Management of Sports-Induced Skin Wounds

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ABSTRACT: Skin wounds are common in sports but are rarely documented by the certified athletic trainer. The literature is unclear about wound types, and none of the articles reviewed reported frequencies. The purpose of this paper is to discuss the frequency of common athletic skin wounds and their specific management. Management of skin wounds can sometimes be problematic. Hydrogen peroxide has been used on wounds since 1947, yet some researchers report that hydrogen peroxide and iodophor solution can delay or interfere with wound healing, or cause damage to the wounded area if use is intense and prolonged. Occlusive dressings have been reported to have considerable advantage in maintaining a moist wound bed and in decreasing healing time. Infection rates beneath occlusive dressings, however, are similar to those associated with other types of dressings. Complications to wounds, with or without the use of occlusive dressings, such as keloids and seborrheic dermatitis, occur in low frequencies. Due to a lack of specific information about sports-induced skin wounds and their management, we recommend that standardized documentation for common wounds be developed along with further study of techniques for management.

Skin wounds are frequent among athletes and other exercisers.[2,3] They occur regularly but usually have little effect on athletic performance; therefore, details about skin wounds among athletes are limited. It appears that athletic trainers (ATCs) did not formally report their experiences with athletic wounds until the 1993 OSHA regulations helped to identify ATCs as an at-risk group.[7,15,28,34,36] Because ATCs provide on-site health care services,[17,33,39] they are the primary managers of acute care for sports-induced skin wounds.

We initially reviewed the literature concerning skin wounds, cleansing agents, and dressings to learn more about how to decrease the risk to athletic trainers as they come into physical contact with an athlete's body fluids and tissues while managing wounds. As a result of the review, however, we became interested in the most effective and efficient wound management technique. Ineffective management may contribute to wound infection or may compromise physical performance.

WOUND FREQUENCY

Few authors report frequency distributions for skin wounds. In 1948, Thorndyke stated, "Without any doubt the most common inflammatory reaction that is encountered in the training of athletes, is the common ordinary blister."[38] We could find no other author in the athletic training literature who has commented as clearly about the frequency of wounds.[2,3,9] Our review of the literature revealed that the terms "often" and "common" described frequencies for all skin wound types except punctures, which occurred "rarely."

In an attempt to assign numerical statistics to the above descriptions, we found no other frequency reports. Our impressions about wounds may be vivid, but our knowledge is vague. We do not know how frequently skin wounds occur, nor how they occur. We do not even know individual characteristics about many wound types that might guide us in developing prevention strategies. If skin wound frequencies are high, the risk of ATC exposure to biohazards is also high. ATC time commitment to, and supply of resources for management and follow-up, must also be substantial. Efforts to record wound care practices may lead to the development of direct wound care studies and standardized documentation.

TYPES OF WOUNDS

Based on empirical evidence, four skin wound types occur frequently in athletics: abrasion, blister, incision, and laceration. Our discussion focuses on abrasions and blisters.

The athletic training literature[2,3,9] indicates that both abrasions and blisters occur when shear stress is applied to the skin. According to the literature on dermatology, blisters occur when the skin is wet.[37] Athletes suffer these two wound types when they slide across playing surfaces or slide within their shoes.

Abrasions

Abrasions typically are categorized into two subgroups: Partial-thickness wounds,[1] wherein the epidermis or superficial dermis is abraded or removed, and full-thickness wounds, wherein skin is removed down to and sometimes including the subcutaneous fat layer.[10] Wound healing studies generally address one or both of these categories.

Blisters

Sulzberger and co-workers[37] produced skin blisters in naval cadets using linear rubbing and twisting or rotating machinery.

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Intact fluid-filled blisters were difficult to produce except on the palms and soles. Other body areas targeted for blistering were the back, buttocks, shins, forearms, upper arms, and thighs. In these areas, abrasions rather than blisters were produced. The specific damage associated with a blister seems to arise from friction combined with moisture. Sulzberger[37] added moisture, in very small increments, to the rubbing or twisting. He reported that as soon as moisture was present, friction at the skin surface gradually increased to a maximum. These findings are important in relation to the role of sweating and the nature of the material worn next to the skin. The researchers concluded that the production of a blister versus that of an abrasion varied with skin thickness; the palms and soles are more likely to blister due to their thick outer stratum corneum or horny layer. This finding was inconsistent, however, within the same person and the same body part.

WOUND CLEANSING

Initiation of wound care is typically marked by cleansing the wound and surrounding skin.[6] In the preparation and cleansing of wounds, ATCs should observe universal precautions. Infection in an open injury poses a potentially serious problem. Complications associated with infection are: 1) tissue damage, 2) foreign body contamination, and 3) bacterial inoculation.[6,20] The principles of wound management are designed to prevent further bacterial inoculation and to debride and cleanse the wound of necrotic tissue and foreign material.[6,11]

Athletic trainers apply special solutions and wash with water or other agents to prevent infection, debride wound material and tissue, and cleanse the surrounding intact skin.[2,3,9] Hydrogen peroxide solutions have been used since 1947[6] to reduce bacterial contamination and to enhance the physical debridement or cleansing of a wound. Studies on the effects of these and more recently developed agents for wound healing began in the 1970s.

Solutions

In 1991, Gruber et al[16] treated partial-thickness wounds with acetic acid (.25%), provodone-iodine (Betadine), or hydrogen peroxide (3%) four times daily. A control wound was treated with normal saline. They found a shorter time to full healing with the hydrogen peroxide-treated group and no difference in the other two solutions from that of normal saline. They observed wound healing by gross visual inspection. These investigators noted complete epithelialization when they saw a pink color at the wounded area and no scab. This visual method of determining healing is common in skin wound research and relevant to our clinical use. However, the authors noted that the peroxide-treated wounds developed air-filled blisters. Further, they suggested that if the wounds were treated this intensely with peroxide for longer than 4 days, the blisters would have ulcerated. Finally, at 3 months, these researchers found no gross differences in pigmentation or texture among wound sites, regardless of treatment.

Ointments

Neosporin ointment (Burroughs Wellcome Co, Research Triangle Park, NC) was developed to prevent further bacterial inoculation and has been studied by Welsh[40] for its effect on wound healing. Welsh showed that Neosporin did not delay or speed healing.

A related topical agent to Neosporin, Polysporin ointment (Burroughs Wellcome), has two antimicrobial agents: bacitracin and polymixin B sulfate. These agents are also found in Neosporin. After separating the two compounds in Polysporin, Eaglstein[11] reported that the use of bacitracin alone stimulated wound healing; neither polymyxin B sulfate nor Polysporin in compound affected wound repair.

Creams

Another antimicrobial agent often prescribed for wounds is Silvadene Cream (silver sulfadiazine). Welsh[40] reported that Silvadine was less effective than Betadine in reducing wound healing time. He compared the two cleansing agents without a control. Since Betadine ointment has been shown to be ineffective in decreasing healing time compared to controls, we hypothesize from Welsh's work that Silvadene either may interfere with re-epithelialization or may alter acute responses in some way.

Welsh[40] also reported significant bacterial reduction with the use of Johnson & Johnson First Aid Cream (Johnson & Johnson, New Brunswick, NJ) compared with Camphophenic gel (Winthrop Consumer Products, New York, NY). However, Camphophenic was thought to interfere with wound healing. First Aid Cream did not affect the rate of healing compared to a control.

Lotions and Soaps

Eaglstein et al[11] conducted studies on numerous topical agents used in wound antisepsis. They identified lotions containing zinc soap, oil-in-water cream, Neosporin, Silvadine, and Pan Oxyl (benzoyl peroxide) lotion (10% and 20%). Each of these agents accelerated epidermal healing when compared with controls. As noted here, Eaglstein's results were different from previous studies. Conflicting results among studies may be related to the common use of study specimens from different animal species. Although similar to monkey and pig skin as used in many studies, Montagna[30] showed that human skin is unique. In the absence of direct human skin comparisons, results of these studies should be interpreted with caution.

Ongoing wound studies have contributed to our knowledge of topical cleansing or antiseptic agents and their toxic effects on a variety of wounds. Through the early process of normal wound healing, the barrier properties of the wounded area are compromised. During the first 10 days, for example, the wound site may be susceptible to the toxic effects of the cleansing and antiseptic agents, since they are now more easily absorbed through the thin wound epithelium. The outer layer of cells, called stratum corneum, is needed to maintain the normal barrier function of the skin.[18,30] Due to the ready absorption of toxicants when new skin with just a few layers or cells covers a wound, Betadine is one of those topical agents that dermatologists recommend to use with caution. Caution is especially important when treating patients with pre-existing thyroid or renal conditions.[11,16,31,40] Since skin wounds are missing the stratum corneum barrier, vehicles or topical bases which are inactive on undamaged skin might have biochemical effects on wounded skin. Sometimes cleaning agents (ie, detergents and soaps) that are effective in retarding bacterial growth become toxic when applied to the altered physiology of the healing wound. The concentration of alkalines in soap or combinations of other compounds such as camphor or alcohol act as toxic agents to wounded skin.[12] Mild Ivory soap, in contrast, has antibacterial effects similar to Betadine but does not interfere with wound repair.[12]

WOUND DRESSING

Once the initial cleansing, debridement, and antiseptic procedures have been completed, the next step is to cover the wound. Acute, bleeding wounds are covered, even if they stop bleeding, in order to prevent further wound inoculation.[2,3,6,8,20] Coverings for wound compression to stop bleeding and those that provide a mechanical barrier are usually one and the same.

Reports on dressings have only recently been updated and exclusively address occlusive dressings.[21] A broad spectrum of occlusive dressings has been commercially available for the past 10 years. Some of these were developed in response to the fear of cross-contamination of HIV via open wounds,[7,11] and some from wound-healing studies, which support the principle of "moist wound healing."[10,13,18,21,25,28,42]

Athletic training literature promoted moist wound healing before the 1950s.[9] Some advantages to skin healing beneath occlusive dressings have been recognized in the medical literature for over 20 years.[7] A moist versus a dry wound environment was evaluated in 1975 by Gruber et al,[16] who reported that the moist environment required 3 days less to reach wound-healing criteria. Despite this advantage, fear that the moist environment beneath an occlusive dressing would produce more infections seemed to retard the commercial development of these dressings until recently.[20]

For purposes of this discussion, occlusive dressings are those that retain moisture in healing tissues. Semi-occlusive dressings readily accumulate moisture vapor under the dressing. Examples of commercially available dressings are shown in the Table.

Composition and Trade Names of Occlusive Dressings

Composition	Trade Name
Polyurethane films	Op-Site Tegaderm Bioclusive*
Polyethylene oxide hydrogel with polyethylene film backing	Vigilon
Hydrocolloid particles (gelatin and pectin) in a hydrophobic polymer	DuoDERM

* Johnson & Johnson, New Brunswick, NJ.

Occlusive dressings increase the speed of healing in both acute and chronic wounds.[11,34] Winter[41] and Hinman[18] showed that shallow wounds epithelialized faster beneath a plastic film than when the wound was allowed to air-dry. Occlusion increased epithelialization in: human incisions treated with Saran wrap (Dow Chemical Co, Indianapolis, IN); human donor sites treated with Tegaderm (3M Health Care, St Paul, MN) or Op-site (Smith and Nephew Medical, Ltd, Memphis, TN); shallow wounds treated with Vigilon (CR Bard, Inc, Murray Hill, NJ) or Duo-DERM (Bristol-Myers Squibb Co, Princeton, NJ); and dermabrasions, excisions, and scalp transplants treated with Vigilon.

According to some researchers, the rate of acute wound epithelialization increases by 30% to 45% beneath occlusive dressings[10,34] when compared to nonocclusive dressings. What factors are present with occlusive dressings that account for the increased rate of epithelialization? Researchers[10,18,34,42] cite the following important factors: 1) moist wound bed providing an easier route for epidermal migration, 2) increased oxygen partial pressure, 3) enhanced availability of growth factors, 4) favorable effects of microflora, and 5) maintenance of the electrical potential between wounded and nonwounded skin. Since three of these areas have the most available literature to support them, they deserve more in-depth clarification and discussion.

Moist Wound Bed

During healing in a dried wound, the epidermis migrates beneath dead tissue and crust to find a living tissue bed. Occlusive dressings prevent crust formation and wound bed tissue dessication.[4,11,18,42] Epidermal migration is presumed to be faster under occlusive dressings, because it takes place over a moist tissue layer rather than being obstructed by crust and dead tissue. Therefore, maintaining a moist wound also promotes a free and unrestricted environment for wound resurfacing.

Microflora

The desirable and undesirable effects of microflora have been studied for centuries.[20] Bacteria found on the surface of intact skin and in all skin wounds are usually considered undesirable. However, in the proper environment, bacteria or their metabolites stimulate epidermal migration and healing.[6,20,26] "It is important to realize that the presence of bacteria within a wound is not indicative of infection, and diagnosis should be based on the classical clinical signs together with supportive microbiology."[21]

The critical value that defines an infectious colony is 100 000 bacteria per gram of tissue. Hutchinson[21] has shown that colonization is present in occlusive wounds but at levels that are less than critical. In Hutchinson's study, only 2.8% of wounds dressed with occlusive dressings became infected, compared to 7.5% of all wounds dressed in some other manner.

Electrical Potential

Of recent interest is evidence supporting epidermal migration under the influence of electrical fields in wounds.[5,22] These studies have shown that the epidermis produces a voltage similar to a battery, the inside being positive and the outside or skin surface being negative. The deeper the wound extends to the basal layer of epidermis, the lower the current produced. When wounds are produced in the skin, the resultant effect on the local electrical circuitry has been likened to that of a short circuit. As the skin wound dries, its current is switched off and the voltage gradient around the periphery of the wound is lost. Researchers suggest that occlusive dressings may increase epidermal migration because they prevent wounds from drying and losing their natural voltage gradient.[5,22]

Occlusive dressings are now promoted for use as postoperative dressings, donor site dressings, ulcer treatments, covers for minor skin abrasions, backing for donor tissues, and dressings for blisters.[6,21] As ATCs become more familiar with the use of occlusive dressings, these adjuncts may become more prominent in the wound management of athletes.

HEALING TIME

Research protocols that have evaluated cleansing agents typically used four treatments per day.[12,16,29,40] Conversely, studies investigating occlusive dressings describe dressing changes at varied time intervals.[11,13,18,28,42] Through 7 days, these occlusive dressings may only have been changed twice.[12,28] Dressings were applied after initial cleansing and debridement for clinical cases, and applied without other treatment for laboratory animals. Researchers evaluated healing time by visual inspection. The effect of these two treatments on wound healing time can be attributed to the agent or dressing, but not to their combination.

Mellion et al[28] saw a 4- to 8-day healing time for abrasions using a hydrocolloid occlusive dressing. Pollack and others[34] treated many of their acute wounds with several types of dressings for 5 to 7 days. At the end of this time, the wounds appeared similar and resurfaced with new cells. Mean healing times, taken from reports that focused on cleansing agents in partial-thickness wounds, ranged from 7 to 15 days.[1,12,31,41,42] It is not clear how thick the epidermis became by the end of treatment in these cases, but most of the studies identified significant resurfacing of cells within 24 hours.

COMPLICATIONS

Pain and Infection

Pain and infection are wound complications previously discussed. The useful effects of pain reduction and decreased tenderness[10,13,20] associated with occlusive dressings have been nearly universally observed clinically. Tissue drying at the wound site with subsequent crust formation may be the cause of pain.[13] Dermatologists conclude that this pain is probably mechanical in nature from normally mobile skin being restricted by crust.[10,13,20] Acute inflammatory pain is commonly present, but persistent or severe pain may be a sign of infection.

Pollack[34] compared clean traumatic wounds to infected ones; surgical or traumatic wounds show mild, nonpainful erythema from the wound borders up to 5 mm around—like a halo. This is usually not associated with a purulent exudate if wound cleanliness is maintained. However, infected wounds exhibit an erythema that reaches beyond 5 mm from the wound's borders. The area is tender to palpation and may or may not be associated with fever, streaking, or swollen lymph nodes.[14] With pressure to the margins, pus or blood serum-like exudate is evident.

Bacteria are present in wounds and aid the wound repair process by stimulating a strong response from migrating white cells.[26,41] Occlusive dressings appear neutral to microorganisms, especially for clean and well-perfused wounds. Increased oxygen partial pressure[23] is thought to be an important factor in re-epithelialization and important to the reduced incidence of infections.[26] As careful observers and without the benefit of cultures, ATCs may often be confused about the presence or absence of infection in a wound. Pollack suggests that tenderness, a wide margin of erythema, and seeping exudate on pressure are important differentiating features of infection.[34]

Seborrheic Dermatitis

Seborrheic dermatitis is considered a common cause of widespread redness, sometimes progressing to erosions surrounding a wound.[19,41] The condition is frequently seen in athletes with a pre-existing dermatitis and may persist well after the wound is healed. The skin appearance may be confused with infection, but is not painful and is often treated with topical corticosteroids.

Eaglstein,[12] in treating wounds with accompanying dermatitis, reported that topical treatments with an anti-inflammatory corticosteroid (0.1% triamcinolone acetonide) slowed the speed of resurfacing of a wound by about 60%. The effect is apparently related to skin atrophy from the use of corticosteroid. Hydrocortisone (1%) (another anti-inflammatory agent) did not affect resurfacing but did reduce the healing wound's collagen biosynthesis capacity. Reduced synthesis of collagen results in reduced wound strength. ATCs should be careful with the use of topical treatments for seborrheic dermatitis in the presence of a wound.

Scars

Hypertrophic scars and keloids are fibrous tumors.[32] Their development is similar to the accumulation of collagen in normal wound healing during the proliferative stage, but instead of fibroplasia peaking at 3 weeks, it may be extended for months. During this proliferating period, there is an increase in the formation of vascular nodules, which then become surrounded by fibroblasts. The nodules continue to enlarge, evolving into hard, avascular, collections of collagen. Hypertrophic scars enlarge in size or bulk, whereas keloids enlarge by cellular proliferation.

Hypertrophic scarring, an uncommon complication in the healing of superficial wounds,[27] is a more frequent problem for full-thickness wounds.[32] Most full-thickness wounds heal with a thicker central portion than outer portion. The thick central scarring usually diminishes in size after 6 months to 1 year, leaving a soft, even scar. Hypertrophic scars are most common within loose skin and over convex surfaces. Keloids can occur anywhere, but most often in areas such as the upper back, shoulders, anterior chest, and upper arm. Patients between 10 and 30 years of age are most susceptible to keloids. Treatment options for hypertrophic scars and keloids are numerous,[32] including surgery, pressure therapy (frequently for months), radiation, corticosteroid injections, systemic chemotherapy, cryotherapy, and combinations of these.

Contraction of tissues occurs during the healing of deep partial-thickness wounds, and continues even after re-epithelialization is complete.[41] During a normal healing course, wound contraction accounts for a decrease in wound size and a smaller scar. Wound contraction is biphasic. First, there is a contraction of new tissue that peaks after re-epithelialization. This is followed by partial relaxation and softening of the scar. Since contraction largely occurs in the direction of underlying muscle, annular wounds may heal with a linear scar along relaxed skin tension lines.[41] Using skin mobilization treatment techniques after the initial 2 weeks of wound protection may assist a contracting skin wound to maintain fascial movement between tissue layers.

Other Complications

Other poor results from wounds are noted in the dermatologic literature as dysesthesias, unstable scars, excessive granulation, hypopigmentation, and telangiectasis.[6,19,20,30,41] Complication rates are unknown and vary even within an individual, as do the many manifestations of healing wounds.

CONCLUSIONS

- The ATC who treats sports-induced skin wounds must observe universal precautions and must physically cleanse and dress the wound. Frequent contact with open and bleeding wounds elevates the ATC's risk of exposure to blood and body fluids. Along with observing proper precautions, the ATC needs to be knowledgeable about proper wound management. Management practices for wounds varies in the selection of both cleansing agent and dressing.
- We suggest using the following guidelines for maximum efficiency and effectiveness in the use of cleansing agents, antiseptic agents, and dressings. Use cleansing agents that stimulate epidermal migration and do not interfere with fibroplasia; 3% hydrogen peroxide (or a dilution thereof) would be effective early in wound resurfacing, and Betadine or another iodophor for cleansing the surrounding normal skin.
- In the case of blisters, remove the overlying skin, clean, and use one of the hydroactive gel dressings. However, leaving the elevated skin intact may be an effective treatment for painful, partial-thickness blisters, provided the underlying wound is otherwise kept clean and the wound occluded. This conclusion is derived from occlusive dressing studies.[10,15,18,21,25,28,42]
- Because the studies reviewed in this article have revealed the fragile nature of the healing wound, we advocate the use of closures and occlusive dressings until the wound has re-epithelialized. Finally, we suggest enhancing the absorption of antibiotic creams and ointments through the skin, by using these products when the altered barrier properties of young wounds are poorest, and when infection possibilities are the greatest. As long as oxygen partial pressures at the wound site are maximized by maintenance of a moist wound bed and maintenance of circulation, these procedures should be sufficient to practice wound care with confidence.
- We suspect that many athletic trainers throughout the country use the above materials and protocols when cleaning and dressing wounds. However, widely different antiseptics and dressings appear to be similarly effective on healing time when studies are conducted in the clinical setting and in animal studies. In order to develop a base of clinical information, we propose that standardized trials be established to study outcome measures on sports-induced wound healing.

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REFERENCES.

1. Agren M, Chvapil M, Franzen L. Enhancement of re-epithelialization with topical zinc oxide in porcine partial-thickness wounds. *J Surg Res.* 1991;50:101–105.
2. American Academy of Orthopaedic Surgeons. The skin. In: *Athletic Training and Sports Medicine.* 2nd ed. Park Ridge, IL: American Academy of Orthopaedic Surgeons; 1991:441–445.
3. Arnheim D, Prentice W. Skin disorders. In: Arnheim D, Prentice W. *Principles of Athletic Training.* 8th ed. St Louis, MO: Mosby Year Book Publishers; 1993:420–435.
4. Bryant W. Wound healing. *CIBA Clinical Symposia.* 1977;29:1–36.
5. Burr H, Taffel M, Harvey S. An electrometric study of the healing wound in man. *Yale J Biol Med.* 1940;13:483–485.
6. Caldwell M. Topical wound therapy-an historical perspective. *J Trauma.* 1990;30(Suppl):S116-S122.
7. Christie A. The tissue injury cycle and new advances toward its management in open wounds. *Athl Train, JNATA.* 1991;26:274–278.
8. Crow R. Sports-related lacerations. *Phys Sportsmed.* 1993;21:143–147.
9. Dolan J, Holladay L. The skin and athletic considerations. In: Dolan J, Holladay L. *First Aid Management.* 4th ed. Danville, IL: The Interstate Printers & Publishers; 1954:301–310.
10. Eaglstein W. Effect of occlusive dressings on wound healing. *Clin Dermatol.* 1984;2:107–111.
11. Eaglstein W. Current wound management: a symposium. *Clin Dermatol.* 1984;2:134–142.
12. Eaglstein W, Mertz P, Alvarez O. Effect of topically applied agents on healing wounds. *Clin Dermatol.* 1984;2:112–115.
13. Eldad A, Simon G, Kadar T, Kushnir M. Immediate dressing of the burn wound-will it change its natural history? *Burns.* 1991:17:233–238.
14. Fahey T, Sadaty A, Jones W, Barber A, Smoller B, Shires T. Diabetes impairs the late inflammatory response to wound healing. *J Surg Res.* 1991;50:308–313.
15. Fletcher S, Whitehill W, Wright K. Medicated compress for blister treatment. *J Athl Train.* 1993;28:81–82.

16. Gruber R, Vistnes L, Pardoe R. The effect of commonly used antiseptics on wound healing. *Plast Reconstr Surg*. 1975;55:472–476.
17. Health care workers required to treat HIV infected patients. *NATA News*. June 1994:19.
18. Hinman C, Maibach H, Winters G. Effect of air exposure and occlusion on experimental human skin wounds. *Nature*. 1963;200:377–378.
19. Huether S, Kravitz M. Structure, function, and disorders of the integument. In: McCance K, Huether S. *Pathophysiology: The Basis for Disease in Adults and Children*. St Louis, MO: CV Mosby Company; 1990:1390–1394.
20. Hunt T. Basic principles of wound healing. *J Trauma*. 1990;30(Suppl): S122–S128.
21. Hutchinson J, Lawrence J. Wound infection under occlusive dressings. *J Hosp Infect*. 1991;17:83–94.
22. Jaffe L, Vanable J. Electric fields and wound healing. *Clin Dermatol*. 1984;2:34–44.
23. Jensen J, Hunt T. The wound healing curve as a practical teaching device. *Surg Gynecol Obstet*. 1991;173:63–64.
24. Levine N. Friction blisters. *Phys Sportsmed*. Mar 1982;10:84–92.
25. Levy A, Barnes R, Rijswijk L. Evaluation of a new dressing in the treatment of sports-related lesions. *Cutis*. 1987;39:161–164.
26. Leyden J. Effects of bacteria on healing of superficial wounds. *Clin Dermatol*. 1984;2:81–85.
27. Maffulli N, Pintore E, Petricciuolo F. Arthroscopy wounds: to suture or not to suture. *Acta Orthop Belg*. 1991;57:154–156.
28. Mellion M, Fandel D, Wagner W, Kwikkel M. Hydrocolloid dressings in the treatment of turf burns and other athletic abrasions. *Athl Train, JNATA*. 1988;23:341–346.
29. Monafo W, West M. Current treatment recommendations for topical burn therapy. *Burns*. 1990;40:364–373.
30. Montagna W, Parakkal P. *The Structure and Function of Skin*. 3rd ed. New York: Academic Press; 1974:159.
31. Mulliken J, Healey N, Glowacki J. Providone-iodine and tensile strength of wounds in rats. *J Trauma*. 1980;20:323–325.
32. Murray J, Pollack S, Pinnell S. Keloids and hypertrophic scars. *Clin Dermatol*. 1984;2:121–133.
33. OSHA US Department of Labor. *Occupational Health and Safety Standards*. 29 CFR 1910.20.
34. Pollack S. The wound healing process. *Clin Dermatol*. 1984;2:8–16.
35. Poole D. The DP technique for blisters. *Athl Train, JNATA*. 1979;14:88–89.
36. Ramsey M. Managing friction blisters. *Phys Sportsmed*. Jan 1992;20:117–124.
37. Sulzberger M, Cortese T, Fishman L, Wiley H. Studies on blister produced by friction. *J Invest Dermatol*. 1966;47:456–465.
38. Thorndyke A. Infections and inflammations. In: *Athletic Injuries: Prevention Diagnosis and Treatment*. 3rd ed. Philadelphia, PA: Lea & Febiger; 1950:118.
39. Tools of the trade. *NATA News*. Apr 1994:4–6.
40. Welsh J. *The Antibacterial and Wound Healing Characteristics of 5% Providone-iodine Cream and Betadine Ointment Compared to Neosporin Ointment*. Norwalk, CT: The Purdue Frederick Co; 1987:5. Purdue Frederick Monograph.
41. Winter G. Epidermal regeneration studies in the domestic pig. In: Maibach H, Rovee D, eds. *Epidermal Wound Healing*. Chicago, IL: Year Book Medical Publishers; 1972:71–112.
42. Young S, Dyson M, Hickman R, Lang S, Osborn C. Comparison of the effects of semi-occlusive polyurethane dressings and hydrocolloid dressings on dermal repair: 1. Cellular changes. *J Invest Dermatol*. 1991;97: 586–592.