CLINICAL REVIEW

Cutaneous manifestations of sports participation

David B. Pharis, MD, Craig Teller, MD, and John E. Wolf, Jr., MD *Houston, Texas*

As interest and participation in sports and recreational activities continue to increase, so do problems of the skin associated with sports participation. Primary care physicians and dermatologists may find the diagnosis of these sometimes unusual skin lesions difficult without knowledge of their association with sporting activities. Likewise, treatment of these skin lesions may be unsatisfactory without an understanding of the unique factors that contributed to the problem. This article reviews the cutaneous manifestations of sports participation. Traumatic and environmental skin injuries, skin infections, and exacerbation of preexisting dermatoses, as well as descriptions, predisposing conditions or causative agents, diagnosis, treatment, and prevention are discussed. (J Am Acad Dermatol 1997;36:448-59.)

Participation in athletic activities has become increasingly popular. As a result, dermatologists are seeing more patients for disorders caused by participation in sports. During most forms of exercise, the integument and its appendages are under constant assault by traumatic and other environmental factors that can cause injury. Physical activity also predisposes to infection of the skin and can exacerbate a preexisting dermatosis. The area of sports dermatology is rapidly expanding, and new entities are being described regularly.

This article reviews previously described cutaneous manifestations of sports participation and provides suggestions for diagnosis, prevention, and treatment.

TRAUMATIC INJURIES

Sports participation often results in injury to the skin, especially the skin of the feet. The primary cause of traumatic skin injury in the athlete is sporting equipment (Table I).

Friction blister

Friction blisters are caused by repetitive sheer stress from excessive rubbing that results in a split in the mid to lower malpighian layer of the epidermis or through the lamina lucida at the dermoepidermal junction.[1] A hot, humid environment, like that inside an athlete's shoe, increases the risk of friction blisters and is likely the reason that the feet are the most common site of blisters.[2] Measures used to prevent them include absorbent socks and foot powder. Some suggest wearing socks made from fabrics that produce less friction like acrylic,[3] or wearing two pairs of socks of different fabrics, thus allowing the interface between them to experience most of the friction.[4,5] Perhaps the most successful preventive measure is properly fitting athletic shoes.[4] Once a blister has formed, leaving the roof intact and draining its contents up to three times in the first 24 hours results in the fastest healing.[6] If the blister becomes unroofed, covering the lesion with a dressing or applying tincture of benzoin is adequate.[4]

Callus

Calluses occur on areas exposed to repeated shearing trauma and friction and frequently develop at sites of previous blisters. Most calluses are painless and many consider them to be a competitive advantage, especially in sports such as gymnastics, weight-lifting, and many racquet sports.[4] For prevention of calluses on the feet, moleskin pads or toe pads can be used. If an athlete desires the removal of a callus, paring the lesion is usually effective. Other effective measures include the use of an abrasive after softening from soaking in warm water or regular application of a salicylic acid plaster.[7]

Corn

Corns, which can be hard or soft, are also known as plantar keratoses or clavi. They are punctate hy-

From the Department of Dermatology, Baylor College of Medicine, One Baylor Plaza, Houston, TX 77030-3948.

Reprint requests: Craig Teller, MD, Department of Dermatology, Baylor College of Medicine, One Baylor Plaza, Houston, TX 77030-3948.

0190-9622/97 $5.00 + 0 **16/1/75577**

Table I. Traumatic injuries to the skin associated with sports participation

Friction blister
Callus
Corn
Ingrown toenail
Black heel (talon noir)
Black palm (tache noir)
Piezogenic papules
Tennis toe
Turf toe
Jogger's nipples
Striae distensae
Effects of anabolic steroids
Atrophic striae
Linear keloid
Green hair
Athlete's nodules
Golfer's nails
Mogul skier's palm
Pulling boat hands
Hooking thumb
Swimmer's shoulder
Runner's rump
Rower's rump
Jazz ballet bottom
Ping pong patches

perkeratoses with a deep central core usually overlying a bony prominence.[5] Corns are usually tender and may prevent an athlete from competing at maximum intensity. The most common locations are the toes or on the plantar surface of the foot overlying the distal head of the metatarsals.[8] Skin irritation secondary to an abnormal bony shape of the foot or improperly fitting shoes cause most corns.[5] Long-term use of ill-fitting shoes can lead to permanent foot deformities and exacerbation of preexisting corns. Hard corns occur on the external surface of the toes where drying occurs, whereas soft corns are interdigital and result from maceration from sweating. Treatment consists of serial paring of the keratin core after soaking for short-term relief. A more definitive therapy is the application of cantharidin and then occlusion with salicylic acid plaster. This procedure will result in blister formation under the keratosis in about 3 days. The blister can then be removed with a scalpel.[5] If the corn is removed from the plantar surface, a decrease in recurrence rate can be accomplished by placing a metatarsal or "cookie" pad under the arch of the foot. This raises the arch and redistributes the weight on the heads of the second and third metatarsals.[9] If corns recur, referral to a surgeon specializing in foot disorders is indicated to evaluate a possible underlying foot deformity.

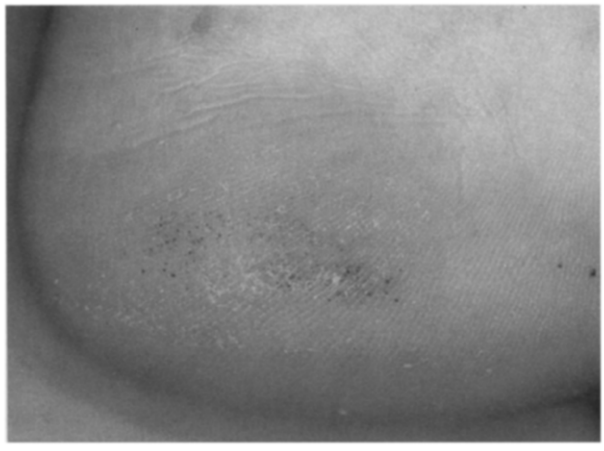

Fig. 1. Black heel (talon noir) on lateral surface of foot.

Ingrown toenails

Although common in the nonathlete, ingrown toenails deserve special attention in the athlete because of the effect they may have on performance. Most commonly, ingrown toenails are caused by ill-fitting shoes that force the lateral nail to enter the dermis, where it acts as a foreign body. This inciting force can be prevented by wearing shoes that have a wide toe box and by trimming the nail straight across. Filing down the middle of the nail may result in a dorsiflexion of the lateral nail and spontaneous clearing of the ingrown nail. Inserting cotton under the lateral nail margin is another simple treatment. Severe conditions may require partial or complete nail avulsion.

Black heel (talon noir)

Black heel is most common in young adults[10, 11] and consists of horizontally arranged petechiae at the upper edge of the heel. It is usually asymptomatic (Fig. 1). This lesion occurs in sports requiring frequent stops and starts such as tennis or basketball. It is caused by the shearing force of the epidermis sliding over the rete pegs of the papillary dermis, thereby resulting in traumatic intraepidermal and intracorneal hemorrhage.[11] A similar lesion known as black palm, or tache noir, occurs on the palms of weightlifters, gymnasts, golfers, tennis players,[12] mountain climbers,[13] and baseball players. The only significance of either lesion is possible confusion with an acral lentiginous malignant melanoma. A biopsy is rarely needed because the petechiae of

Fig. 2. Piezogenic papules on medial surface of foot visible while bearing weight.

black heel or palm are punctate and are frequently bilateral. The diagnosis can be confirmed by removing a few puncta with a scalpel, mixing with a small amount of water, and confirming the presence of blood by means of an occult blood screening test. Treatment consists primarily of rest and will result in involution of the lesion in 2 to 3 weeks. Faster results can be obtained by simply paring the lesion. A felt heel pad in the athlete's shoe may prevent recurrence.[14]

Piezogenic papules

Piezogenic papules are multiple 2 to 5 mm skin-colored painful papules on the lateral or medial surfaces of the heel. They result from herniation of subdermal fat into the dermis and are usually noticeable only upon standing.[15] (Fig. 2) Dover[4] suggests that as many as 10% to 20% of the population, especially women and children, may have piezogenic papules, but he includes both symptomatic and asymptomatic lesions in this estimate.[4] We prefer the use of the term *piezogenic papules* to indicate only painful lesions. They are most common in long distance runners and may be so painful that running must be discontinued. A heel cup in the athlete's shoe may help to reduce pain.[1]

Tennis toe

Tennis toe refers to painful subungual hemorrhages that occur most commonly in the first and second toe, sometimes bilaterally.[16] The hemorrhages are often accompanied by nail dystrophy, especially onycholysis and periungual hyperkeratosis. They may occasionally resemble malignant melanoma.[17] The use of the term *tennis toe* is somewhat misleading as it also may be caused by jogging,[17] skiing,[1] hiking, and climbing.[4] Any sport that causes repetitive slippage of the foot anteriorly against the shoe or frequent dorsiflexion of the toes in a shoe with a limited toe box can cause tennis toe.[1] Prevention and treatment include the use of properly fitting footwear allowing the toes adequate room to dorsiflex (including cutting a one inch incision in the shoe over the injured toe if necessary[18]), use of a toe pad, or use of a side-to-side strap in the shoe that prevents anterior slippage. Trimming the toenails to their shortest point in a straight tangential line that does not cause discomfort may also be preventive.[1,5] Draining of blood under the nail results in immediate pain relief but is rarely indicated because rest and soaking the foot is adequate treatment for most patients.[5] Because the pain of tennis toe is typically of short duration, unremitting pain may be evidence of a fracture and should be investigated with a radiograph.[19]

Turf toe

Athletes who play on artificial turf, such as football and soccer players, can experience a unique injury known as turf toe. It is caused by acute tendinitis of the flexor and extensor tendons of the great toe resulting from attempts to stop or "cut" quickly on a surface with little give.[20,21] The first toe is painful, red, and swollen. This condition might be confused with acute gouty arthritis or acute paronychia if a history of sports activity is not elicited. The only effective treatment is rest and the use of footwear designed for use on artificial turf.[4]

Jogger's nipples

Jogger's nipples are painful, fissured, eroded nipples that occasionally bleed. This condition occurs immediately after long distance running in women who do not wear a bra and men who wear hard fiber, such as nylon, shirts.[22] The lesions result from constant irritation and friction from hard fabrics against the unprotected nipple and areola. Preventive measures for women include wearing an athletic bra and soft fiber shirts such as semisynthetics and silk. Men can prevent jogger's nipples by simply running without a shirt. Others suggest the use of adhesive bandages or the application of petroleum jelly to the nipples immediately before running.[23]

Striae distensae

In as many as 70% of adolescent girls and 40% of adolescent boys, especially those who lift weights regularly, striae develop. The most common type, striae distensae, is caused by rupture of elastic fibers into the reticular dermis, probably as a result of a corticosteroid effect on elastic tissue.[24, 25] Striae are arranged perpendicular to lines of skin tension and occur most commonly over the anterior shoulders, lower back, and thighs.[11] Although many over-the-counter products claim success in treating striae, there is little evidence that they are effective. A nonrandom, uncontrolled study of the use of 0.1% tretinoin cream showed significant improvement in 15 of 16 patients who completed the study.[26] However, a later study with 0.025% tretinoin cream failed to show any significant improvement.[27]

Effects of anabolic steroids

The presence of atrophic striae, especially in combination with severe acne, a receding hair line, or hypertrichosis should suggest the use of anabolic steroids by the patient.[28] The use of performance-enhancing drugs in athletes is common, especially in weightlifters.[29, 30] Regular anabolic steroid intake increases the content of cholesterol and free fatty acids in skin surface lipids and increases the size of the sebaceous gland. These changes, in combination with an increased population of *Propionibacterium acnes*, can result in severe nodulocystic acne.[28] There have also been reports of linear keloid formation in adolescent male weight lifters 8 to 14 months after the use of anabolic steroids. This skin damage is caused by the rapid development of underlying muscle groups, especially the deltoids.[31] Nodulocystic acne will resolve after the discontinuation of steroids, but frequently not without scarring. The other skin manifestations of anabolic steroid use, such as alopecia, striae, and keloids, are permanent.

Green hair

Those who swim on a regular basis, especially athletes with fair skin and hair, may experience a green discoloration of their hair.[32-34] Green hair is caused by the uptake of copper by the hair shaft.[35] The copper may be leached from old copper pipes[36] or may result from the use of a copper-containing aligicide.[35] Prevention includes washing the hair immediately after swimming and maintaining the pH of the pool water between 7.4 and 7.6. Treatment includes the use of copper-chelating shampoos such as Ultraswim or Metalex for 30 minutes or 3% hydrogen peroxide soaks for 3 hours.[36]

Athlete's nodules

Athlete's nodules are 0.5 to 4.0 cm asymptomatic flesh-colored nodules on the dorsum of the feet, knees, or knuckles as a result of recurrent trauma and friction.[37, 38] These are frequent in surfers, boxers, football players, and even marble players and may be accompanied by hyperkeratosis of the overlying epidermis.[39] Cohen, Eliezri, and Silvers[37] described athlete's nodules on the dorsum of a middle-aged man's feet as a result of tight-fitting athletic shoes he wore as a younger athlete. Microscopic examination revealed an increase in collagen bundles in the reticular dermis resembling connective tissue nevi of the collagen type. They speculated that these collagenomas, or athlete's nodules, were the result of recurrent trauma to the dorsum of the foot from athletic shoes. The same process could occur in other athletes as a result of trauma from surfboards, boxing gloves, or pressing the knuckles firmly against concrete in an intense game of marbles.[38]

Some authors resist the naming of all clinically similar nodules found in athletes as athlete's nodules and believe that many may be formed by different pathophysiologic processes. An example is the surfer's nodule, ulcer, or knot that appears on the anterior tibia, knee, or dorsum of the foot in surfers. Five different types have been described based on clinical appearance and underlying pathology.[37] The lesions range from some similar to those described by Cohen,[37] to reactive inflammation in response to a foreign body such as a grain of sand,[41] to lesions with chipping or spurring of the underlying bone. The other two types described are an infrapatellar bursal cyst and a lesion that histopathologically resembled a ganglion cyst on the dorsum of the foot.[37]

Basler and Jacobs[42] described a nodular lesion consisting of what they called "pseudo-bursae" on the dorsum of the foot that appears and disappears in weeks. They observed this in a woman who wore tight-fitting athletic shoes. They referred to the condition as "Nike nodules." Similar lesions have also been described in hockey players and are referred to as "skate bites." Treatment of all of these mostly asymptomatic lesions consists of either watchful waiting or surgical removal. Recurrence rates after surgical removal vary widely. There have been reports of successful treatment of collagenomas with

Journal of the American Academy of Dermatology
March 1997

Table II. Environmental injuries to the skin associated with sports participation

Frostnip
Frostbite
Sunburn
Phototoxicity
Damage from long-term sun exposure
Photoaging
Actinic keratosis
Basal cell carcinoma
Squamous cell carcinoma
Malignant melanoma
Allergic contact dermatitis
Xerosis

intralesional steroids, so a therapeutic trial might be considered before excision.[37]

Several other miscellaneous skin injuries have been described that are of little clinical consequence and have minimal effect on performance.

Golfer's nails

Golfer's nails are splinter hemorrhages or linear dark streaks of the fingernails that occur in golfers who grip the shaft of the club too tightly. This excess pressure produces hemorrhage in the nail plate and underlying vascular bed. The blood in the dermis is reabsorbed and that attached to the nail plate grows out with the nail. Prevention involves learning the proper way to grip the club.[43]

Mogul skier's palm

Mogul skier's palm consists of hypothenar ecchymoses on the volar aspect of the palm resulting from the repetitive planting of ski poles. The ecchymoses may have a golden hue because of the presence of hemosiderin from past hemorrhages. They usually clear after the end of the skiing season.[44]

Pulling boat hands

Pulling boat hands occur in crew team members and consist of subcutaneous vascular injuries combined with epidermal blister formation. They are thought to be the result of mechanical injury in combination with cold exposure. Calluses soon form in place of the acute lesions and may serve as a competitive advantage.[45]

Hooking thumb

Hooking thumb occurs exclusively in competitive weight lifters. It consists of abrasions, hematomas, bullae, denudation, calluses, and subungual hematomas on the distal third of the thumb. Hooking is a method of gripping the weight bar with the thumb under the index and middle finger to provide a better grip for lifts like the clean and jerk and the squat thrust. Hooking thumb may hamper performance because of pain but resolves after discontinuation of the hooking maneuver.[46]

Swimmer's shoulder

Swimmer's shoulder is an erythematous plaque on the shoulder resulting from irritation from an unshaven face during freestyle swimming. Shaving before swimming prevents the problem.[47]

Runner's rump

Runner's rump consists of small ecchymoses on the superior portion of the gluteal cleft in long distance runners. It is thought to result from constant friction at this site and is of no consequence.[1]

Rower's rump

Rower's rump is a frictional form of lichen simplex chronicus resulting from rowing while sitting on an unpadded seat for long periods of time. Treatment consists of substituting a padded seat and the use of a flourinated steroid cream for the lichenified lesion.[48]

Jazz ballet bottom

Jazz ballet bottom is a natal cleft abscess in young women in the absence of a pilonidal cyst. It is associated with local trauma from ballet exercises.[49]

Ping pong patches

In the tradition of alliteration in describing cutaneous sports injuries, ping pong patches are erythematous macules 2 to 3 cm in diameter that are caused by the high-velocity impact of the ball on the forearms and dorsal aspect of the hands.[50]

ENVIRONMENTAL INJURIES

Many of the most popular sporting activities are enjoyed outdoors. This means the athlete is at times exposed to extremes of temperature, wind, and the damaging effects of the sun. Sporting equipment may also cause an allergic or irritant reaction (Table II).

Frostnip

Of the cold-induced injuries to the skin, frostnip, a milder form of frostbite with only superficial freezing of the skin and subcutaneous tissues, is the most common. Winter athletes, as well as spectators, must be aware of the dangers of cold exposure. Exposed skin of the face and neck is most susceptible to frostnip.[51] However, unusual areas can be affected as reported in the case of penile frostnip in a long distance runner.[52] The skin with frostnip is initially gray white and loses sensation as freezing progresses. After warming, the skin becomes red, swollen, and often stings. Blisters may form in the first 24 to 36 hours. Crusting then occurs and usually clears by 2 weeks without scarring. The previously frostnipped skin may throb or burn for an additional 2 weeks and may continue to be cold sensitive for months.[51]

Frostbite

Frostbite implies the freezing of the skin, subcutaneous tissue, muscle, and even bone.[51] Both exposed and unexposed areas are susceptible but the anterior neck is most commonly affected.[41] The combination of exercise, cold, wind, and water from either perspiration or immersion can reduce insulation of clothing to 10% of its normal value.[53] Metal is an efficient heat conductor and can cause frostbite instantaneously by direct contact with exposed, especially moist skin.[4] Athletes competing at higher altitudes are at increased risk for frostbite because of the decrease in ambient oxygen tension. This, combined with cutaneous vasoconstriction to maintain core body temperature in a cold environment, exacerbates the oxygen starvation of cutaneous tissues.[54] Metabolism of the cell stops at –2° C.[55] At this temperature, proteins and enzymes are destroyed and ice crystals form in the extracellular space. The extracellular ice crystals draw water from the cell and this dehydration further contributes to cell damage.[1] Initial symptoms of frostbite include burning and pain that quickly progress to complete anesthesia with continued exposure. Swelling, blistering, and discomfort of the affected tissue is often delayed up to a week. The resulting necrosis and sloughing of tissue can last several weeks and reepitheliazation can take months. Severe throbbing and shooting pain may be experienced in the affected area for 2 to 8 weeks after the initial injury. Pruritus and increased perspiration may persist for 6 months.[51] Treatment of frostbite consists of rapid warming after there is no further risk of cold exposure. A repeat freeze in the same tissue will greatly increase necrosis, analogous to the repeat freeze used in cryotherapy. Use of a water bath at 38° to 44° C for 20 minutes appears to be the best method of warming.[51, 56] Exposure to an open fire or rubbing the skin during warming may increase tissue damage.[4]

Fig. 3. Phototoxic eruption from dicloxacillin. Notice hemorrhagic blisters on dorsolateral surface of hand and sparing of skin covered by watch band.

Prevention of both frostnip and frostbite is the best treatment. Wearing multiple layers of thin clothing allows trapping of heat between the layers and absorption of excess perspiration.[51, 53] The application of petroleum jelly or zinc oxide to exposed areas, or simply waiting to shower and shave until after cold exposure to take advantage of the natural insulatory effect of sebum, will help prevent cold-induced skin damage.[1] Having a partner test skin sensation periodically while exposed may also alert one to sensory loss and impending frostnip or frostbite.

Sun-induced skin injury

Many popular sports and recreational activities are performed out-of-doors, often with minimal protective clothing. The most commonly encountered injury is sunburn.[11, 57] Drug-induced photosensitivity is another problem[58] (Fig. 3). The long-term effects of chronic sun exposure and repeated sunburns may be accelerated in outdoor athletes.[59] Athletes should be advised about proper sun protection.[60, 61] Skiers, for example, should protect areas exposed to reflected sunlight from the snow (often striking areas of skin normally unexposed) and be informed that the intensity of UV light is 20% greater at 5000 feet than at sea level.[62]

Journal of the American Academy of Dermatology
March 1997

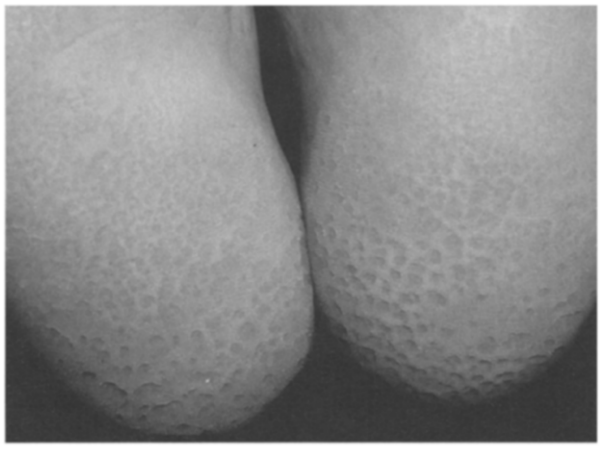

Fig. 4. Pitted keratolysis of both heels.

Allergic contact dermatitis

The athlete comes in contact with many substances that may cause allergic contact dermatitis. The most common offending agents are adhesives from benzoin and rubber-backed athletic tapes.[63, 64] Other allergens include leather sporting equipment,[11] antioxidants and accelerators in rubber in bathing caps, swimming goggles and masks ("mask burn"), nose clips, and ear plugs,[65] athletic shoes that leach chromate and rubber additives,[12, 66] fiberglass in hockey sticks,[19] and topical creams containing salicylates, antibiotics, antiseptics, or iodine.[11] The use of drying powder and absorbent socks may prevent the sweat-induced leaching of allergens from shoes.[19] Sporting good manufacturers can provide products made from hypoallergenic materials like silicone rubber swimming masks and wet suits without the allergenic nylon lining. These products may not be sold to the general public but can be obtained by contacting the manufacturer's customer service department directly.[65]

Xerosis

Xerosis is the most common cause of body pruritus in athletes. It is caused by frequent showering and the exposure of the skin to the drying effects of the elements. The athlete will complain of pruritus at the start of exercise as the body begins to perspire. The application of an emollient lotion once daily, especially before exercise, may eliminate the pruritus.

INFECTIONS

Athletes are exposed to many common as well as unusual infectious agents because of the close interpersonal contact required in sports such as wrestling and from the warm, moist environment of sports venues and locker rooms (Table III).

Table III. Infections of the skin associated with sports participation

Pitted keratolysis
Swimmer's ear
Herpes gladiatorum
Impetigo
Tinea corporis gladiatorum
Tinea pedis
Seabather's eruption
Swimmer's itch

Pitted keratolysis

Pitted keratolysis is an infection of the feet caused by *Corynebacterium* species. The infection is characterized by 1 to 3 mm discrete and coalescing craterlike pits on the plantar surface of the feet and toes, especially the weight-bearing areas (Fig. 4). The affected area also frequently has a dirty appearance and may have a foul odor but is often asymptomatic. The infection usually clears with elimination of moisture. Persistent infection will clear with treatment with topical or oral erythromycin, topical clindamycin, or topical 5% formalin. Preventive measures include maintaining a dry environment in the shoe with foot powder and absorbent socks, and the use of 20% aluminum chloride solution (Drysol) as a drying agent.[11]

Swimmer's ear

Swimmer's ear, or otitis externa, is a mixed infection and inflammation caused by gram-negative organisms such as *Pseudomonas*.[67, 68] Water exposure in the external canal causes maceration of epithelial tissue, and depletion of cerumen and the relatively acidic pH that acts as a bacteriostatic and fungistatic barrier. Minor trauma from ear cleaning with cotton swabs may increase the maceration of epithelium and introduce bacteria into the inflamed tissue.[69] Treatment includes cessation of swimming until the problem resolves, cleaning of the canal by gentle suction, and then insertion of a wick to serve as a conduit for astringents and for an antibiotic/antiinflammatory solution such as colistin neomycin/hydrocortisone, polymyxin propylene glycol hydrocortisone, or polymyxin neomycin hydrocortisone drops. The wick may also serve as a debriding ve-

hicle. Useful astringent solutions include 20% silver nitrate, Burow's solution, or both.[68, 70-75] Severe cases, especially in diabetic patients, may need to be treated with oral antibiotics. Oral steroids may provide relief when inflammation is severe. The use of 2% acetic acid in propylene glycol has been shown to decrease the incidence by simulating the natural environment of the external canal.[76, 77]

Herpes gladiatorum

Wrestlers experience a type of herpes simplex virus (HSV) infection known as herpes gladiatorum. This is a cutaneous HSV-1 infection resulting from direct skin to skin contact between an infected host with active lesions and a susceptible opponent usually with open abrasions. The head and neck area, especially the right side of the face, is the most commonly affected area because of the "lock-up" position that wrestlers assume during competition.[78] The lesions appear 1 to 2 weeks after exposure at the site of a previous abrasion and are characterized by the typical grouped vesicles on an erythematous plaque. The vesicles may evolve into pustules and become eroded, enlarging to become crusted or moist ulcerations.[79] Associated symptoms may include regional lymphadenopathy, fever, chills, sore throat, and headache.[78] Treatment includes stopping all affected athletes from practicing or competing, and administration of acyclovir orally (200 mg five times a day for 7 days) or intravenously (5 mg/kg every 8 hours for 5 days) depending on the severity.[78, 79] Topical acyclovir is apparently no more effective than drying agents like alcohol.[58] If the diagnosis is in doubt, an HSV culture may be helpful to ensure proper treatment and counseling for public health purposes.[78, 80] Culture fluid should be taken from an intact vesicle before acyclovir is started or from the base of an erosion early in the course of treatment.[79] Prevention of infection by careful attention to hygiene, showering before and after events, and inspection of all participants before competition are the most effective measures in preventing transmission of the HSV-1 virus.[81]

Other infections that wrestlers are predisposed to include staphylococcal and streptococcal impetigo. Both are transmitted in a similar manner to herpes gladiatorum and may superinfect herpes simplex. Oral erythromycin or cloxacillin for 10 days, topical agents like mupirocin 2% ointment, and cessation of wrestling until healing has occurred are effective treatments.[58] An outbreak of tinea corporis gladiatorum in a high school wrestling team has been described[82] (Fig. 5). Suggested control measures include careful inspection of athletes before participation, and antifungal medications. Griseofulvin should be used for 1 month for persons with two or more lesions or with any facial lesions. Topical ketoconazole or econazole should be used for 1 month by all others.[82]

Fig. 5. Tinea corporis gladiatorum in the distribution of skin occlusion with a wrestling singlet.

Tinea pedis

Probably the most common infection of the skin in both the serious and recreational athlete is tinea pedis. Predisposing factors include the warm, moist environment of the athletic shoe, the increased environmental CO_2 level that produces infective hyphae, and the occlusive effect of the sock, sweat, and shoe combination.[83-85] The most common sources of infection are the shower, locker room, and pool floors that harbor the dermatophytes.[86] There are three types of tinea pedis: moccasin, pustular-midsole, and interdigital. The moccasin form is chronic and usually asymptomatic. The pustular-midsole variety is acute and often pruritic. The interdigital type is the most common and can be of a mixed bacterial and fungal cause in macerated interdigital epithelium.[58, 84] It is frequently pruritic and often painful.[58]

Effective antifungal agents include the fungistatic imidazoles[87-92] and the fungicidal allyamines such as naftifine and terbinafine. Drying powder is useful in both prophylaxis and treatment by reducing friction and maceration and by absorbing excess perspiration. Reinfection can be reduced with the use of terbinafine because of its prolonged binding in the stratum corneum.[93, 94] Prophylactic tolnaftate pow-

Table IV. Preexisting dermatoses that may be exacerbated by sports participation

Acne mechanica
Exercise-induced urticaria/anaphylaxis
Cholinergic urticaria
Solar urticaria
Essential cold urticaria

der may reduce the incidence of reinfection, and topical antifungals may be used as prophylaxis in high-risk persons.[87] In the interdigital form, use of an astringent like 20% aluminum chloride to dry macerated toe webbings aids in killing both bacteria and fungi and helps provide symptomatic relief.[95]

Seabather's eruption

Athletes who participate in salt water sports along the Gulf Coast of Florida, off the coast of Long Island, and in many parts of the Caribbean may experience an eruption in a bathing suit distribution known as seabather's eruption. Seabather's eruption is a pruritic, urticarial, erythematous, maculopapular eruption that progresses to itchy papules, wheals, and pustules that resemble insect bites before developing into eschars. Associated symptoms may include chills, fever, swelling, headache, nausea, and vomiting, especially in children. The eruption may take 1 to 2 days to develop and will last for 2 to 14 days without intervention. The causative agents vary depending on the coastal region, but all appear to be stinging planula larvae of the phylum Cnidaria, which includes jellyfish, man-of-war, sea anemone, and fire coral.[96, 97] Two recently documented outbreaks involved the jellyfish Linuche unguicula off the coast of Florida[96] and the anemone Edwardsiella lineata off the coast of Long Island, New York.[97] Prevention includes the notification of public health officials who will need to close the offending beach area to all swimmers, thorough cleaning of swimwear, and showering after swimming in potentially infected waters. Treatment, although not all patients respond, includes cool compresses, antihistamines, and topical steroids. Severe cases may require oral corticosteroids for control of symptoms.[96] Successful treatment of severe eruptions has also been reported with oral thiabendazole, 1.5 gm twice a day for 2 days.[98]

Swimmer's itch

A similar eruption that develops after swimming, usually in fresh water of the northern United States and Canada, is known as swimmer's itch. It is caused by an inflammatory reaction to the cercarial form of nonhuman schistosomes or flukes. The itching results from an acute urticarial reaction to the organism. The acute reaction clears, and then a papular eruption occurs, similar to the delayed hypersensitivity reaction. A differentiating feature of swimmer's itch from seabather's eruption is that the former occurs on exposed areas whereas the latter is usually on skin covered by a bathing suit. Treatment of swimmer's itch is symptomatic. The lesions regress spontaneously and completely clear in 1 to 2 weeks.[99]

EXACERBATION OF PREEXISTING DERMATOSES

Many skin conditions in an athlete may worsen with physical activity because of an increase in perspiration, heat, and irritation from equipment (Table IV).

Acne mechanica

Acne vulgaris is often a preexisting condition in a form of acne in athletes known as acne mechanica. However, there have been reports of patients without a prior history of acne vulgaris who have a severe case of acne mechanica.[100] Acne that suddenly flares in severity and is resistant to treatment, or the development of acne conglobata, should raise the suspicion of anabolic steroid use.[28] Acne mechanica is a papulopustular eruption caused by pressure, occlusion, friction, and heat.[101, 102] The most common location is the chin and other friction-prone sites such as the occiput, and shoulders of athletes like football and hockey players who wear helmets and shoulder pads. Women who wear occlusive leotards of synthetic fabrics for dance aerobics or other forms of exercise may also be affected.[1] The stress on the skin appears to be the key element in acne mechanica rather than the inflammation and hyperkeratinization of the pilosebaceous unit seen in acne vulgaris. Severe forms of acne mechanica, especially in dark-skinned athletes, can progress to acne keloidalis.[1, 103] Both acne mechanica and keloidalis respond suboptimally to traditional acne therapy. Both usually clear spontaneously at season's end with the possible need for intralesional triamcinolone for the keloidalis form.[4] Prevention includes the use of a clean absorbent cotton T-shirt underneath any equipment. In addition, rigorous cleaning with abrasives

and the use of an astringent with a keratolytic agent after each workout is probably the most effective preventive measure. Effective astringent/keratolytic agents include 3% salicylate and 8% resorcinol in 70% ethanol (0.5% clindamycin can be added if needed).[104] If oral isotretinoin is prescribed, the side effects of muscle and joint pain, lethargy, and decreased energy may inhibit performance in competitive athletes.[41]

Exercise-induced anaphylaxis

Many forms of urticaria may be induced or exacerbated by athletic activity. The most severe form is exercise-induced anaphylaxis. This occurs most commonly in atopic persons or persons with a food allergy who are conditioned athletes. It develops a short time after the start of exercise with cutaneous warmth, progressing to pruritus, erythema, urticaria, angioedema, respiratory distress, and possible vascular collapse.[105-107] Two underlying mechanisms have been elucidated. One appears to be mediated by activation of the complement pathway; the other is histamine-mediated and may be a severe form of cholinergic urticaria.[65] Treatment of an acute episode includes those measures used for any anaphylactic episode (i.e., epinephrine and antihistamines). Preventive measures include never exercising alone, exercise modification, and taking a long-acting nonsedating antihistamine 1 hour before exercise or 40 mg of prednisone 12 hours before exercise if the antihistamine is ineffective.[65] An epinephrine injection (EpiPen) should be on hand during exercise to abort the development of a severe episode.

Cholinergic urticaria

Cholinergic urticaria consists of pinpoint erythematous wheals surrounded by an erythematous flare after exposure to heat or during exercise, usually after the start of sweating. Cholinergic urticaria is often refractory to treatment but may respond to antihistamines, especially hydroxyzine. If symptoms are not controlled with antihistamines, avoiding heat and exercise may be the only means of obtaining relief.[11]

Solar urticaria

Solar urticaria develops within minutes after exposure to sunlight or artificial UV light and usually clears 1 hour after exposure. Itching and burning are the first symptoms. Erythema is next, and then wheals occur. The reaction is less common in chronically exposed areas such as the face and hands than in normally unexposed areas such as the trunk. All wavelengths of UV light (290 to 500 nm) can cause solar urticaria, necessitating complete sun block of all areas during sun exposure. Desensitization therapy with specific wavelengths is effective in maintaining symptom-free periods, as is PUVA therapy.[108]

Cold urticaria

Essential cold urticaria can occur in winter athletes and swimmers. Small and large urticarial plaques can be generalized or confined to the area exposed to the cold. Diagnosis can be confirmed by application of ice to the skin for 5 minutes. An urticarial plaque or wheals develop immediately on warming.[11] If the ice cube test is negative and the clinical suspicion remains high, retesting with cold water of different temperatures should be performed.[109] Treatment includes avoiding cold, wearing protective clothing, and using antihistamines.[110]

REFERENCES

1. Basler RSW. Skin lesions related to sports activity. Prim Care 1983;10:479-94.
2. Joy RJT. The effect of prior heat acclimatization upon military performance in hot climates. Mil Med 1964;129:51-7.
3. Herring KM, Richie DH. Friction blisters and sock fiber composition. A double blind controlled study. J Am Podiatr Med Assoc 1990;80:63-71.
4. Dover JS. Sports dermatology. In: Fitzpatrick TB, Eisen AZ, Wolf K, et al, editors. Dermatology in general medicine. New York: McGraw Hill, 1991:1617-23.
5. Basler RSW. Skin injuries in sports medicine. J Am Acad Dermatol 1989;21:1257-62.
6. Cortese TA, Fukuyama K, Epstein WL, et al. Treatment of friction blisters. Arch Dermatol 1968;97:717-21.
7. Montgomery RM. Tennis and its skin problems. Cutis 1977;19:480-2.
8. Bart B. Skin problems in athletes. Minn Med 1983;66: 239-41.
9. Sheard C. Simple management of plantar clavi. Cutis 1992;50:138.
10. Wilkinson DS. Black heel. Cutis 1977;20:393-6.
11. Houston SD, Knox JM. Skin problems related to sports and recreational activities. Cutis 1977;19:487-91.
12. Resnick SS, Lewis LA, Cohen BH. The athlete's foot. Cutis 1977;20:351-5.
13. Izumi AK. Pigmented palmar petechiae (black palm). Arch Dermatol 1974;109:261.
14. Ayers S Jr, Mihan R. Calcaneal petechiae [letter]. Arch Dermatol 1972;106:262.
15. Shelley WB, Raunsley AM. Painful feet due to herniation of fat. JAMA 1968;205:308-9.
16. Gibbs R. Tennis toe. Arch Dermatol 1973;107:918.
17. Sher RK. Jogger's toe. Int J Dermatol 1978;17:719-20.
18. Schuster RO. Foot types and the influence of environment on the foot of the long distance runner. Ann N Y Acad Sci 1977;301:881-7.

19. Levine N. Dermatologic aspects of sports medicine. J Am Acad Dermatol 1980;3:415-24.
20. Doller J, Strother S. Turf toe. J Am Podiatr Assoc 1978;68:512-4.
21. Basler RSW. Dermatologic aspects of sports participation. Curr Concepts Skin Dis 1985;6:15-9.
22. Levit F. Jogger's nipples [letter]. N Engl J Med 1977; 297:1127.
23. Brazin SA. Dermatologic hazards of long-distance running. J Am Mil Dermatol 1979;5:8-9.
24. Sisson WR. Colored striae in adolescent children. J Pediatr 1954;45:520-30.
25. Shelley WB, Cohen W. Striae migrans [letter]. Arch Dermatol 1964;90:193.
26. Elson ML. Treatment of striae distensae with topical tretinoin. J Dermatol Surg Oncol 1990;16:267-70.
27. Prabanich S, Simpson FG, Held B, et al. Low dose tretinoin does not improve striae distensae: a double-blind, placebo controlled study. Cutis 1994;54:121-4.
28. Scott MJ III, Scott AM. Effects of anabolic androgenic steroids on the pilosebaceous unit. Cutis 1992;50:113-6.
29. Hallagan JB, Hallagan LF, Snyder MB. Anabolic-androgenic steroid use by athletes. N Engl J Med 1989; 321:1042-5.
30. Buckley WE, Yesalis CE, Friedl HK. Estimated prevalence of anabolic steroid use among male high school seniors. JAMA 1988;260:3441-5.
31. Scott MJ Jr, Scott MJ III, Scott AM. Linear keloids resulting from abuse of anabolic androgenic steroid drugs. Cutis 1994;53:41-3.
32. Holmes LB, Goldsmith LA. The man with green hair [letter]. N Engl J Med 1974;291:1037.
33. Cooper R, Goodman J. Green hair. N Engl J Med 1975; 292:483-4.
34. Bhat GR, Lukenbach ER, Kennedy RK, et al. The green hair problem: a preliminary investigation. J Soc Cosmet Chem 1979;30:1-8.
35. Lampe RM, Henderson AL, Hansen GH. Green hair [letter]. JAMA 1977;237:2092.
36. Goldschmidt H. Green hair [letter]. Arch Dermatol 1979; 115:1288.
37. Cohen PR, Eliezri YD, Silvers DN. Athlete's nodules: sports-related connective tissue nevi of the collagen type (collagenomas). Cutis 1992;50:131-5.
38. Cohen PR, Eliezri YD, Silvers DN. Athlete's nodules [letter]. J Am Acad Dermatol 1991;24:317-8.
39. Erickson JG, von Gemmingen GR. Surfer's nodules and other complications of surfboarding. JAMA 1977; 167:134-6.
40. Muller SA. Dermatologic disorders in athletes. J Ky Med Assoc 1976;74:225-8.
41. Basler RSW. Sports-related skin injuries. In: Callen JP, Dahl MV, Golitz LE, et al, editors. Advances in dermatology; vol 4. Chicago: Year Book Medical Publishers, 1989:29-50.
42. Basler RSW, Jacobs SI. Athlete's nodules [letter reply]. J Am Acad Dermatol 1991;24:318.
43. Ryan AM, Goldsmith LA. Golfer's nails. Arch Dermatol 1995;131:857-8.
44. Swinehart JM. Mogul skier's palm: traumatic hypothenar ecchymosis. Cutis 1992;50:117.
45. Toback AC, Korson R, Krusinski PA. Pulling boat hands: a unique dermatosis from coastal New England. J Am Acad Dermatol 1985;12:649-55.
46. Scott MJ Jr, Scott NI, Scott LM. Dermatologic stigmata in sports: weightlifting. Cutis 1992;50:141-5.
47. Koehn GG. Skin injuries in sports medicine [letter]. J Am Acad Dermatol 1991;24:152.
48. Tomecki KJ, Mikesell JF. Rower's rump [letter]. J Am Acad Dermatol 1987;16:890-1.
49. Radford PJ, Greatorex RA. Jazz ballet bottom. Br Med J 1987;295:1173.
50. Scott MJ Jr, Scott MJ III. Ping pong patches. Cutis 1989; 43:363-4.
51. Washburn B. Frostbite. N Engl J Med 1962;266:974-89.
52. Hershkowitz M. Penile frostbite, an unforeseen hazard of jogging [letter]. N Engl J Med 1977;292:178.
53. Pugh LG. Clothing insulation and accidental hypothermia in youth. Nature 1966;209:1281-6.
54. Pugh LG. Cold stress and muscular exercise, with special reference to accidental hypothermia. Br Med J 1967; 1:333-7.
55. D'Ambrosia RD. Cold injuries encountered in a winter resort. Cutis 1977;20:365-8.
56. Anonymous. Treatment of frostbite. Med Lett Drugs Ther 1976;18:105-6.
57. Fitzpatrick TB, Johnson AJ, Polano MK, et al. Color atlas and synopsis of clinical dermatology: common and serious diseases. New York: McGraw-Hill, 1992:210-3.
58. Conklin RJ. Common cutaneous disorders in athletes. Sports Med 1990;9:100-19.
59. Williams H, Brett J, du Vivier A. Cyclist's melanoma. J R Coll Physicians Lond 1989;23:114-5.
60. Menter JM, Hollins TD, Sayre RM, et al. Protection against UV photocarcinogenesis by fabric materials. J Am Acad Dermatol 1994;31:711-6.
61. Sayre RM, Marlowe E, Agin PP, et al. Performance of six sunscreen formulations on human skin. Arch Dermatol 1979;115:46-9.
62. Anonymous. Sunscreens. Med Lett Drugs Ther 1979; 21:46-8.
63. Freeman MJ, Bergfeld WF. Skin diseases of football and wrestling participants. Cutis 1977;20:333-41.
64. Cullen SI, Tonkin A, May FE. Allergic contact dermatitis to compound tincture of benzoin spray. J Trauma 1974;14:348-50.
65. Fisher AA. Sports related allergic dermatology. Cutis 1992;50:95-6.
66. Fisher AA. Contact dermatitis. Philadelphia: Lea & Febiger, 1973:158-9.
67. Hoadley AW, Knight DE. External otitis among swimmers and nonswimmers. Arch Environ Health 1975;30:445-8.
68. Cassisi N, Davidson T, Cohn A, et al. Diffuse otitis externa: clinical and microbiologic findings in the course of a multinuclear study on a new otic solution. Ann Otol Rhinol Laryngol 1977;86(suppl 39):1-16.
69. Bell DN. Otitis externa: a common self inflicted condition. Postgrad Med 1985;78(3):101-4.
70. McDonald TJ, Neel HB III. External otitis. Postgrad Med 1975;57:95-7.
71. Taylor JS. Otitis externa: treatment using a new expandable wick. South Med J 1975;68:698.
72. Smothers CR. Chemical treatment of external otitis. South Med J 1977;70:543-5.
73. Freedman R. Versus placebo in treatment of acute otitis externa. Ear Nose Throat J 1978;57:198-204.
74. Walike JW. Management of acute ear infections. Otolaryngol Clin North Am 1979;12:439-45.
75. Anonymous. Swimmer's ear [editorial]. Br Med J 1974; 2:213.
76. Garrity JD, Halliday TC. Prevention of swimmer's ear by

simple prophylactic regimen. Curr Ther Res 1974;16: 437-40.
77. Hutchison SL, Wright DN. Prophylaxis of predisposed otitis externa. Ann Octol 1975;84:16-21.
78. Becker TM. Herpes gladiatorum: a growing problem in sports medicine. Cutis 1992;50:150-2.
79. Fitzpatrick TB, Johnson AJ, Polano MK, et al, editors. Color atlas and synopsis of clinical dermatology: common and serious diseases. New York: McGraw-Hill, 1992:70-3.
80. Porter PS, Baughman RD. Epidemiology of herpes simplex among wrestlers. JAMA 1965;1994:150-2.
81. Dyke LM, Merikangas VR, Bruton OC, et al. Skin infection in wrestlers due to herpes simplex virus. JAMA 1965; 194:153-4.
82. Beller M, Gessner BD. An outbreak of tinea corporis gladiatorum on a high school wrestling team. J Am Acad Dermatol 1994;31:197-201.
83. Allen AM, King RD. Occlusion, carbon dioxide and fungal skin infections. Lancet 1978;1:360-2.
84. Leyden JJ, Kligman AM. Interdigital athlete's foot. Arch Dermatol 1978;114:1466-72.
85. Jones HE, Reinhardt JH, Renaldi MG. Immunologic susceptibility to chronic dermatophytosis. Arch Dermatol 1973;110:213-20.
86. Ajello L, Getz ME. Recovery of dermatophytes from shoes and shower stalls. J Invest Dermatol 1954;22:17-24.
87. Gentles JC, Jones GR, Roberts DT. Efficacy of miconazole in the topical treatment of tinea pedis in sportsmen. Br J Dermatol 1975;93:79-84.
88. Clayton YM, Knight AG. A clinical double-blind trial of topical miconazole and clotrimazole against superficial fungal infections and erythrasma. Clin Exp Dermatol 1976;1:225-32.
89. Ongley RC. Efficacy of topical miconazole treatment of tinea pedis. Can Med Assoc J 1978;119:353-4.
90. Clayton YM, Conner BL. Comparison of clotrimazole cream, Whitfield's ointment and nystatin ointment for the topical treatment of ringworm infections, pityriasis versicolor, erythrasma and candidiasis. Br J Dermatol 1973; 89:297-303.
91. Herman HW. Clinical efficacy studies of haloprogin, a new topical antimicrobial agent. Arch Dermatol 1972;106: 839-42.
92. Katz R, Cahn B. Haloprogin therapy for dermatophyte infections. Arch Dermatol 1972;106:837-8.
93. English MP. Some controversial aspects of tinea pedis. Br J Dermatol 1962;74:50-6.
94. Broughton RH. Reinfection from socks and shoes in tinea pedis. Br J Dermatol 1955;67:249-54.
95. Leyden JJ, Kligman AM. Aluminum chloride in the treatment of symptomatic athlete's foot. Arch Dermatol 1975;111:1004-10.
96. Wong DE, Meinking TL, Rosen LB, et al. Seabather's eruption. J Am Acad Dermatol 1994;30:399-406.
97. Freudenthal AR, Joseph PR. Seabather's eruption. N Engl J Med 1993;329:542-4.
98. Burnett J. Sea bather's eruption. Cutis 1992;50:98.
99. Fitzpatrick TB, Eisen AZ, Wolf K, et al. editors. Dermatology in general medicine. New York: McGraw Hill, 1991:2779.
100. Farber GA, Burks JW, Hegre AM, et al. Football acne: an acneiform eruption. Cutis 1977;20:356-60.
101. Mills OH, Kligman A. Acne mechanica. Arch Dermatol 1975;1:481-3.
102. Schwartz WF. Unilateral acne: the case for thermogenic aggravation, and a clue to basic acne therapy. Cutis 1981;27:530-1.
103. Shelley WB. Consultations in dermatology II. Philadelphia: WB Saunders, 1974:110-3.
104. Baker RSW. Acne mechanica in athletes. Cutis 1992; 50:125-8.
105. Kaplan AP. Exercise-induced hives. J Allergy Clin Immunol 1984;73:704-7.
106. Sheffer AL, Austin KF. Exercise-induced anaphylaxis. J Allergy Clin Immunol 1984;73:699-703.
107. Sheffer AL, McFadden GR Jr, Grossman J, et al. Exercise-induced syncope with pruritus, urticaria and angioedema. J Allergy Clin Immunol 1979;63:174-5.
108. Fitzpatrick TB, Johnson AJ, Polano MK, et al. Color atlas and synopsis of clinical dermatology: Common and serious diseases. New York: McGraw-Hill, 1992:238-41.
109. Sarkany I, Gaylarde PM. Negative reactions to ice in cold urticaria. Br J Dermatol 1971;85:46.
110. Wanderer AA, Ellis EF. Treatment of cold urticaria with cyproheptadine. J Allergy Clin Immunol 1971;48: 366.