperature. Athletic trainers must also consider the temperature of the solution to facilitate healing. The recommended temperature of the cleansing solution is between 98.6°F and 107.6°F (37°C and 42°C).^{13,14,44,45} Mitotic activity (reproduction of cells essential to healing) decreases as the wound temperature drops after cleansing or dressing changes. It can take up to 40 minutes for the wound bed to return to its original temperature and up to 3 hours for mitotic activity to return to normal.⁴⁵ Therefore, using a cool cleansing solution may delay the healing process.¹⁴ Although research directly examining the effects

of various solution temperatures on wound healing is lacking, some evidence suggests that lower temperatures may delay wound healing.⁴⁵

Debridement

Debridement is the removal of necrotic or devitalized tissue, microorganisms, contaminated tissue, fibrin or foreign bodies, and cellular debris from the wound bed.^{53,54,75,185} After acute skin trauma, debridement should be used to decrease the risk of infection and create an environment suitable for healing. Thorough cleansing of the wound should be performed initially and, if necessary, debridement used to remove remaining debris or devitalized tissue from the wound bed. The wound should be debrided until only normal vascularized tissue remains.^{54,56} Debridement can decrease the bacterial concentration within the wound bed, decreasing the bacterial bioburden⁸³; improve the function of leukocytes, reducing the risk of infection; shorten the inflammatory phase, decreasing the energy required for healing; and remove debris and tissue from the wound bed, eliminating the physical barrier to healing.⁶⁶

Technique. Many debridement methods are available to health care providers. The methods that ATs can consider using include irrigation, hydrotherapy, wet-to-dry, wet-to-moist, scrubbing, conservative sharp, chemical, and autolytic debridement.

Irrigation. *Irrigation* is the delivery of normal saline or potable tap water to the wound bed in a constant or pulsed stream.^{3,57–59} Irrigation can serve as an extension of cleansing to remove loose superficial debris or necrotic tissue from superficial- to full-thickness abrasions, avulsions, blisters, incisions, lacerations, and punctures.^{3,57–59} Recommended pressure ranges from 4 to 15 psi (27.58–103.4 kPa) for all wounds except punctures, for which 2 to 4 psi (13.79–27.58 kPa) is recommended to avoid driving debris and contaminants deeper into the wound.^{3,57,59,61}

Hydrotherapy. Whirlpool baths and soaks use potable tap water to soften and remove devitalized tissue and toxic debris and dilute the bacterial content of the wound bed. The body part and wound are submerged in water between 95.9°F and 102.2°F (35.5°C and 39.0°C) for 20 to 30 minutes.⁶³ Although commonly used, hydrotherapy is not recommended for acute skin trauma. This technique can increase the risk of cross-contamination from the whirlpool tub or other container, and it is neither cost-effective nor time-effective to drain, properly clean and disinfect, and refill the whirlpool tub or container after each patient.⁶³

Wet to Dry. *Wet-to-dry debridement* is the use of woven gauze with large pores that is premoistened with normal saline, potable tap water, or chemicals and placed directly on the wound bed.⁵⁵ The gauze is allowed to remain on the wound bed undisturbed for 8 to 24 hours until dry and then quickly removed.^{66,68} Devitalized tissue adheres to the gauze as drying occurs and is debrided from the wound with removal of the gauze. Wet-to-dry debridement is not recommended for acute skin trauma. Wet-to-dry debridement is nonselective in tissue removal; as a result, healthy granulation tissue may also adhere to the gauze and be removed, adversely affecting the healing process^{57,59,64,67,69} and causing additional pain.^{65,70}

Wet to Moist. Similar to wet-to-dry debridement, *wet-to-moist debridement* is the placement of woven gauze with large pores that is premoistened with normal saline or potable tap water over the wound bed. The gauze is allowed to remain on the wound bed for minutes to hours and removed before drying is complete. Wet-to-moist debridement may be used for superficial- to full-thickness abrasions, avulsions, blisters, incisions, and lacerations. This technique is a rapid method of debridement and allows for removal of debris, devitalized tissue, and eschar (black necrotic tissue, scab), while protecting healthy granulation tissue and producing minimal pain.⁵⁹

Scrubbing. *Scrubbing* is the use of a sponge with high porosity (90 pores/in² [14 pores/cm²]) or a surgical scrub brush, along with normal saline or potable tap water, to scour the wound bed from the middle toward the wound margins in a circular pattern.⁶⁶ Scrubbing can be used with superficial- to partial-thickness abrasions, avulsions, blisters, incisions, and lacerations contaminated with large quantities of small debris (eg, sand, grass, clay, asphalt).⁶⁶ This technique is nonselective, with potential removal of healthy granulation tissue, and the mechanical pressure of the sponge or brush can produce pain.

Conservative Sharp Debridement. *Conservative sharp debridement* is the use of sterile scissors and forceps or tweezers to remove loosely adhering devitalized tissue that lies superficial to viable tissue on the wound bed.⁷¹ Conservative sharp debridement of acute skin trauma is typically performed in a single visit and may be used on superficial- to full-thickness abrasions, avulsions, blisters, incisions, and lacerations.⁷² Applicable state practice acts may allow ATs to perform this technique but should be verified.¹⁷⁴

Chemical Debridement. *Chemical debridement* is the application of sodium hypochlorite, hydrogen peroxide, or povidone-iodine in water-based solutions; silver; or honey directly to the wound bed or the use of chemical-impregnated dressings over the wound. Although chemicals may facilitate debridement, their use is controversial and not recommended for acute skin trauma.^{71,73,74} Some authors have recommended chemicals for debridement of infected wounds⁷¹ or with wet-to-moist debridement.⁷⁵ The effervescent effect of hydrogen peroxide may aid debridement, but hydrogen peroxide and sodium hypochlorite are thought to be cytotoxic to healthy granulation tissue.⁷⁴ Silver and silver-impregnated dressings have been used for centuries to provide antimicrobial debridement of colonized and clinically infected wounds. In an evidence-based medicine review, Vermeulen et al¹²¹ demonstrated insufficient evidence for the use of silver-impregnated dressings to manage contaminated or infected acute and chronic wounds.

Autolytic Debridement. *Autolytic debridement* is the use of the body's mechanisms to promote proteolytic digestion of necrotic tissue in a moist environment created by the application of occlusive dressings such as alginates, films, foams, hydrogels, and hydrocolloids.^{76,79,83,85} The moist environment allows endogenous collagenase enzymes within the wound to liquefy necrotic tissue, which can then be more easily digested by macrophages.^{51,55,57,58,75,77,80,82} The body selectively digests only nonviable tissue and the moist environment allows for painless debridement.⁷¹ Autolytic debridement can be used

with superficial- to full-thickness abrasions, avulsions, incisions, and lacerations; superficial- to full-thickness blisters after removal of the necrotic roof with conservative sharp debridement⁷¹; and superficial- to partial-thickness punctures. Autolytic debridement is slow, occurring over several days, and is not recommended for infected wounds.^{57,66,75}

In an evidence-based medicine review, Lewis et al⁷⁹ examined the use of dressings in the autolytic debridement of postoperative incisions. Overall, modern occlusive dressings were favored over gauze dressings for healing. Findings from individual trials in the review⁷⁹ produced no clear evidence of differences in rates of healing among polyurethane foam, silicone foam, and alginate dressings. Several authors^{78,81,84} have investigated the effectiveness of autolytic, wet-to-dry, and enzymatic debridement among various chronic wounds. Autolytic debridement using hydrogel and hydrocolloid dressings and wet-to-dry debridement using normal saline-soaked gauze produced a satisfactory environment to promote healing of pressure ulcers.⁸¹ In this study, the hydrogel dressing was preferred because the transparent construction allowed for visual assessment of the wound bed and removal of the dressing did not damage healthy tissue. Other authors⁸⁴ have shown hydrogel dressings to be effective in managing chronic and necrotic wounds with slough. For debridement of leg ulcers, no differences were demonstrated between autolytic and enzymatic debridement in rates of healing.⁷⁸

Dressings

It is well established that acute wounds should be covered with a dressing to support the healing process rather than be left uncovered and exposed to the external environment.^{2,86-90} Dressings used with acute skin trauma must promote an environment that will facilitate complete healing in the shortest possible amount of time.^{100,112} A variety of nonocclusive and occlusive dressings are available, but purchasing a wide selection is likely not cost-effective for athletic training facilities. Athletic trainers may choose to select and become proficient in using a few brands of dressings from each category.

Nonocclusive Dressings. Nonocclusive dressings are readily accessible and used in most athletic training facilities for the management of acute skin trauma.^{181,186} These dressings are available in a variety of forms and include woven, nonwoven, and impregnated sterile gauze, nonadherent pads, adhesive strips and patches, and wound-closure strips.

Primary Dressings. Primary dressings are designed to make contact with the wound bed.¹⁸⁷ Nonocclusive dressings can be used as primary dressings for several wound types. Woven and nonwoven gauze can be used for clinically infected abrasions, avulsions, blisters, incisions, lacerations, and punctures.^{91,92} These dressings can be applied alone or in combination with topical antimicrobial agents^{91,92} and may be cost-effective when compared with other dressings.⁹¹ Woven, nonwoven, and impregnated gauze strips and rolls can be used for puncture wounds with cavities.⁹³⁻⁹⁶ The dressings eliminate dead space, prevent premature closure of the wound surface, and allow healing to occur from the wound base upward.^{93-96,188} Wound-closure strips can be used for superficial, linear lacerations

and postoperative incisions with minimal static and dynamic tension.^{3,97,98} Woven gauze with an open-weave pattern can be used for mechanical (wet-to-moist) debridement of superficial- to full-thickness abrasions, avulsions, blisters, incisions, and lacerations.^{55,68,91}

Woven and nonwoven gauze, nonadherent pads, and adhesive strips or patches premoistened with normal saline^{95,99} or potable tap water can be used as temporary primary dressings for superficial- to partial-thickness abrasions, avulsions, and blisters and superficial-thickness incisions, lacerations, and punctures.⁹¹ This dressing technique is appropriate when an immediate return to physical activity is necessary. Further evaluation and appropriate cleansing, debridement, and dressing should be conducted in the athletic training facility. Woven and nonwoven gauze, nonadherent pads, and adhesive strips or patches can also be applied to wounds sustained on irregular body surfaces when other dressings cannot be held in place.

Secondary Dressings. Secondary dressings are designed to be used in combination with primary dressings to provide additional absorption, protection, or occlusion for the wound bed.¹⁸⁷ Woven or nonwoven gauze, nonadherent pads, and adhesive strips or patches can be used as secondary dressings for superficial- to full-thickness abrasions, avulsions, blisters, lacerations, punctures, and traumatic and postoperative incisions.^{91,94,100,101} Woven and nonwoven gauze can be applied over the primary dressing to absorb moderate-to-heavy amounts of exudate, which can leak from or strike through the primary dressing.¹⁰⁰ *Strike-through* is the leakage of exudate from the wound bed that becomes visible through the dressing. Gauze and nonadherent pads can provide additional padding and protection to the wound. Roll gauze and adhesive strips or patches can also assist in securing the primary dressing to the periwound tissues.

Occlusive Dressings. Semipermeable and impermeable occlusive dressings are designed to interact with the wound to facilitate healing and lessen the risk of infection and adverse reactions. Among the numerous classes of occlusive dressings, alginates, films, foams, hydrogels, hydrocolloids, and dermal adhesives are the most accessible to ATs for managing acute skin trauma.

Primary Dressings. For several wound types, occlusive dressings should be used as primary dressings and applied directly to the wound bed. Alginate, film, foam, hydrocolloid, and hydrogel dressings should be used for superficial- to partial-thickness abrasions, avulsions, blisters, incisions, lacerations, and punctures.^{2,90,91,95,102–112} Films and hydrogels are indicated for superficial-thickness wounds based on their ability to manage low levels of exudate. Films are nonabsorbent; with higher levels of exudate, channels (progression of exudate from the wound bed to the perimeter of the dressing) can form in the dressing and compromise the seal edge and barrier properties with subsequent leakage.⁹¹ Hydrogels can absorb minimal-to-moderate amounts of exudate and are unique in that they can also donate moisture to the wound bed,¹⁰⁷ thereby lowering the risk of desiccation in minimally draining superficial wounds.¹⁰⁸ Partial-thickness wounds with moderate exudate should be managed with hydrocolloids or hydrogels. Hydrogels should be closely monitored because maceration of the wound and periwound tissues can occur

from the high water content and slow absorption of exudate.^{103,106} Alginate and foam dressings are indicated for partial-thickness wounds with heavy exudate. High absorbency allows alginates and moderate-to-high moisture vapor transmission (evaporation of fluid from the wound bed through the dressing) and high absorbency enables foam dressings to manage large amounts of exudate.¹⁰⁴

Partial- to full-thickness abrasions, avulsions, and blisters should be managed with alginates, foams, and hydrocolloids; lacerations and incisions can be managed with alginates, foams, hydrocolloids, or dermal adhesives.[‡] Hydrocolloids should be applied to partial- to full-thickness abrasions, avulsions, and blisters accompanied by moderate exudate.^{109,115} Hydrocolloids can also be used for lacerations and traumatic and postoperative incisions with moderate exudate and adequate tissue approximation.^{113,114,116} Alginates and foams should be used to manage the heavy exudate typically produced by partial- to full-thickness abrasions, avulsions, blisters, lacerations, and traumatic and postoperative incisions with adequate tissue approximation.^{91,109,117} Lacerations and traumatic and postoperative incisions in areas of low skin tension that require tissue approximation can be closed with dermal adhesives.^{97,98,118,119}

Several classes of occlusive dressings can be used for contaminated and clinically infected wounds. Alginates and foams can be applied to clinically infected abrasions, avulsions, blisters, incisions, and lacerations.^{91,94,120,121} The high absorbency of these dressings can effectively manage the significant amounts of drainage associated with infection, but daily changes are required.^{91,94,120} Antimicrobial silver dressings can be used for contaminated and clinically infected abrasions, avulsions, blisters, incisions, and lacerations.^{120,121} Alginate, film, foam, and hydrocolloid dressings are available with different concentrations and release rates of silver.

Secondary Dressings. Occlusive dressings can be used as secondary dressings in combination with primary dressings on superficial- to full-thickness abrasions, avulsions, blisters, lacerations, punctures, and traumatic and postoperative incisions. Although most occlusive dressings are available in sheet form with an adhesive backing, some foams and hydrogels are nonadhesive and require a secondary dressing.^{91,105,108,109} Films are appropriate as secondary dressings to secure foams and hydrogels to provide occlusion¹²² and can also be applied in combination with other occlusive dressings in sheet form for additional adherence to the periwound tissues and to prevent leakage of excess exudate from heavily draining wounds. Films and hydrocolloids can serve as secondary dressings for lacerations and traumatic and postoperative incisions that have been closed with sutures,^{3,101,113,114,116,123} staples,¹⁰¹ or dermal adhesives.^{3,101}

Healing. Authors of evidence-based medicine reviews have focused on nonocclusive and occlusive dressing interventions for split-thickness skin graft (STSG) donor sites and traumatic and postoperative wounds in various populations. Among the broad categories of nonocclusive and occlusive dressings, strong evidence indicated that occlusive dressings were favored over nonocclusive

‡References 2, 90, 91, 97, 98, 102, 106, 109–119.

dressings for rates of healing in STSGs.¹⁰² This surgical wound is the equivalent of a superficial- to partial-thickness abrasion.¹⁷⁵ In the review,¹⁰² hydrocolloids and films were favored over nonocclusive dressings and hydrocolloids were also superior to other occlusive dressings for healing. Individual trials in the review¹⁰² revealed that alginates and foams were superior to nonocclusive dressings for healing. An additional review¹¹¹ showed no differences among nonocclusive and occlusive dressings in rates of healing among STSGs and postoperative incisions. An individual trial in this review¹¹¹ demonstrated that foams were superior to nonocclusive dressings in healing of STSGs. Evidence-based reviews examining the effects of nonocclusive and occlusive dressings impregnated with silver on uninfected¹⁸⁹ and contaminated and infected^{121,190} acute and chronic wounds provide no clear evidence to support their effectiveness in increasing healing rates.

Authors of other evidence-based medicine reviews have reported on the efficacy of dermal adhesives for traumatic and postoperative wounds. A Cochrane review⁹⁸ examined standard wound closure (sutures, staples, wound-closure strips) and dermal adhesives for postoperative incisions. The findings demonstrated that sutures lessened *dehiscence* (separation of wound edges); wound-closure strips improved surgeons' assessments of cosmetic appearance; and sutures, staples, and wound-closure strips increased surgeons' satisfaction regarding ease of use. For time to complete closure, the review revealed inconsistent findings between standard wound-closure techniques (sutures, staples, wound-closure strips) and dermal adhesives. Among dermal adhesives, low-viscosity adhesive was favored over high-viscosity products, and octyl cyanoacrylate was favored over butyl cyanoacrylate for time to complete closure. An additional Cochrane review⁹⁷ reported on dermal adhesives and standard wound closure (sutures, staples, wound-closure strips) for traumatic lacerations. Dermal adhesives lessened pain, the time to complete closure, and the rate of erythema but increased the risk of dehiscence. No differences were found in cosmetic outcomes. Standard wound-closure techniques (sutures, staples, wound-closure strips) were favored as easier to use. A comparison of the dermal adhesives butyl cyanoacrylate and octyl cyanoacrylate demonstrated no differences in levels of pain, time to complete closure, dehiscence, or cosmetic outcome.

Experimental and clinical investigations present additional evidence for the benefits of occlusive dressings. In several small trials examining rates of healing in standardized abrasions, authors found that hydrocolloids and films were superior to nonocclusive dressings,⁹⁰ other occlusive dressings,^{2,112} and no dressing.^{2,90} Hydrocolloids used as secondary dressings for sutured traumatic lacerations and postoperative incisions showed increased patient mobility^{191,192} and greater exudate control¹⁹² when compared with nonocclusive dressings. For rates of healing, findings were inconsistent when hydrocolloids as secondary dressings for sutured and nonsutured lacerations and incisions were compared with nonocclusive dressings.^{113,193,194}

Pain. Increased levels of pain are associated with the use of nonocclusive dressings. In an evidence-based medicine review, Wiechula¹⁰² noted that nonocclusive dressings used as primary dressings for STSGs produced greater levels of pain on visual analogue scales at rest and with ambulation

than occlusive dressings. Findings from individual trials in the review¹⁰² demonstrated that hydrocolloids, films, and foams resulted in less pain than nonocclusive dressings. Several authors^{191,194,195} have shown that hydrocolloids used as secondary dressings decreased levels of pain in sutured lacerations and postoperative incisions compared with nonocclusive dressings.

Infection. Rates of infection with various nonocclusive and occlusive dressings have been reported in the literature. In a review¹¹⁰ of 75 studies (3047 wounds) examining infection under occlusive dressings and 36 studies (1085 wounds) examining infection under nonocclusive dressings used as controls for occlusive dressings, researchers demonstrated overall infection rates of 2.6% and 7.1%, respectively. This significant finding in overall rates was supported by further analyses of infection for dressings by wound type (eg, ulcers, burns, STSGs, abrasions, and lacerations), with occlusive dressings favored over nonocclusive dressings.¹¹⁰ In an evidence-based medicine review,¹⁰² occlusive dressings produced less infection than nonocclusive dressings in the management of STSGs. Among specific dressings, hydrocolloids and films were favored over nonocclusive dressings for decreased rates of infection. In a Cochrane review,⁹⁷ rates of infection did not differ between the dermal adhesives butyl cyanoacrylate and octyl cyanoacrylate in the closure of traumatic lacerations. In contrast, a separate Cochrane review⁹⁸ revealed no differences in rates of infection between nonocclusive standard wound closure (sutures, staples, wound-closure strips) and occlusive dermal adhesives in closure of postoperative incisions. Authors of several evidence-based medicine reviews examined the effects of nonocclusive and occlusive dressings impregnated with silver on uninfected¹⁸⁹ and contaminated and infected^{121,190} acute and chronic wounds. No clear evidence supported their effectiveness to prevent or control infection. An evidence-based review¹⁰¹ examined the efficacy of nonocclusive and occlusive dressings for postoperative incisions healing by primary intention (tissue approximation using sutures, staples, dermal adhesives, or a combination of these). The authors¹⁰¹ found no clear evidence for differences in rates of surgical-site infection among nonocclusive dressings, occlusive dressings, and no dressings. Additionally, there was no clear evidence for the most effective dressing to lessen rates of infection.

Identification of Infection and Adverse Reactions

The goal in treating acute skin trauma is to achieve rapid healing while providing optimal function and cosmetic results and minimizing adverse events. Adverse outcomes may occur when a phase of healing is delayed or prolonged; may result from the cleansing, debridement, or dressing technique or material used; or be due to the health status of the patient. Most acute skin trauma heals without consequence, although it is important to manage these wounds appropriately to lessen the number of poor outcomes and allow functional return to activity as quickly as possible.

Infection. The most common cause of impaired wound healing is infection.¹³⁰ In acute skin trauma, the barrier to bacteria is compromised by disruption of the epidermis, dermis, or subcutaneous tissues (or all 3) and loss of

protective defense mechanisms. Wounds can then become contaminated with bacteria. Although bacteria are present, multiplication of bacteria has not yet taken place.¹²⁶ Colonization is a normal state, with multiplying microorganisms present in the wound but no host reaction and no clinical indication or evidence of tissue damage.¹⁹⁶ Bacterial contamination of the wound will not delay the healing process, and colonization of skin microflora may actually enhance healing.³⁹ Healing becomes impaired when colonization progresses to critical colonization and then to infection.³⁹ *Critical colonization* is the transition state between colonization and invasive wound infection.¹⁹⁶ Critical colonization is reached when the host defenses are unable to maintain the balance of organisms at colonization.¹²⁶ This transition period is a multifactorial process and is specific to the individual patient and the particular bacteria in the wound.¹²⁶ At critical colonization, the granulation bed of the wound may appear unhealthy, but there is no tissue invasion and the only clinical feature may be delayed healing.¹⁹⁶

Infection occurs when the presence of multiplying bacteria overwhelms the host defenses and subsequent host injury occurs.¹²⁶ The most common causes of uncomplicated skin and soft tissue infections are group A β -hemolytic *Streptococcus* and *Staphylococcus aureus* bacteria.^{197–202} Data from the SENTRY Antimicrobial Surveillance Program monitoring skin and soft tissue infections indicated that *S aureus* was the most common pathogen among complicated and hospitalized patients, followed by *Pseudomonas aeruginosa*, *Enterococcus*, *Escherichia coli*, *Enterobacter*, *Klebsiella*, and *Streptococcus*.²⁰³ Streptococcal infections may be underrepresented because many cases, in contrast with other organisms, are mild and do not require patients to be hospitalized.²⁰⁴ Additionally, the incidence of streptococcal infections is difficult to assess given the lack of specimens and reliance on studies using unconventional identification methods.¹⁹⁷ The progression to infection is multifactorial and depends on the number of bacteria, their virulence and pathogenicity, and the host's ability to mount an immune response.¹⁹⁶ Intact skin often contains microflora at 10^5 organisms per gram of tissue.¹⁹⁶ Infection can occur when the level of bacterial growth exceeds 10^5 organisms per gram of tissue, although it can also occur at lower levels for more virulent bacteria such as β -hemolytic *Streptococcus*.¹²⁵ Depending on the nature of the wound and the organism involved, more than 50% of patients with wounds containing $>10^5$ organisms per gram of tissue will develop an infection.²⁰⁵ Factors affecting wound infection include history, location, and timing, which can predict the level of bacteria in a wound.¹³⁰ The longer the time from injury until treatment, the greater the bacterial bioburden of the wound. The mean time for those with $>10^5$ organisms per gram of tissue is 5.17 hours from the time of injury.¹³⁰ When an infection does occur, wound healing is impaired through multiple mechanisms. Infection decreases oxygen tension; prolongs the inflammatory phase; impairs leukocyte chemotaxis and migration; phagocytosis, intracellular killing, angiogenesis, and epithelialization; and produces collagen breakdown.¹²⁴

Clinical features of infection are due to the excessive inflammatory response surrounding the wound. Cellulitis occurs when the infection spreads through the dermis and subcutaneous tissue.¹³⁴ Signs and symptoms may include

fever, pain, edema, erythema, warmth, wound dehiscence, and delayed wound healing.^{124–128} Cellulitis borders are smooth and ill defined. Patients with more severe infections may present with vesicles, bullae, pustules, necrosis, ascending lymphangitis, and regional lymphadenopathy.¹²⁷

Infection should be recognized and addressed promptly to prevent progression. Antibiotic treatment should be directed toward the most common pathogens or suspected cause of infection.^{197–202} Systemic antibiotics rather than topical antibiotics are appropriate for cellulitis. For nonpurulent cellulitis, empiric treatment is aimed at β -hemolytic *Streptococcus* and methicillin-susceptible *S aureus* and increasing coverage for community-associated methicillin-resistant *S aureus* (CA-MRSA) for those who worsen in 48 hours or develop an abscess.²⁰⁴ If a simple abscess is present or the wound is draining purulent material, the patient should be referred to a physician for possible incision and drainage. If the wound is drained and the infection does not appear to be spreading through the subcutaneous tissue, then systemic antibiotics are not needed.¹³⁰ For those with a purulent cellulitis, empiric antibiotic therapy is given for CA-MRSA.²⁰⁴ More information on the treatment of bacterial skin infections can be found in the National Athletic Trainers' Association position statement on skin diseases.¹²⁹

Prevention of infection is paramount to optimizing healing of acute skin trauma. Wound cleansing, debridement, dressing techniques, and antimicrobials may all play roles in preventing infection.^{39,125,130–132} Skin antiseptics and systemic and topical antibiotics also play roles in preventing infection. Antiseptics such as iodine and chlorhexidine decrease bacterial counts and the number of bacteria colonizing the skin. An evidence-based medicine review¹³³ demonstrated that skin antisepsis was effective in reducing the risk of surgical-site infection. Therefore, it may be beneficial to cleanse periwound tissues with an antiseptic before primary closure of traumatic incisions and lacerations with wound-closure strips or dermal adhesives.

Systemic and topical antibiotics should be used appropriately by a physician because of the potential for side effects and development of resistance. Prophylactic systemic antibiotics are rarely needed, because acute wounds that are not grossly contaminated and have undergone appropriate cleansing and debridement have very low overall infection rates.¹³⁵ Authors¹³⁴ of an evidence-based medicine review evaluating prophylactic antibiotics for simple, nonbite wounds in the emergency room found they offered no benefit for lessening rates of infection. Overall, prophylactic antibiotics resulted in a greater incidence of infection compared with controls.¹³⁴ However, the use of prophylactic systemic antibiotics should be considered when associated risk factors for infection are present. Soft tissue injuries associated with a fracture, an animal or human bite, or an intraoral laceration are treated with antibiotics for 3 to 5 days, with the medication and length of treatment dependent on the injury type.¹³⁸ Prophylactic antibiotics should also be considered when factors associated with a higher risk of infection are present. These include mechanism of injury, amount of tissue damage, presence of contaminants, location of wound, comorbid medical conditions, and age of the wound. Wounds that are visibly contaminated or deep, expose tendon or bone, are jagged or stellate, or contain foreign bodies are considered

at higher risk of infection.^{138,206} Location of the wound predicts infection: infection rates are higher in the thigh and arm and lower in the face and scalp.²⁰⁷ Patient comorbidities, including extremes of age, diabetes, chronic renal failure, obesity, malnutrition, and immunocompromise due to illness or medications, can increase the risk of infection.¹³⁸ Wounds that receive delayed treatment are at higher risk, and a delay greater than 10 hours becomes an important risk factor for infection.²⁰⁷ Questions regarding the need for systemic prophylactic antibiotics require the patient to be referred to a physician. Topical antimicrobial agents may effectively reduce rates of infection with acute skin trauma. Authors¹⁴² of a small review of clinical investigations examining the efficacy of topical antimicrobials revealed a decrease in infection rates among superficial- and partial-thickness abrasions, lacerations, punctures, and sutured lacerations compared with topical preparations without an antibiotic. Findings from individual trials in this review¹⁴² showed no differences in rates of infection among triple antibiotic (neomycin, bacitracin, polymyxin B), mupirocin, bacitracin zinc ointment, and povidone-iodine cream. However, the period of use should be limited to help prevent emergence of resistant bacterial strains, hypersensitivity reactions, and adverse effects on wound healing.^{135–141}

Adverse Reactions. Adverse reactions may occur with materials used to treat acute skin trauma. Allergic contact dermatitis can be caused by cleansing solutions and dressings but is more common with topical antimicrobial agents and neomycin in particular.^{145,162} Clinical features vary and may include pruritus, eczematous plaque, edema and erythema with vesicles, or more generalized dermatitis.¹⁶² Treatment involves removal of the offending substance and application of topical corticosteroids.¹⁶² Patients with more severe cases may require systemic corticosteroids and oral antihistamines.¹⁶² Less frequently, immunoglobulin E-mediated allergic reactions and anaphylaxis may occur with topical antimicrobial use. Clinical features occur shortly after administration and can include pruritus, urticaria, dyspnea, throat swelling, nausea, diarrhea, dizziness, and death.^{143,144} Treatment of anaphylaxis is emergent and includes epinephrine, corticosteroids, and antihistamines.¹⁴³ Chemical burns have occurred from prolonged contact with povidone-iodine in whirlpool baths and soaks or under an occlusive device or dressing.¹⁶³ Povidone-iodine has also been associated with metabolic acidosis, cardiovascular instability, renal insufficiency, and death when used indiscriminately for large wounds.^{40,163} Patients with chemical burns may present with pain, well-demarcated erythema, bullae, and vesicles. Treatment is supportive with topical antibiotics and dressings that promote healing.¹⁶³ Foreign-body reactions can develop from the use of dermal adhesives.¹⁴⁶ Clinical features are wound-site tenderness followed by the appearance of a tumor or mass.¹⁴⁶ The adhesive should be removed and the wound monitored.¹⁴⁶

Nonocclusive and occlusive wound dressings have been implicated in immediate and delayed reactions due to occlusion or excessive adhesion of the dressing to the wound.¹⁴⁷ Folliculitis, caused by occlusion of the skin, occurs at the base of hair follicles over the periwound tissues.²⁰⁸ Clinical features may include multiple papules and pustules in areas covered by an occlusive dressing,

commonly areas that were shaved before dressing application. Treatment consists of dressing removal and referral for possible culture and systemic antibiotic therapy.²⁰⁸ Maceration, which is often associated with chronic wounds, may also occur from acute skin trauma and contribute to delayed healing.¹⁶⁴ It is a function of wound exudate and can extend the wound and contribute to pain.¹⁶⁴ Maceration can be identified clinically by white discoloration of the periwound tissues. Erythematous maceration can occur, causing the skin to appear red and inflamed, and be associated with burning, stinging, or itching.¹⁶⁵ Wounds that are developing maceration require dressing removal and reassessment to determine if dressing selection, prolonged dressing wear time, or associated infection is contributing to the production of exudate.^{164,165} Desiccation of nonocclusive dressings and adhesion of occlusive dressings to the wound bed require remoistening before removal to prevent tissue avulsion.^{57,59,64,67,69,91}

Adverse outcomes may occur because of the status or health of the patient. Although the data are inconclusive, malnutrition may contribute to a delay in healing and the development of infection.¹⁵⁴ Anti-inflammatory medications such as topical or systemic steroids,^{155,156} nonsteroidal anti-inflammatories,^{157,158} and COX-2 inhibitors¹⁵⁹ may suppress wound healing. Smoking may also impair healing for multifactorial reasons, including a decrease in collagen production. Diabetes may delay tissue repair and wound contraction, reduce incision-breaking strength, and increase susceptibility to infection.¹⁶⁰ Wound healing may also be delayed in those with chronic renal insufficiency, acute or chronic liver disease, peripheral vascular disease, or AIDS.^{130,148–153,161}

Criteria for Referral. Most patients with acute skin trauma can be treated by ATs without complication. Indications for physician referral include deep wounds that require tissue approximation with sutures or staples; heavily contaminated wounds that require more extensive cleansing, debridement, or possibly prophylactic antibiotics; and wounds with tendon or nerve injury. Consideration for referral of an acute uncomplicated wound should be given for patients with immunocompromised conditions. Patients with wounds that can be managed in the athletic training facility should be treated and followed closely. A delay in normal healing, development of an allergic reaction, or clinical features of infection or adverse reactions including erythema, warmth, edema, pus, or pain inconsistent with findings require physician referral.

Follow-Up

Appropriate management of acute skin trauma requires the AT to monitor the patient, the wound area, and the dressing until healing is complete. Daily visual inspections of the patient, wound bed, and periwound tissues are performed to identify signs and symptoms associated with the development of adverse reactions and infection. Some dressings (eg, wound-closure strips, films, hydrogels, dermal adhesives) can remain in place while the AT partially visualizes the wound bed.

Daily visual inspections of the dressing are also used to guide the frequency of dressing changes. Changes are based on the type of dressing being used, dressing integrity, barrier and absorbency properties, and healing of the

wound. Nonocclusive and occlusive dressings are designed to remain on the wound bed for varying periods of time in the absence of dressing-integrity problems, adverse reactions, or infection (Table 5). However, these recommendations are only guidelines, and without adverse reactions or infection, alginates, films, foams, hydrogels, and hydrocolloids may remain on the wound bed longer than 7 days.²⁰⁹

The integrity and physical properties of a dressing will influence performance and healing outcomes. Wrinkling or bunching of the dressing and the formation of channels from the wound bed to the edge, separation of the dressing edges from the periwound tissues, or excessive accumulation of exudate leading to strike-through can result in leakage of exudate.^{89,91,94} Leakage of exudate and failure of the dressing to maintain a barrier to the external environment can increase the risk of cross-contamination and infection and may lead to wound desiccation.^{89,166,167}

Dressing transitions are also necessary as the wound progresses through the phases of healing.⁸ No one dressing is appropriate for every wound,^{210,211} and few dressings are suited for treating a single wound throughout the healing process.¹⁰⁰ Dressing transitions may be required as healing progresses, based on cellular and chemical changes in the wound bed, such as decreasing levels of exudate and new epithelial growth.¹⁰⁰ Dressings that become saturated with exudate in the early phases of healing may require additional changes to lessen the risk of leakage and maceration.^{91,168} Reductions in exudate levels as healing progresses indicate the need for a change to a dressing that is less absorbent or has a lower moisture vapor transmission rate to maintain sufficient moisture over the wound bed.^{91,94,104} Dry wounds with minimal exudate may require a moisture-donating dressing to prevent desiccation of the wound bed.¹⁰⁸

Several suggestions are provided to assist with clinical application of wound dressings. Nonocclusive and occlusive dressings are available in a variety of sizes to fit the diameter of the wound, and most can be cut to obtain a proper fit. Generally, the dressing should be 1 to 2 cm larger than the wound bed to provide an adequate edge seal on the periwound tissues.^{91,105,122,170} Most nonocclusive dressings require a secondary dressing, whereas the majority of occlusive dressings do not. Nonocclusive dressings can be secured with adhesive gauze (eg, Cover-Roll; BSN Medical Inc, Charlotte, NC; or Omnifix; Hartmann USA, Rock Hill, SC) or nonadherent, self-adherent, or adherent tapes and wraps. Adhesive gauze applied to the edges of films, foams, and hydrogels and over the entire surface of alginates and hydrocolloids effectively secures these dressings to patients for practices and competitions.^{2,112} Dressing removal should be approached with caution to avoid trauma to the wound bed. Maintaining moisture over the wound bed prevents occlusive dressings from adhering and protects against damage to the tissue upon removal.¹⁰⁵ Dressings that are adhered to the wound bed should be moistened with normal saline or tap water through irrigation before removal.⁹¹ With minimal exudate over the wound, some occlusive dressings (eg, films) can adhere to the wound bed and require moistening for removal.¹⁰⁵

Education for patients on wound dressings and the recommended guidelines for wear is needed to ensure

compliance. Patients must be encouraged to follow the directions provided by the athletic training staff for daily monitoring and identifying the signs and symptoms of infection and adverse reactions. Guidelines specific to occlusive-dressing wear and length of time over the wound bed should prevent unnecessary dressing changes. As a moist environment is created, the collection of exudate will be visible under transparent film and hydrogel dressings. This brownish fluid should not be confused with infection.¹⁰³ Alginates and hydrocolloids react with and absorb exudate over the wound bed, producing a gel-like mass.^{91,105,173} This gel can have a foul odor and should not be mistaken for infection.^{91,105} Absorption of exudate can cause swelling and expansion of occlusive dressings over the wound bed. Direct forces from contact with equipment, playing surfaces, and other individuals can rupture the dressings or force exudate to the dressing edges, resulting in the formation of channels and subsequent leakage.

Supplies for Athletic Training Facilities and Kits

Athletic training facilities and kits should have supplies for the management of acute skin trauma. Minimal supplies are required for kits used during practices and competitions. Timely cleansing, debridement, and dressing of wounds are often necessary to return patients to activity as soon as possible. Infection-control guidelines must be followed when managing patients with acute skin trauma. The Occupational Safety and Health Administration,²¹² Centers for Disease Control and Prevention,²¹³ National Athletic Trainers' Association,¹²⁹ and National Collegiate Athletic Association²¹⁴ guidelines provide infection-control measures to implement in the management of acute wounds. The measures include developing a written organizational control plan; educating staff members regarding control measures; promoting the cleansing and disinfecting of tables, supplies, and laundry; ensuring hand-hygiene and personal-hygiene practices among staff members and patients; adhering to sport association recommendations; and verifying documentation of recommended measures.

CONCLUSIONS

Certified ATs and other allied health and medical professionals must be able to manage patients with acute skin trauma to promote healing and lessen the risk of complications. This position statement presents recommendations to educate clinicians about cleansing, debridement, and dressing techniques; recognition, management, and prevention of infection and adverse reactions; and monitoring and educating the patient.

DISCLAIMER

The NATA and NATA Research & Education Foundation publish position statements as a service to promote the awareness of certain issues to their members. The information contained in the position statement is neither exhaustive nor exclusive to all circumstances or individuals. Variables such as institutional human resource guidelines, state or federal statutes, rules, or regulations, as well as regional environmental conditions, may impact the relevance and implementation of those recommenda-

§References 91, 94, 100, 104, 108, 168, 169.

tions. The NATA and NATA Foundation advise members and others to carefully and independently consider each of the recommendations (including the applicability of same to any particular circumstance or individual). The position statement should not be relied upon as an independent basis for care but rather as a resource available to NATA members or others. Moreover, no opinion is expressed herein regarding the quality of care that adheres to or differs from the NATA and NATA Foundation position statements. The NATA and NATA Foundation reserve the right to rescind or modify its position statements at any time.

This position statement is primarily intended to present acute wound care in the context of clinical practice in the United States. However, many sections are applicable to international clinical practice as well.

REFERENCES

1. Beam JW. Management of superficial to partial-thickness wounds. *J Athl Train*. 2007;42(3):422–424. (Level of evidence [LOE]: 3)
2. Beam JW. Occlusive dressings and the healing of standardized abrasions. *J Athl Train*. 2008;43(6):600–607. (LOE: 2)
3. Honsik KA, Romeo MW, Hawley CJ, Romeo SJ, Romeo JP. Sideline skin and wound care for acute injuries. *Curr Sports Med Rep*. 2007;6(3):147–154. (LOE: 3)
4. Brem H, Sheehan P, Rosenberg HJ, Schneider JS, Boulton AJ. Evidence-based protocol for diabetic foot ulcers. *Plast Reconstr Surg*. 2006;117(suppl 7):S193–S209. (LOE: 3)
5. Markenson D, Ferguson JD, Chameides L, et al. Part 13: first aid. 2010 American Heart Association and American Red Cross International consensus on first aid science with treatment recommendations. *Circulation*. 2010;122(16 suppl 2):S582–S605. (LOE: 1)
6. Standards for wound management, 2nd ed. Australian Wound Management Association Web site. http://www.awma.com.au/publications/2011_standards_for_wound_management_v2.pdf. Published March 2010. Accessed January 21, 2016. (LOE: 1)
7. Beam JW. Acute wound management: cleansing, debridement, and dressing. *Athl Ther Today*. 2008;13(1):2–6. (LOE: 3)
8. Ebell MH, Siwek J, Weiss BD, et al. Strength of recommendation taxonomy (SORT): a patient-centered approach to grading evidence in the medical literature. *Am Fam Physician*. 2004;69(3):548–556. (LOE: 3)
9. Towler J. Cleansing traumatic wounds with swabs, water or saline. *J Wound Care*. 2001;10(6):231–234. (LOE: 3)
10. Owens B, Wenke J. Early wound irrigation improves the ability to remove bacteria. *J Bone Joint Surg Am*. 2007;89(8):1723–1726. (LOE: 2)
11. Nicks BA, Ayello EA, Woo K, Nitzki-George D, Sibbald RG. Acute wound management: revisiting the approach to assessment, irrigation, and closure considerations. *Int J Emerg Med*. 2010;3(4):399–407. (LOE: 3)
12. Blunt J. Wound cleansing: ritualistic or research-based practice? *Nurs Stand*. 2001;16(1):33–36. (LOE: 3)
13. Fletcher J. Wound cleansing. *Prof Nurse*. 1997;12(11):793–796. (LOE: 3)
14. Davies C. Wound care. Cleansing rites and wrongs. *Nurs Times*. 1999;95(43):71–72, 75. (LOE: 3)
15. Morison M. Wound cleansing—which solution? *Prof Nurse*. 1989;4(5):220–225. (LOE: 3)
16. Magson-Roberts S. Is tap water a safe alternative to normal saline for wound cleansing? *J Community Nurs*. 2006;20(8):19–20, 22–24. (LOE: 3)
17. Carr M. Wound cleansing: sorely neglected? *Prim Intention*. 2006;14(4):150–152, 154, 156–157, 160–161. (LOE: 3)

18. Hampton S. The use of tap water for wound cleansing. *J Community Nurs*. 2004;18(12):16–20. (LOE: 3)
19. Fernandez R, Griffiths R, Ussia C. Effectiveness of solutions, techniques and pressure in wound cleansing. *JBI Libr Syst Rev*. 2004;2(7):371–423. (LOE: 1)
20. Fernandez R, Griffiths R. Water for wound cleansing. *Cochrane Database Syst Rev*. 2012;2:CD003861. (LOE: 1)
21. Bergstrom N, Bennett MA, Carlson CE, et al. Pressure ulcer treatment. *Clin Pract Guidel Quick Ref Guide Clin*. 1994;15:1–144. (LOE: 2)
22. Rodeheaver G, Pettry D, Thacker J, Edgerton M, Edlich R. Wound cleansing by high pressure irrigation. *Surg Gynecol Obstet*. 1975;141(3):357–362. (LOE: 3)
23. Bee T, Maniya S, Fany Z, et al. Wound bed preparation-cleansing techniques and solutions: a systematic review. *Singapore Nurs J*. 2009;36(1):16–20, 22. (LOE: 2)
24. Barr J. Principles of wound cleansing. *Ostomy Wound Manage*. 1995;41(suppl 7A):S15–S21. (LOE: 3)
25. Fernandez RS, Griffiths RD, Ussia C. Wound cleansing: which solution, what technique? *Prim Intention*. 2001;9(2):51–54, 56–58. (LOE: 1)
26. Trevelyan J. Wound cleansing. *Nurs Times*. 1996;92(50):44–46. (LOE: 3)
27. Young T. Common problems in wound care: wound cleansing. *Br J Nurs*. 1995;4(5):286–289. (LOE: 3)
28. Thomlinson D. To clean or not to clean? *Nurs Times*. 1987;83(9):71–75. (LOE: 3)
29. Cunliffe P, Fawcett T. Wound cleansing: the evidence for the techniques and solutions used. *Prof Nurse*. 2002;18(2):95–99. (LOE: 3)
30. Valente JH, Forti RJ, Freundlich LF, Zandieh SO, Crain EF. Wound irrigation in children: saline solution or tap water? *Ann Emerg Med*. 2003;41(5):609–616. (LOE: 2)
31. Moscati R, Mayrose J, Fincher L, Jehle D. Comparison of normal saline with tap water for wound irrigation. *Am J Emerg Med*. 1998;16(4):379–381. (LOE: 2)
32. Griffiths RD, Fernandez RS, Ussia CA. Is tap water a safe alternative to normal saline for wound irrigation in the community setting? *J Wound Care*. 2001;10(10):407–411. (LOE: 2)
33. Bansal B, Wiebe R, Perkins S, Abramo T. Tap water for irrigation of lacerations. *Am J Emerg Med*. 2002;20(5):469–472. (LOE: 2)
34. Lindholm C, Bergsten A, Berglund E. Chronic wounds and nursing care. *J Wound Care*. 1999;8(1):5–10. (LOE: 3)
35. Kramer S. Effect of povidone-iodine on wound healing: a review. *J Vasc Nurs*. 1999;17(1):17–23. (LOE: 2)
36. Khan M, Naqvi A. Antiseptics, iodine, povidone iodine and traumatic wound cleansing. *J Tissue Viability*. 2006;16(4):6–10. (LOE: 2)
37. Vermeulen H, Westerbos S, Ubbink D. Benefit and harm of iodine in wound care: a systematic review. *J Hosp Infect*. 2010;76(3):191–199. (LOE: 2)
38. Drosou A, Falabella A, Kirsner RS. Antiseptics on wounds: an area of controversy. *Wounds*. 2003;15(5):149–166. (LOE: 3)
39. Atiyeh BS, Dibo SA, Hayek SN. Wound cleansing, topical antiseptics and wound healing. *Int Wound J*. 2009;6(6):420–430. (LOE: 2)
40. Smith RG. A critical discussion of the use of antiseptics in acute traumatic wounds. *J Am Podiatr Med Assoc*. 2005;95(2):148–153. (LOE: 3)
41. Rabenberg VS, Ingersoll CD, Sandrey MA, Johnson MT. The bactericidal and cytotoxic effects of antimicrobial wound cleansers. *J Athl Train*. 2002;37(1):51–54. (LOE: 2)
42. Lineaweaver W, Howard R, Soucy D, et al. Topical antimicrobial toxicity. *Arch Surg*. 1985;120(3):267–270. (LOE: 3)
43. Wilson JR, Mills JG, Prather DO, Dimitrijevic SD. A toxicity index of skin and wound cleansers used on in vitro fibroblasts and

- keratinocytes. *Adv Skin Wound Care*. 2005;18(7):373–378. (LOE: 3)
44. Watret L, Armitage M. Making sense of wound cleansing. *J Community Nurs*. 2002;16(4):27–34. (LOE: 3)
 45. Lock PM. The effects of temperature on mitotic activity at the edge of experimental wounds. In: Sundel B, ed. *Symposium on Wound Healing: Plastic Surgical and Dermatological Aspects*. Moindal, Sweden: A. Lindgren & Soner; 1980. (LOE: 3)
 46. Courtenay M, Church JC, Ryan TJ. Larva therapy in wound management. *J R Soc Med*. 2000;93(2):72–74. (LOE: 2)
 47. Dowsett C, Claxton K. Reviewing the evidence for wound bed preparation. *J Wound Care*. 2006;15(10):439–442. (LOE: 2)
 48. Foster DT, Rowedder LJ, Reese SK. Management of sports-induced skin wounds. *J Athl Train*. 1995;30(2):135–140. (LOE: 3)
 49. Gottrup F. Debridement: another evidence problem in wound healing. *Wound Repair Regen*. 2009;17(3):294–295. (LOE: 3)
 50. Kumagai SG, Mahoney CR, Fitzgibbons TC, McMullen ST, Connolly TL, Henkel L. Treatment of diabetic (neuropathic) foot ulcers with two-stage debridement and closure. *Foot Ankle Int*. 1998;19(3):160–165. (LOE: 2)
 51. Rodeheaver GT. Pressure ulcer debridement and cleansing: a review of current literature. *Ostomy Wound Manage*. 1999;45(suppl 1A):S80–S85. (LOE: 2)
 52. Steed DL, Donohoe D, Webster MW, Lindsley L. Effect of extensive debridement and treatment on the healing of diabetic foot ulcers. *J Am Coll Surg*. 1996;183(1):61–64. (LOE: 2)
 53. Williams D, Enoch S, Miller D, Harris K, Price P, Harding KG. Effect of sharp debridement using curette on recalcitrant nonhealing venous leg ulcers: a concurrently controlled, prospective cohort study. *Wound Repair Regen*. 2005;13(2):131–137. (LOE: 2)
 54. Attinger CE, Bulan EJ. Debridement. The key initial first step in wound healing. *Foot Ankle Clin*. 2001;6(4):627–660. (LOE: 3)
 55. Bradley M, Cullum N, Sheldon T. The debridement of chronic wounds: a systematic review. *Health Technol Assess*. 1999;3(17, pt 1):1–78. (LOE: 2)
 56. Cornell RS, Meyr AJ, Steinberg JS, Attinger CE. Debridement of the noninfected wound. *J Am Podiatr Med Assoc*. 2010;100(5):353–359. (LOE: 3)
 57. Ayello EA, Cuddigan J, Kerstein MD. Skip the knife: debriding wounds without surgery. *Nursing*. 2002;32(9):58–63. (LOE: 3)
 58. Calianno C, Jakubek P. Wound bed preparation: laying the foundation for treating chronic wounds, part I. *Nursing*. 2006;36(2):70–71. (LOE: 3)
 59. Hess CT. When to use gauze dressings. *Adv Skin Wound Care*. 2000;13(6):266–268. (LOE: 3)
 60. Stewart JL, Carlson HC, Briggs RL, Green VA. The bacteria-removal efficiency of mechanical lavage and rubber-bulb syringe irrigation in contaminated avulsive wounds. *Oral Surg Oral Med Oral Pathol*. 1971;31(6):842–848. (LOE: 2)
 61. Green VA, Carlson HC, Briggs RL, Stewart JL. A comparison of the efficacy of pulsed mechanical lavage with that of rubber-bulb syringe irrigation in removal of debris from avulsive wounds. *Oral Surg Oral Med Oral Pathol*. 1971;32(1):158–164. (LOE: 2)
 62. Burke DT, Ho C, Saucier MA, Stewart G. Effects of hydrotherapy on pressure ulcer healing. *Am J Phys Med Rehabil*. 1998;77(5):394–398. (LOE: 2)
 63. Hess CL, Howard MA, Attinger CE. A review of mechanical adjuncts in wound healing: hydrotherapy, ultrasound, negative pressure therapy, hyperbaric oxygen, and electrostimulation. *Ann Plast Surg*. 2003;51(2):210–218. (LOE: 3)
 64. Edwards J, Stapley S. Debridement of diabetic foot ulcers. *Cochrane Database Syst Rev*. 2010;(1):CD003556. (LOE: 2)
 65. Kirshen C, Woo K, Ayello EA, Sibbald RG. Debridement: a vital component of wound bed preparation. *Adv Skin Wound Care*. 2006;19(9):506–517. (LOE: 3)
 66. Debridement. In: Myers BA, ed. *Wound Management: Principles and Practice*. 2nd ed. Upper Saddle River, NJ: Pearson Prentice Hall; 2008:70–93. (LOE: 3)
 67. Sibbald RG, Williamson D, Orsted HL, et al. Preparing the wound bed-debridement, bacterial balance, and moisture balance. *Ostomy Wound Manage*. 2000;46(11):14–22, 24–28, 30–35. (LOE: 3)
 68. Singhal A, Reis ED, Kerstein MD. Options for nonsurgical debridement of necrotic wounds. *Adv Skin Wound Care*. 2001;14(2):96–100. (LOE: 3)
 69. Steed DL. Treatment with growth factors and skin equivalents. *Contemp Surg*. 2000;suppl:17–23. (LOE: 3)
 70. Anderson I. Debridement methods in wound care. *Nurs Stand*. 2006;20(24):65–66, 68, 70. (LOE: 3)
 71. Gwynne B, Newton M. An overview of the common methods of wound debridement. *Br J Nurs*. 2006;15(19):S4–S10. (LOE: 3)
 72. Vowden KR, Vowden P. Wound debridement, part 2: sharp techniques. *J Wound Care*. 1999;8(6):291–294. (LOE: 3)
 73. Beam JW. Topical silver for infected wounds. *J Athl Train*. 2009;44(5):531–533. (LOE: 3)
 74. O’Toole EA, Goel M, Woodley DT. Hydrogen peroxide inhibits human keratinocyte migration. *Dermatol Surg*. 1996;22(6):525–529. (LOE: 2)
 75. Ayello EA, Cuddigan JE. Debridement: controlling the necrotic/cellular burden. *Adv Skin Wound Care*. 2004;17(2):66–75. (LOE: 3)
 76. Bale S, Banks V, Halestein S, Harding KG. A comparison of two amorphous hydrogels in the debridement of pressure sores. *J Wound Care*. 1998;7(2):65–68. (LOE: 2)
 77. Benbow M. Methods of wound debridement. *Nurs Times*. 1998;94(16):78–80, 83. (LOE: 3)
 78. König M, Vanscheidt W, Augustin M, Kapp H. Enzymatic versus autolytic debridement of chronic leg ulcers: a prospective randomised trial. *J Wound Care*. 2005;14(7):320–323. (LOE: 2)
 79. Lewis R, Whiting P, ter Riet G, O’Meara S, Glanville J. A rapid and systematic review of the clinical effectiveness and cost-effectiveness of debriding agents in treating surgical wounds healing by secondary intention. *Health Technol Assess*. 2001;5(14):1–131. (LOE: 2)
 80. McGuinness W, Rice J. The management of chronic wounds. *Aust Nurs J*. 2009;16(11):37–39. (LOE: 3)
 81. Mulder GD, Altman M, Seeley JE, Tintle T. Prospective randomized study of the efficacy of hydrogel, hydrocolloid, and saline solution-moistened dressings on the management of pressure ulcers. *Wound Repair Regen*. 1993;1(4):213–218. (LOE: 2)
 82. Ousey K, McIntosh C. Understanding wound bed preparation and wound debridement. *Br J Community Nurs*. 2010;15(3):S22, S24, S26. (LOE: 3)
 83. Panuncialman J, Falanga V. The science of wound bed preparation. *Clin Plast Surg*. 2007;34(4):621–632. (LOE: 3)
 84. Thomas S, Jones H. Clinical experiences with a new hydrogel dressing. *J Wound Care*. 1996;5(3):132–133. (LOE: 3)
 85. Trudigan J. Investigating the use of Aquafilm Hydrogel in wound management. *Br J Nurs*. 2000;9(14):943–948. (LOE: 3)
 86. Winter GD. Formation of the scab and the rate of epithelialization of superficial wounds in the skin of the young domestic pig. *Nature*. 1962;193:293–294. (LOE: 2)
 87. Hinman CD, Maibach H. Effect of air exposure and occlusion on experimental human skin wounds. *Nature*. 1963;200:377–378. (LOE: 2)
 88. Alvarez OM, Mertz PM, Eaglstein WH. The effect of occlusive dressings on collagen synthesis and re-epithelialization in superficial wounds. *J Surg Res*. 1983;35(2):142–148. (LOE: 2)
 89. Mertz PM, Marshall DA, Eaglstein WH. Occlusive wound dressings to prevent bacterial invasion and wound infection. *J Am Acad Dermatol*. 1985;12(4):662–668. (LOE: 2)

90. Claus EE, Fusco CF, Ingram T, Ingersoll CD, Edwards JE, Melham TJ. Comparison of the effects of selected dressings on the healing of standardized abrasions. *J Athl Train*. 1998;33(2):145–149. (LOE: 2)
91. Dressing selection and bandaging. In: Myers BA, ed. *Wound Management: Principles and Practice*. 2nd ed. Upper Saddle River, NJ: Pearson Prentice Hall; 2008:123–159. (LOE: 3)
92. Barnett A, Berkowitz RL, Mills R, Vistnes LM. Comparison of synthetic adhesive moisture vapor permeable and fine mesh gauze dressings for split-thickness skin graft donor sites. *Am J Surg*. 1983;145(3):379–381. (LOE: 2)
93. Harvey C. Wound healing. *Orthop Nurs*. 2005;24(2):143–157. (LOE: 3)
94. Myer A. Dressings. In: Kloth LC, McCulloch JM, eds. *Wound Healing: Alternatives in Management*. 3rd ed. Philadelphia, PA: F. A. Davis Company; 2002:232–270. (LOE: 3)
95. Nelson DB, Dilloway MA. Principles, products, and practical aspects of wound care. *Crit Care Nurs Q*. 2002;25(1):33–54. (LOE: 3)
96. Cho M, Hunt TK. The overall clinical approach to wounds. In: Falanga V, ed. *Cutaneous Wound Healing*. London, England: Martin Dunitz, Ltd; 2001:141–154. (LOE: 3)
97. Farion KJ, Russell KF, Osmond MH, et al. Tissue adhesives for traumatic lacerations in children and adults. *Cochrane Database Syst Rev*. 2002;(3):CD003326. (LOE: 1)
98. Dumville JC, Coulthard P, Worthington HV, et al. Tissue adhesives for closure of surgical incisions. *Cochrane Database Syst Rev*. 2014;11:CD004287. (LOE: 2)
99. Jones AM, San Miguel L. Are modern wound dressings a clinical and cost-effective alternative to the use of gauze? *J Wound Care*. 2006;15(2):65–69. (LOE: 3)
100. Thomas S. A structured approach to the selection of dressings. World Wide Wounds Web site. <http://www.worldwidewounds.com/1997/july/Thomas-Guide/Dress-Select.html>. Published July 1997. Accessed January 21, 2016. (LOE: 3)
101. Dumville JC, Gray TA, Walter CJ, Sharp CA, Page T. Dressings for the prevention of surgical site infection. *Cochrane Database Syst Rev*. 2014;9:CD003091. (LOE: 2)
102. Wiechula R. The use of moist wound-healing dressings in the management of split-thickness skin graft donor sites: a systematic review. *Int J Nurs Pract*. 2003;9(2):S9–S17. (LOE: 1)
103. Alvarez O. Moist environment for healing: matching the dressing to the wound. *Ostomy Wound Manage*. 1988;21:64–83. (LOE: 3)
104. Bolton L. Operational definition of moist wound healing. *J Wound Ostomy Continence Nurs*. 2007;34(1):23–29. (LOE: 2)
105. Kannon GA, Garrett AB. Moist wound healing with occlusive dressings: a clinical review. *Dermatol Surg*. 1995;21(7):583–590. (LOE: 2)
106. Ovington LG. Dressings and skin substitutes. In: McCulloch JM, Kloth L, eds. *Wound Healing: Evidence-Based Management*. 4th ed. Philadelphia, PA: F. A. Davis Company; 2010:180–195. (LOE: 3)
107. Thomas S, Hay P. Fluid handling properties of hydrogel dressings. *Ostomy Wound Manage*. 1995;41(3):54–56, 58–59. (LOE: 2)
108. Eisenbud D, Hunter H, Kessler L, Zulkowski K. Hydrogel wound dressings: where do we stand in 2003? *Ostomy Wound Manage*. 2003;49(10):52–57. (LOE: 3)
109. Cannon BC, Cannon JP. Management of pressure ulcers. *Am J Health Syst Pharm*. 2004;61(18):1895–1905. (LOE: 3)
110. Hutchinson JJ, McGuckin M. Occlusive dressings: a microbiologic and clinical review. *Am J Infect Control*. 1990;18(4):257–268. (LOE: 2)
111. Chaby G, Senet P, Vaneau M, et al. Dressings for acute and chronic wounds: a systematic review. *Arch Dermatol*. 2007;143(10):1297–1304. (LOE: 2)
112. Beam JW. Effects of occlusive dressings on healing of partial-thickness abrasions. *Athl Train Sports Health Care*. 2012;4(2):58–66. (LOE: 2)
113. Ma KK, Chan MF, Pang SM. The effectiveness of using a lipidocolloid dressing for patients with traumatic digital wounds. *Clin Nurs Res*. 2006;15(2):119–134. (LOE: 2)
114. Falanga V. Occlusive wound dressings: why, when, which? *Arch Dermatol*. 1988;124(6):872–877. (LOE: 3)
115. Hess CT. When to use hydrocolloid dressings. *Adv Skin Wound Care*. 2000;13(2):63–64. (LOE: 3)
116. Hollander JE, Singer AJ. Laceration management. *Ann Emerg Med*. 1999;34(3):356–367. (LOE: 3)
117. Piacquadio D, Nelson DB. Alginates. A “new” dressing alternative. *J Dermatol Surg Oncol*. 1992;18(11):992–995. (LOE: 3)
118. Perron AD, Garcia JA, Parker Hays E, Schafermeyer R. The efficacy of cyanoacrylate-derived surgical adhesive for use in the repair of lacerations during competitive athletics. *Am J Emerg Med*. 2000;18(3):261–263. (LOE: 2)
119. Branfield AS. Use of tissue adhesives in sport? A new application in international ice hockey. *Br J Sports Med*. 2004;38(1):95–96. (LOE: 2)
120. Hess CT, Kirsner RS. Orchestrating wound healing: assessing and preparing the wound bed. *Adv Skin Wound Care*. 2003;16(5):246–257. (LOE: 3)
121. Vermeulen H, van Hattem JM, Storm-Versloot MN, Ubbink DT, Westerbos SJ. Topical silver for treating infected wounds. *Cochrane Database Syst Rev*. 2007;1:CD005486. (LOE: 2)
122. Fonder MA, Mamelak AJ, Lazarus GS, Chanmugam A. Occlusive wound dressings in emergency medicine and acute care. *Emerg Med Clin North Am*. 2007;25(1):235–242. (LOE: 3)
123. Wijetunge DB. Management of acute and traumatic wounds: main aspects of care in adults and children. *Am J Surg*. 1994;167(1A):S56–S60. (LOE: 3)
124. Lee CK, Hansen SL. Management of acute wounds. *Surg Clin North Am*. 2009;89(3):659–676. (LOE: 3)
125. Wysocki AB. Evaluating and managing open skin wounds: colonization versus infection. *AACN Clin Issues*. 2002;13(3):382–397. (LOE: 3)
126. Scanlon E. Wound infection and colonisation. *Nurs Stand*. 2005;19(24):57–58, 60, 62. (LOE: 3)
127. Bailey E, Kroshinsky D. Cellulitis: diagnosis and management. *Dermatol Ther*. 2011;24(2):229–239. (LOE: 3)
128. Dryden MS. Skin and soft tissue infection: microbiology and epidemiology. *Int J Antimicrob Agents*. 2009;34(suppl 1):S2–S7. (LOE: 3)
129. Zinder SM, Basler RSW, Foley J, Scarlata C, Vasily DB. National Athletic Trainers’ Association position statement: skin diseases. *J Athl Train*. 2010;45(4):411–428. (LOE: 2)
130. Franz MG, Steed DL, Robson MC. Optimizing healing of the acute wound by minimizing complications. *Curr Probl Surg*. 2007;44(11):691–763. (LOE: 3)
131. Dulecki M, Pieper B. Irrigating simple acute traumatic wounds: a review of the current literature. *J Emerg Nurs*. 2005;31(2):156–160. (LOE: 2)
132. Field CK, Kerstein MD. Overview of wound healing in a moist environment. *Am J Surg*. 1994;167(1A):S2–S6. (LOE: 3)
133. Lee I, Agarwal RK, Lee BY, Fishman NO, Umscheid CA. Systematic review and cost analysis comparing use of chlorhexidine with use of iodine for preoperative skin antisepsis to prevent surgical site infection. *Infect Control Hosp Epidemiol*. 2010;31(12):1219–1229. (LOE: 1)
134. Cummings P, Del Beccaro MA. Antibiotics to prevent infection of simple wounds: a meta-analysis of randomized studies. *Am J Emerg Med*. 1995;13(4):396–400. (LOE: 1)
135. Hood R, Shermock KM, Emerman C. A prospective, randomized pilot evaluation of topical triple antibiotic versus mupirocin for the prevention of uncomplicated soft tissue wound infection. *Am J Emerg Med*. 2004;22(1):1–3. (LOE: 2)

136. Atiyeh BS, Ioannovich J, Al-Amm CA, El-Musa KA. Management of acute and chronic open wounds: the importance of moist environment in optimal wound healing. *Curr Pharm Biotechnol.* 2002;3(3):179–195. (LOE: 3)
137. Lio PA, Kaye ET. Topical antibacterial agents. *Med Clin North Am.* 2011;95(4):703–721. (LOE: 2)
138. Nakamura Y, Daya M. Use of appropriate antimicrobials in wound management. *Emerg Med Clin North Am.* 2007;25(1):159–176. (LOE: 2)
139. Langford JH, Artemi P, Benrimoj SI. Topical antimicrobial prophylaxis in minor wounds. *Ann Pharmacother.* 1997;31(5):559–563. (LOE: 2)
140. Maddox JS, Ware JC, Dillon HC. The natural history of streptococcal skin infection: prevention with topical antibiotics. *J Am Acad Dermatol.* 1985;13(2, pt 1):207–212. (LOE: 2)
141. Dire DJ, Coppola M, Dwyer DA, Lorette JJ, Karr JL. Prospective evaluation of topical antibiotics for preventing infections in uncomplicated soft-tissue wounds repaired in the ED. *Acad Emerg Med.* 1995;2(1):4–10. (LOE: 2)
142. Waterbrook AL, Hiller K, Hays DP, Berkman M. Do topical antibiotics help prevent infection in minor traumatic uncomplicated soft tissue wounds? *Ann Emerg Med.* 2013;61(1):86–88. (LOE: 1)
143. Saryan JA, Dammin TC, Bouras AE. Anaphylaxis to topical bacitracin zinc ointment. *Am J Emerg Med.* 1998;16(5):512–513. (LOE: 3)
144. Krauthaim AB, Jermann TH, Bircher AJ. Chlorhexidine anaphylaxis: case report and review of the literature. *Contact Dermatitis.* 2004;50(3):113–116. (LOE: 3)
145. Gette MT, Marks JG, Maloney ME. Frequency of postoperative allergic contact dermatitis to topical antibiotics. *Arch Dermatol.* 1992;128(3):365–367. (LOE: 2)
146. Dragu A, Unglaub F, Schwarz S, et al. Foreign body reaction after usage of tissue adhesives for skin closure: a case report and review of the literature. *Arch Orthop Trauma Surg.* 2009;129(2):167–169. (LOE: 3)
147. Goossens A, Cleenewerck MB. New wound dressings: classification, tolerance. *Eur J Dermatol.* 2010;20(1):24–26. (LOE: 3)
148. Burns JL, Mancoll JS, Phillips LG. Impairments to wound healing. *Clin Plast Surg.* 2003;30(1):47–56. (LOE: 3)
149. Greenhalgh DG. Wound healing and diabetes mellitus. *Clin Plast Surg.* 2003;30(1):37–45. (LOE: 3)
150. Anstead GM. Steroids, retinoids, and wound healing. *Adv Wound Care.* 1998;11(6):277–285. (LOE: 3)
151. Freiman A, Bird G, Metelitsa AI, Barankin B, Lauzon GJ. Cutaneous effects of smoking. *J Cutan Med Surg.* 2004;8(6):415–423. (LOE: 2)
152. Park JE, Barbul A. Understanding the role of immune regulation in wound healing. *Am J Surg.* 2004;187(5A):S11–S16. (LOE: 3)
153. Cheung AH, Wong LM. Surgical infections in patients with chronic renal failure. *Infect Dis Clin North Am.* 2001;15(3):775–796. (LOE: 3)
154. Patterson GK, Martindale RG. Nutrition and wound healing. In: McCulloch JM, Kloth L, eds. *Wound Healing: Evidence-Based Management.* 4th ed. Philadelphia, PA: F. A. Davis Company; 2010:44–50. (LOE: 3)
155. Ehrlich HP, Hunt TK. Effects of cortisone and vitamin A on wound healing. *Ann Surg.* 1968;167(3):324–328. (LOE: 2)
156. Ponc M, de Haas C, Bachra BN, Polano MK. Effects of glucocorticosteroids on primary human skin fibroblasts, I: inhibition of the proliferation of cultured primary human skin and mouse L929 fibroblasts. *Arch Dermatol Res.* 1977;259(2):117–123. (LOE: 2)
157. Salcido RS. Do anti-inflammatories have a role in wound healing? *Adv Skin Wound Care.* 2005;18(2):65–66. (LOE: 3)
158. Telfer NR, Moy RL. Drug and nutrient aspects of wound healing. *Dermatol Clin.* 1993;11(4):729–737. (LOE: 3)
159. Busti AJ, Hooper JS, Amaya CJ, Kazi S. Effects of preoperative antiinflammatory and immunomodulating therapy on surgical wound healing. *Pharmacotherapy.* 2005;25(11):1566–1591. (LOE: 2)
160. Kearney MT, Duncan ER, Kahn M, Wheatcroft SB. Insulin resistance and endothelial cell dysfunction: studies in mammalian models. *Exp Physiol.* 2008;93(1):158–163. (LOE: 2)
161. Falanga V, Eaglstein WH. The “trap” hypothesis of venous ulceration. *Lancet.* 1993;341(8851):1006–1008. (LOE: 2)
162. Lee PW, Elsaie ML, Jacob SE. Allergic contact dermatitis in children: common allergens and treatment: a review. *Curr Opin Pediatr.* 2009;21(4):491–498. (LOE: 3)
163. Rees A, Sherrod Q, Young L. Chemical burn from povidone-iodine: case and review. *J Drugs Dermatol.* 2011;10(4):414–417. (LOE: 3)
164. White RJ, Cutting KF. Interventions to avoid maceration of the skin and wound bed. *Br J Nurs.* 2003;12(20):1186–1201. (LOE: 3)
165. Hollinworth H. Challenges in protecting peri-wound skin. *Nurs Stand.* 2009;24(7):53–54, 56, 58. (LOE: 3)
166. Ameen H, Moore K, Lawrence JC, Harding KG. Investigating the bacterial barrier properties of four contemporary wound dressings. *J Wound Care.* 2000;9(8):385–388. (LOE: 3)
167. Bowler PG, Delargy H, Prince D, Fondberg L. The viral barrier properties of some occlusive dressings and their role in infection control. *Wounds Compend Clin Res Pract.* 1993;5(1):1–8. (LOE: 3)
168. Ayello EA, Dowsett C, Schultz GS, et al. TIME heals all wounds. *Nursing.* 2004;34(4):36–41. (LOE: 3)
169. Ennis WJ, Meneses P. Complications in repair. In: McCulloch JM, Kloth LC, eds. *Wound Healing: Evidence-Based Management.* 4th ed. Philadelphia, PA: F. A. Davis Company; 2010:51–64. (LOE: 3)
170. Hom DB, Adams G, Koreis M, Maisel R. Choosing the optimal wound dressing for irradiated soft tissue wounds. *Otolaryngol Head Neck Surg.* 1999;121(5):591–598. (LOE: 3)
171. How to care for your wound after it's treated with Dermabond Advanced topical skin adhesive. Ethicon Web site. http://www.smarterpatient.com/sites/default/files/managed-documents/dermabond_advanced_caring_for_your_wound-english.pdf. Accessed January 22, 2016. (LOE: 3)
172. Histoacryl and Histoacryl Blue topical skin adhesive. TissueSeal Web site. <http://www.tissueseal.com/PackageInsertfinaldk.pdf>. Accessed January 22, 2016. (LOE: 3)
173. Fletcher J. Understanding wound dressings: hydrocolloids. *Nurs Times.* 2005;101(46):51. (LOE: 3)
174. Miller MG, Berry DC. Recognition and management of soft tissue injuries. In: Miller MG, Berry DC, eds. *Emergency Response Management for Athletic Trainers.* Philadelphia, PA: Lippincott Williams & Wilkins; 2011:283–309. (LOE: 3)
175. Davidson JM. Animal models for wound repair. *Arch Dermatol Res.* 1998;290(suppl):S1–S11. (LOE: 3)
176. Lazarus GS, Cooper DM, Knighton DR, et al. Definitions and guidelines for assessment of wounds and evaluation of healing. *Arch Dermatol.* 1994;130(4):489–493. (LOE: 3)
177. Waspe J. Tissue viability. Treating leg ulcers with high pressure irrigation devices. *Nurs Stand.* 1996;11(6):53–54. (LOE: 3)
178. Flanagan M. Wound cleansing. In: Morrison M, Moffat C, Bridel-Nixon J, Bale S, eds. *Nursing Management of Chronic Wounds.* London, United Kingdom: Mosby Co; 1997:87–102. (LOE: 3)
179. Williams C. Wound irrigation techniques: new Steripod normal saline. *Br J Nurs.* 1999;8(21):1460–1462. (LOE: 3)
180. Fernandez RS, Griffiths R, Ussia C. Water for wound cleansing. *Int J Evid Based Healthc.* 2007;5(3):305–323. (LOE: 1)
181. Goldenberg MS. Wound care management: proper protocol differs from athletic trainers' perceptions. *J Athl Train.* 1996;31(1):12–16. (LOE: 2)
182. Gannon R. Wound cleansing: sterile water or saline? *Nurs Times.* 2007;103(9):44–46. (LOE: 3)

183. Brennan S, Leaper D. The effect of antiseptics on the healing wound: a study using the rabbit ear chamber. *Br J Surg*. 1985;72(10):780–782. (LOE: 2)
184. Foresman PA, Payne DS, Becker D, Lewis D, Rodeheaver GT. A relative toxicity index for wound cleansers. *Wounds*. 1993;5(5):226–231. (LOE: 3)
185. Surgical site infections: prevention and treatment. National Institute for Health and Care Excellence Web site. <http://www.nice.org.uk/guidance/cg74>. Accessed January 22, 2016. (LOE: 1)
186. Beam JW. Instruction of wound management in entry-level undergraduate CAAHEP accredited athletic training education programs. *J Athl Train*. 2005;40(suppl 2):S17–S18. (LOE: 3)
187. Baranoski S. Wound assessment and dressing selection. *Ostomy Wound Manage*. 1995;41(suppl 7A):S7–S12. (LOE: 3)
188. Bethell E. Why gauze dressings should not be the first choice to manage most acute surgical cavity wounds. *J Wound Care*. 2003;12(6):237–239. (LOE: 3)
189. Storm-Versloot MN, Vos CG, Ubbink DT, Vermeulen H. Topical silver for preventing wound infection. *Cochrane Database Syst Rev*. 2010;(3):CD006478. (LOE: 2)
190. Bergin S, Wraight P. Silver based wound dressings and topical agents for treating diabetic foot ulcers. *Cochrane Database Syst Rev*. 2006;(1):CD005082. (LOE: 2)
191. Hermans MH. Clinical benefit of a hydrocolloid dressing in closed surgical wounds. *J ET Nurs*. 1993;20(2):68–72. (LOE: 2)
192. Michie DD, Hugill JV. Influence of occlusive and impregnated gauze dressings on incisional healing: a prospective, randomized, controlled study. *Ann Plast Surg*. 1994;32(1):57–64. (LOE: 2)
193. Heffernan A, Martin AJ. A comparison of a modified form of Granuflex (Granuflex Extra Thin) and a conventional dressing in the management of lacerations, abrasions and minor operation wounds in an accident and emergency department. *J Accid Emerg Med*. 1994;11(4):227–230. (LOE: 2)
194. Thomas DW, Hill CM, Lewis MA, Stephens P, Walker R, Von Der Weth A. Randomized clinical trial of the effect of semi-occlusive dressings on the microflora and clinical outcome of acute facial wounds. *Wound Repair Regen*. 2000;8(4):258–263. (LOE: 2)
195. Motta GJ. Dressed for success: how moisture-retentive dressings promote healing. *Nursing*. 1993;23(12):26–33. (LOE: 3)
196. Edwards R, Harding KG. Bacteria and wound healing. *Curr Opin Infect Dis*. 2004;17(2):91–96. (LOE: 3)
197. Abrahamian FM, Talan DA, Moran GJ. Management of skin and soft-tissue infections in the emergency department. *Infect Dis Clin North Am*. 2008;22(1):89–116. (LOE: 3)
198. Hook EW, Hooton TM, Horton CA, Coyle MB, Ramsey PG, Turck M. Microbiologic evaluation of cutaneous cellulitis in adults. *Arch Intern Med*. 1986;146(2):295–297. (LOE: 2)
199. Sigurdsson AF, Gudmundsson S. The etiology of bacterial cellulitis as determined by fine-needle aspiration. *Scand J Infect Dis*. 1989;21(5):537–542. (LOE: 2)
200. Eriksson B, Jorup-Rönström C, Karkkonen K, Sjöblom AC, Holm SE. Erysipelas: clinical and bacteriologic spectrum and serological aspects. *Clin Infect Dis*. 1996;23(5):1091–1098. (LOE: 2)
201. Kielhofner MA, Brown B, Dall L. Influence of underlying disease process on the utility of cellulitis needle aspirates. *Arch Intern Med*. 1988;148(11):2451–2452. (LOE: 2)
202. Bernard P, Bedane C, Mounier M, Denis F, Catanzano G, Bonnetblanc JM. Streptococcal cause of erysipelas and cellulitis in adults: a microbiologic study using a direct immunofluorescence technique. *Arch Dermatol*. 1989;125(6):779–782. (LOE: 2)
203. Moet GJ, Jones RN, Biedenbach DJ, Stilwell MG, Fritsche TR. Contemporary causes of skin and soft tissue infections in North America, Latin America, and Europe: report from the SENTRY Antimicrobial Surveillance Program (1998–2004). *Diagn Microbiol Infect Dis*. 2007;57(1):7–13. (LOE: 2)
204. Rajan S. Skin and soft-tissue infections: classifying and treating a spectrum. *Cleve Clin J Med*. 2012;79(1):57–66. (LOE: 3)
205. Thomson PD, Smith DJ. What is infection? *Am J Surg*. 1994;167(1A):S7–S10. (LOE: 3)
206. Hollander JE, Singer AJ, Valentine SM, Shofer FS. Risk factors for infection in patients with traumatic lacerations. *Acad Emerg Med*. 2001;8(7):716–720. (LOE: 2)
207. Lammers RL, Hudson DL, Seaman ME. Prediction of traumatic wound infection with a neural network-derived decision model. *Am J Emerg Med*. 2003;21(1):1–7. (LOE: 2)
208. O'Dell M. Skin and wound infections: an overview. *Am Fam Physician*. 1998;57(10):2424–2432. (LOE: 3)
209. Nemeth AJ, Eaglstein WH, Taylor JR, Peerson LJ, Falanga V. Faster healing and less pain in skin biopsy sites treated with an occlusive dressing. *Arch Dermatol*. 1991;127(11):1679–1683. (LOE: 2)
210. Turner T. Which dressing and why? *Nurs Times*. 1982;78(29):1–3. (LOE: 3)
211. Barr J. Physiology of healing: the basis for the principles of wound management. *Medsurg Nurs*. 1995;4(5):387–392. (LOE: 3)
212. Bloodborne pathogens and needlestick prevention standards. US Department of Labor Occupational Safety & Health Administration Web site. http://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=STANDARDS&p_id=10051. Accessed January 22, 2014. (LOE: 2)
213. Preventing healthcare-associated infections. Centers for Disease Control and Prevention Web site. <http://www.cdc.gov/HAI/prevent/prevention.html>. Accessed January 22, 2014. (LOE: 2)
214. Guideline 2J: skin infections. In: Parsons JT, ed. *2014–15 NCAA Sports Medicine Handbook*. 25th ed. Indianapolis, IN: National Collegiate Athletic Association; 2014:65–71. (LOE: 3)

Address correspondence to Joel W. Beam, EdD, LAT, ATC, Clinical and Applied Movement Sciences, Brooks College of Health, University of North Florida, 1 UNF Drive, Jacksonville, FL 32224-2673. Address e-mail to jbeam@unf.edu.