Winter Sports Dermatology: A Review

Sumedha Lamba Englund, MD; Brian B. Adams, MD, MPH

As more individuals choose to maintain their fitness level year-round, they inevitably encounter skin problems. During these athletic pursuits, the skin must endure ongoing insult, serving as the interface between the athlete and environmental factors unique to the sport and season. Therefore, primary care physicians and dermatologists must understand how athletic activity and weather contribute to the development of dermatoses. By appropriately recognizing winter sport dermatoses, the practitioner can best provide tailored effective treatment that enables the patient to quickly return to the winter sport.

Cutis. 2009;83:42-48.

Proper diagnosis and treatment of winter sport dermatoses requires an awareness of both the activity and the effect of cold exposure on the skin. Cold injuries include freezing and nonfreezing injuries. We first discuss cold injuries that may develop in any winter athlete because of exposure to the environment and then describe the specific dermatologic conditions resulting from participation in various winter sports.

CUTANEOUS EFFECTS OF COLD EXPOSURE

Cold Injuries: Freezing

Frostnip—Frostnip is the most common skin injury caused by cold weather.[1-4] The term *frostnip* denotes a hypothermic injury that involves only the superficial skin. The most commonly affected areas include the face, nose, cheeks, chin, and ears. Affected areas feel numb and have a blue to purple hue. A throbbing or burning sensation may persist for weeks.

Accepted for publication October 1, 2007.

Dr. Englund is from the University of Louisville School of Medicine, Kentucky. Dr. Adams is from the Department of Dermatology, University of Cincinnati School of Medicine, Ohio, and Veterans Administration Medical Center, Cincinnati.

The authors report no conflict of interest.

Correspondence: Brian B. Adams, MD, MPH, Department of Dermatology, University of Cincinnati, PO Box 670592, Cincinnati, OH 45267-0523 (adamsbb@email.uc.edu).

Penile frostnip was reported in a winter jogger after running in below-freezing, windy weather while wearing polyester trousers and cotton underwear with an anterior opening.[5] To prevent frostnip, athletes should dress in multiple layers and wear insulated clothing that withstands the outside temperature.[1,2] By adopting a layering effect when dressing, air is effectively trapped and serves as insulation.[6,7] Synthetic clothes wick away moisture compared to natural fibers that become damp with perspiration. Wet clothing reduces its insulation value to 10% of its effectiveness when dry.[4,8] Ski masks provide protection for the face, though they may be cumbersome.[6] Petrolatum-based emollients keep the skin at a higher temperature compared to untreated skin.[9,10] Natural sebum enhances this effect; therefore, delaying washing and shaving until after participation in outdoor activities are complete preserves this effect.[6]

Frostbite—Frostbite refers to the freezing of tissue caused by severe cold exposure that damages skin, subcutaneous tissue, muscle, and even bone. Athletes experience a higher risk for developing frostbite resulting from cold exposure at high altitudes. The combined effects of decreased oxygen tension and increased oxygen demand secondary to exercise worsen the cutaneous anoxia caused by compensatory peripheral vasoconstriction.[11,12] In addition, the evaporation of sweat increases cutaneous heat loss and hastens hypothermic injury.[13] Lastly, athletes engaged in high-velocity sports such as downhill skiing, snowmobiling, snowboarding, or luge have amplified rates of heat loss. The skin loses more heat at 10°C in 20-mph wind than at −10°C in still air.[14,15]

With sustained heat loss, blood flow to the skin diminishes, thermal heat loss ensues, and the early symptoms of pain and burning progress to numbness. Frostbite initially causes sudden blanching of a nose or ear with subsequent anesthesia.[16] Rapid rewarming in a circulating warm water bath of 38°C to 44°C treats frostbite, but one must only implement treatment after the possibility of further freezing has been eliminated because subsequent refreezing causes a greater degree of tissue necrosis.[6]

Cold Sensitivity Disorders: Nonfreezing

Perniosis (Chilblain)—Perniosis (chilblain) most commonly involves the hands and feet and occurs after prolonged exposure to a cold wet environment. Indurated, edematous, pruritic, red to violaceous plaques develop within several hours following cold exposure.[17,18] Perniosis can occur at other sites and location varies according to activity. Cutaneous vasoconstriction with subsequent hypoxemia causes perniosis. The relatively thicker layer of subcutaneous fat found on most women predisposes them to developing perniosis.[19]

A type of nodular pernio called *shoe boot pernio* occurs after wearing waterproof boots with wet inner linings.[20] Four cases of perniosis have been reported in men who waded through extremely cold mountain rivers.[21] Within 2 days, the men developed tender bruising on their thighs. A biopsy specimen showed edema of the papillary dermis with prominent perivascular and perifollicular lymphocytic infiltration and fat necrosis with foamy macrophages in the upper subcutaneous tissue consistent with deep perniosis and panniculitis. The lesions resolved within a week with rest and conservative management.[21] Wearing insulated, water-resistant clothing prevents perniosis in these athletes.

Raynaud Phenomenon—Transient benign vasospasm of the fingers occurs commonly, especially in young women. Raynaud phenomenon (RP) describes a dull pain with sudden blanching of the fingers linked to cold exposure and not associated with an underlying connective tissue disease. Patients with RP have more severe symptoms and more frequent episodes during the winter.[22] The etiology of the seasonal variation of RP cannot be explained simply by an increased vasoconstrictive response. This seasonal influence on finger skin temperature and microvascular blood flow that persists during the winter occurs irrespective of temperature.[23] During exposure to cold, athletes may feel a dull pain and typically only part of the hands suddenly becomes unevenly ivory white then blue and slightly swollen. Warming the hands fully relieves the athlete.

Athletes can prevent RP by wearing layers of thin, acrylic, moisture-wicking gloves with an outer waterproof layer; many commercially available varieties exist. Athletes with RP also must avoid uncovered contact with metal objects such as ski poles, which can quickly trigger an attack. For recalcitrant cases, extended-release nifedipine 30 mg at bedtime reduces the number of attacks.[24] Fish oil supplementation provides some added benefit.[25]

Cold-Dependent Exercise-Induced Anaphylaxis—Exercise-induced anaphylaxis (EIA) exhibits warmth, pruritus, erythema, urticaria, angioedema, respiratory distress, gastrointestinal symptoms (nausea, diarrhea, colic), and vascular collapse occurring in varying frequencies.[26-28] The symptoms of EIA usually occur within 5 minutes of exercise.[29,30]

Two possible mechanisms have been proposed for EIA. The first mechanism involves the activation of the complement pathway; the second theory implicates mast cell degranulation mediated via IgE, lactate, or creatine kinase resulting in the widespread release of histamine.[30-33] The resultant histaminemia causes the symptoms occurring with EIA.[34]

Exercise-induced anaphylaxis, a misnomer, rarely results in respiratory or vascular collapse.[35-38] Many factors, such as food ingestion (most common), medications (nonsteroidal anti-inflammatory drugs or aspirin), the menstrual cycle, and cold exposure, can exacerbate EIA.[39]

There is one reported case of cold-dependent EIA in a runner.[40] A 16-year-old adolescent boy had a 4-year history of recurrent urticaria and dyspnea after winter physical exercise such as jogging, playing handball, or riding a bicycle. Ingesting food, including wheat, did not cause the episode. A provocation test with 15 minutes of exercise and 2 minutes of cold stimulation immediately before or immediately after the exercise, however, did elicit localized wheals. The authors emphasized that although a variant of EIA, cold-dependent EIA is a distinct entity not to be confused with cold urticaria, food-dependent EIA, cholinergic urticaria, or cold-induced cholinergic urticaria.[40] Cold exposure may serve as an additional factor in all forms of EIA. A report of cold-dependant, food-dependent EIA exists.[41] In addition, in a study of 199 participants with EIA, cold was a factor for EIA in 23% of participants.[42] Indeed, another study of 279 participants with EIA demonstrated that avoidance of exercise in extremely cold or hot weather reduced the incidence of attacks in 44% of participants.[31]

The immediate treatment of all forms of EIA (cold-dependent, food-dependent, and others) focuses on maintaining airway patency and providing vascular support as needed. A long-acting, nonsedating antihistamine taken 1 hour before exercising helps prevent EIA.[34,35,43] If antihistamine therapy alone fails, prednisone 40 mg 12 hours prior to exercise may prove helpful.[44] Cromolyn sodium prevents the respiratory symptoms while ketoprofen relieves the dermatologic symptoms of EIA.[38,41] Exercise-induced anaphylaxis does not necessarily occur each time the athlete exercises; however, athletes with the disorder should carry injectable epinephrine (0.3–0.5 mL of 1:1000 solution) and exercise with a partner familiar with their condition.[30,39]

Cold Urticaria—Cold urticaria represents 1% to 3% of all forms of urticaria and develops in winter athletes.[2,37] Small to large urticarial plaques occur after exposure to cold weather or a cold object. Symptoms may occur in a generalized fashion or just at the site of cold contact and flare after rewarming.[37] To confirm the diagnosis, an ice cube is applied to the skin of the back or the ventral aspect of the forearm for 3 to 5 minutes. On rewarming, an urticarial plaque in the same shape and location as the ice cube will appear.[37,45] If results of the ice cube test are negative but cold urticaria is suspected, application of varying temperatures of cold water may induce a response.[2]

Cold urticaria most often has no cause but may relate to cold-dependent immunoglobulins associated with diseases such as cryofibrinogenemia, cryoglobulinemia, paroxysmal nocturnal hemoglobinuria, or connective tissue diseases. Treatment of the underlying diseases may ameliorate the symptoms of secondary cold urticaria.[37] In the idiopathic form, antihistamines such as oral cyproheptadine hydrochloride (4 mg given 3 times daily or the minimal effective dose) provide some benefit.[46] Some authors have noted success with cold desensitization.[47,48]

DERMATOSES UNIQUE TO SPECIFIC WINTER SPORTS

Skiing and Snowboarding

UV Radiation Exposure—For a variety of reasons, including increased altitude, reflectance, athlete's attitude toward sun protection, and increased sweating, outdoor athletes experience high levels of UV radiation. Athletes at particular risk include alpine sports enthusiasts, such as hikers, snow skiers, and snowboarders.[7] An altitude-related increase in UV radiation exists, especially for shortwave radiation.[49,50] One study conducted at the Mount Hutt ski field in New Zealand demonstrated that the maximum UV radiation intensity was 20% to 30% greater than the UV radiation intensity experienced on the same day at the seaside town of Christchurch, which is 90 km east and 2 km below Mount Hutt.[51] Both sites share the same latitude. The authors provided multiple reasons explaining the higher level of UV radiation on Mount Hutt. First, at a higher altitude, less air exists to scatter the radiation. Second, fresh snow enhances the reflection of UV radiation. Finally, the depleted layer of tropospheric ozone permits the transmission of higher levels of UV radiation.[51]

Snow-covered surfaces may reflect up to 100% of the UV radiation they receive. This scattered UV radiation increases the total UV radiation by 2% to 3% with each 100-m rise in altitude.[52,53] Furthermore, reflected light often strikes unprotected areas of the skin (for example, under the chin and on the lower lip).[54] Thus, skiers with an average skin type, at noon, and at 11,000 ft may sustain a minimal erythema dose (MED) of only 6 minutes.[52] Specifically, a study reported that skiers with Fitzpatrick skin type II received total UVB exposure ranging from 12 to 185 mJ/cm^2 (0.5–7.6 times the MED).[55]

Unfortunately, only 57% (89/156) of skiers and snowboarders use sun protection.[56] Outdoor winter athletes should wear sweatproof, broadband-blocking sunscreen with a sun protection factor of at least 30.[57,58] Athletes must reapply sunscreen frequently, not only because sunscreen becomes less effective after periods of intense sweating or rubbing but because sweating reduces the MED.[59,60]

Recurrent Herpes Labialis—The lips of outdoor athletes receive a great deal of UV radiation[61] that can reactivate herpes labialis.[62-64] The use of photoprotective sunscreens can prevent this reactivation.[65] One study showed that sunscreen significantly reduced recurrence of herpes labialis (1/19, 5%) at 6 days compared with placebo (11/19, 58%)($P<.01$).[66]

Prophylactic treatment with acyclovir also decreased the incidence of herpes labialis in participants exposed to sun. Of 75 skiers treated with acyclovir 400 mg twice daily first starting 12 hours prior to sun exposure, only 5 (7%) developed lesions within a week compared to 19 (26%) of 72 individuals in the placebo group ($P<.001$).[67] A small study showed that valacyclovir prevents recurrent herpes labialis and requires a daily dose.[68]

Mogul Skier's Palm—The repeated planting of ski poles by skiers, especially mogul skiers, results in ecchymosis of the hypothenar termed *mogul skier's palm*.[69,70] For reasons that remain unclear, this innocuous blemish occurs almost exclusively in young adults and adolescents.[71] Mogul skiers must plant their poles forcefully and drive their hands down quickly as they maneuver snow mounds (moguls) in the ski path. This action causes a repeated lateral shearing force on the epidermis, sliding it over the rete pegs of the papillary dermis and causing intraepidermal, and ultimately, intracorneal hemorrhage.

The ecchymosis has a purpuric to golden hue because of the presence of hemosiderin from prior hemorrhages. This benign ecchymosis shares an appearance similar to acral lentiginous malignant melanoma. However, because the lesions usually appear as bilateral punctate petechiae, a biopsy need not be performed. Clinicians can confirm the diagnosis by removing a few puncta with a scalpel, mixing the tissue with a small amount of water, and confirming the presence of blood through an

occult blood test. The palms usually clear in 2 to 3 weeks after ski season ends.[69] The lesion also can be pared.

Skier's Toenail—Pressure in the toe box of a ski boot or skate can cause bleeding under the toenail. These subungual hematomas result from repetitive trauma caused by quick starts and stops and also occur in other sports such as running and tennis.[72-77] In skiers, an improperly fitted ski boot that allows the distal toe to repeatedly strike the front of the toe box causes a blackened toenail. Subungual hyperkeratosis, onycholysis, nail thickening, and secondary fungal infection also occur with continual injury.[43,78,79] The differential diagnosis of a persistently blackened toenail also should include onychomycosis and subungual malignant melanoma.[72,74,76]

Potassium hydroxide testing, culture, or periodic acid-Schiff staining of subungual debris can differentiate onychomycosis from skier's toenail.[72,74,76,77] Clinicians should be suspicious of melanoma if discoloration of the nail appears on the periungual region (Hutchinson sign), if pigment extends to the lateral edge, or if different hues exist within the pigmented area.[72,74,77,79] Suspicion for melanoma or a fractured toe warrants performing a biopsy or radiography, respectively.[72,74,76,79]

If a patient presents with an acute subungual hematoma, physicians can provide relief and prevent the spread of hemorrhage and nail loss by evacuating the accumulated blood. Several methods providing ungual fenestration exist including simple incision, punch biopsy, hot wire puncture, and thermal cautery.[8,75] Rest and warm water soaks help to relieve chronic lesions.[2,7,44,75] With time, usually after several months, resolution of skier's toenail occurs. To preemptively mitigate concern, clinicians should warn athletes that the toenail may stay black for several months.[72,74,77,80]

To prevent skier's toenail, a skier must wear properly fitted ski boots. The anterior toe box should only allow minor forward slippage.[2,7,81] In addition, short toenails cut straight across and close to the skin prevent the condition.[2,7,72,76,77,82] If over-the-counter methods prove ineffective, patients should consult a podiatrist.[74]

Tobogganing

Tobogganer's Thighs—The extended duration of cold and wet exposure puts tobogganers and sledders at risk for cutaneous and physical injury.[83] A 16-year-old obese adolescent boy developed erythrocyanosis and chilblain on the inner aspects of his thighs after he spent all day wearing very tight denim jeans in his toboggan.[84]

The lesions appeared as pruritic, erythematous, dusky nodules that ultimately ulcerated. In combination, the compressive clothing, cold exposure, and thick layer of subcutaneous fat on the thighs caused vasoconstriction that purportedly produced tobogganer's thighs. Tobogganer's thighs resolve with time, cessation of tobogganing, and rest indoors. Critical preventative measures include wearing insulated loose-fitting pants, losing weight, and shortening the duration of time spent outside.

Snowmobiling

Bullous Frostbite (Polaris Vulgaris)—Snowmobilers experience a high risk for frostbite, not only because of the frigid conditions in which they ride but because of the wind velocity they sustain in their snowmobiles. Commonly called *polaris vulgaris* after the snowmobile manufacturer Polaris, one reported case of the severe form exists.[85] A Minnesota snowmobiler developed bullous frostbite on his unprotected neck as he attained speeds of 40 mph (wind chill factor, −73.4°C). The combination of high wind velocity and extreme cold exposure led to 2 large blisters with clear yellow exudate. The snowmobiler was effectively treated with silver sulfadiazine cream 1% along with a semipermeable dressing.[85] To protect the underside of the neck, synthetic or absorbent terrycloth can be wrapped around the neck along with a muffler. Snowmobilers also must wear an insulated helmet, mask, and insulated gloves to protect other vulnerable areas.[86]

Vibration-Induced White Finger—Sympathetic stimuli trigger the initial attack of vibration-induced white finger (VWF), and for snowmobilers, the stimulus is cold.[87] Vibration-induced white finger manifests in various stages. In the earliest stage, the patient may report numbness or tingling of the fingers hours after riding the snowmobile. This numbness or tingling may precede finger blanching when exposed to cold. Rewarming the hands causes pain and hyperemia. Snowmobilers usually have asymmetric symptoms, unlike primary RP. With continued exposure, the extent, duration, and frequency of blanching increases as more fingers are affected by the condition.[88] Symptoms of VWF may be reduced by avoiding exposure to cold and vibration and by implementing measures that increase peripheral circulation such as smoking cessation. For severe cases, calcium channel blocker therapy improves VWF.

To prevent VWF, snowmobilers should wear 2 layers of waterproof gloves: one insulated waterproof layer and one antivibration layer. In most cases, the condition resolves if the snowmobiler limits cold exposure.[87]

Ice Hockey and Ice Skating

Contact Dermatitis—Winter athletes develop many types of allergic skin reactions including both irritant and allergic contact dermatitis.[32] Fiberglass in hockey

sticks caused an epidemic of irritant contact dermatitis in National Hockey League ice hockey players.[75,79] Dyes in hockey gloves also have caused allergic contact dermatitis.[89] The location of the rash and the history provide the most important diagnostic clues.

Skate Bite—Skate bites represent a form of athlete's nodules found on the ankles of hockey players and ice skaters who wear tight-fitting skates.[2,90] Indeed, all winter athletes who wear tight-fitting boots or shoes are prone to develop athlete's nodules.[2,7,91] These flesh-colored asymptomatic nodules occur at sites of recurrent friction and trauma. The location of the lesion and history of athletic activity support the diagnosis. The differential diagnosis depends on the location and includes the following: gout, ganglion cyst, epidermoid cyst, hypertrophic scar, granuloma annulare, callus, and elastoma.[91] A punch biopsy confirms the diagnosis.[81] Histology reveals an increase in collagen bundles in the reticular dermis resembling a collagenoma. Treatment of these lesions includes either surgical removal or intralesional steroids.[91] To prevent athlete's nodules, use of protective padding at the sites of trauma should be encouraged.[90]

CONCLUSION

As more individuals seek improved health through participation in athletic activity, associated skin findings also increase. Knowledge of the actions and equipment used in winter sports and their effects on the skin facilitates the identification of the athlete's dermatoses. Furthermore, by appropriately recognizing winter skin ailments, physicians can effectively treat them in a manner that reduces recovery time and allows the patient to rapidly return to the winter sport. Finally, while some of the skin reactions associated with winter sports occur as interesting oddities, others require urgent treatment. Therefore, practitioners should have a keen awareness of all winter sport dermatoses.

REFERENCES

1. Levine N. Dermatologic aspects of sports medicine. *J Am Acad Dermatol*. 1980;3:411-424.
2. Pharis DB, Teller C, Wolf JE. Cutaneous manifestations of sports participation. *J Am Acad Dermatol*. 1997;36: 448-459.
3. Atton AV, Tunnessen WW. The athlete and his skin. *Clin Rev Allergy*. 1988;6:403-429.
4. Basler RS. Skin lesions related to sports activity. *Prim Care*. 1983;10:479-494.
5. Herskowitz M. Penile frostbite, an unforeseen hazard of jogging. *N Engl J Med*. 1977;296:178.
6. Basler RS. Skin injuries in sports medicine. *J Am Acad Dermatol*. 1989;21:1257-1262.
7. Basler RS. Sports-related skin injuries. *Adv Dermatol*. 1989;4:29-49.
8. Mikelionis J. Mountains, snow, and skin. *Cutis*. 1977; 20:346-347.
9. Thorleifsson A, Wulf HC. Emollients and the response of facial skin to a cold environment. *Br J Dermatol*. 2003;148:1149-1152.
10. Lehmuskallio E, Rintamäki H, Anttonen H. Thermal effects of emollients on facial skin in the cold. *Acta Derm Venereol*. 2000;80:203-207.
11. Pugh LG. Cold stress and muscular exercise, with special reference to accidental hypothermia. *Br Med J*. 1967;1:333-337.
12. Kroeger K, Jannsen S, Niebel W. Frostbite in a mountaineer. *VASA*. 2004;33:173-176.
13. Middleton JD. The effect of temperature on extensibility of isolated corneum and its relation to skin chapping. *Br J Dermatol*. 1969;81:717-721.
14. Lloyd EL. ABC of sports medicine: temperature and performance I: cold. *BMJ*. 1994;309:531-534.
15. Gavhed D, Makinen T, Holmer I, et al. Face cooling by cold wind in walking subjects. *Int J Biometereol*. 2003;47:148-155.
16. Glickman FS. Hikers' hazards. *Cutis*. 1977;19:497-500.
17. Roach JJ. Coping with killing cold. *Phys Sports Med*. 1975;3:34.
18. Goette DK. Chilblains (perniosis). *J Am Acad Dermatol*. 1990;23:257-262.
19. Winter kibes in horsey women. *Lancet*. 1980;2:1345.
20. Coskey RJ, Mehregan AH. Shoe boot pernio. *Arch Dermatol*. 1974;109:56-57.
21. Price RD, Murdoch DR. Perniosis (chilblains) of the thigh: report of five cases, including four following river crossings. *High Alt Med Biol*. 2001;2:535-538.
22. Leppert J, Ringqvist A, Ahiner J, et al. Seasonal variation in cyclic GMP response on whole-body cooling in women with primary Raynaud's phenomenon. *Clin Sci (Lond)*. 1997;93:175-179.
23. Gardner-Medwin JM, Macdonald IA, Taylor JY, et al. Seasonal differences in finger skin temperature and microvascular blood flow in healthy men and women are exaggerated in women with primary Raynaud's phenomenon. *Br J Clin Pharmacol*. 2001;52:17-23.
24. Landry GJ, Edwards JM, Porter JM. Current management of Raynaud's syndrome. *Adv Surg*. 1996;30:333-347.
25. DiGiacomo RA, Kremer JM, Shah DM. Fish-oil dietary supplementation in patients with Raynaud's phenomenon: a double-blind, controlled, prospective study. *Am J Med*. 1989;86:158-164.
26. Kaplan AP. Exercise-induced hives. *J Allergy Clin Immunol*. 1984;73:704-707.
27. Sheffer AL, Austin KF. Exercise-induced anaphylaxis. *J Allergy Clin Immunol*. 1984;73:699-703.
28. Sheffer AL, McFadden GR Jr, Grossman J, et al. Exercise-induced syncope with pruritus, urticaria,

and angioedema. *J Allergy Clin Immunol*. 1979;63: 174-175.
29. Leshaw SW. Itching in active patients. *Phys Sportsmed*. 1998;26:47-53.
30. Adams BB. Exercise-induced anaphylaxis in a marathon runner. *Int J Dermatol*. 2002;41:394-396.
31. Shaddick NA, Liang MH, Partridge AJ, et al. The natural history of exercise-induced anaphylaxis: survey results from 10-year follow-up study. *J Allergy Clin Immunol*. 1999;104:123-127.
32. Fisher AA. Sports-related allergic dermatitis. *Cutis*. 1992;50:95-97.
33. Casale TB, Keahey TM, Kaliner M. Exercise-induced anaphylactic syndromes: insights into diagnostic and pathophysiologic features. *JAMA*. 1986;255:2049-2053.
34. Sheffer AL, Tong AKF, Murphy GF, et al. Exercise-induced anaphylaxis: a serious form of physical allergy associated with mast cell degranulation. *J Allergy Clin Immunol*. 1985;75:479-484.
35. Sheffer AL, Austen KF. Exercise-induced anaphylaxis. *J Allergy Clin Immunol*. 1980;66:106-111.
36. Sheffer AL, Soter NA, McFadden ER, et al. Exercise-induced anaphylaxis: a distinct form of physical allergy. *J Allergy Clin Immunol*. 1983;71:311-316.
37. Briner WW, Sheffer AL. Exercise-induced anaphylaxis. *Med Sci Sports Exerc*. 1992;24:849-850.
38. Nichols AW. Exercise-induced anaphylaxis and urticaria. *Clin Sports Med*. 1992;11:303-312.
39. Castells MC, Horan RJ, Sheffer AL. Exercise-induced anaphylaxis. *Clin Rev Allergy Immunol*. 1999;17: 413-424.
40. Li M, Sayama K, Tohyama M, et al. A case of cold-dependent exercise-induced anaphylaxis. *Br J Dermatol*. 2002;147:368-370.
41. Shimizu T, Furumoto H, Kinoshita E, et al. Food-dependent exercise-induced anaphylaxis occurring only in winter. *Dermatology*. 2000;200:279.
42. Wade JP, Liang MH, Sheffer AL. Exercise-induced anaphylaxis: epidemiologic observations. *Prog Clin Biol Res*. 1989;297:175-182.
43. Mailler-Savage EA, Adams BB. Skin manifestations of running. *J Am Acad Dermatol*. 2006;55:290-301.
44. Fisher AA. Sports-related allergic dermatitis. *Cutis*. 1992;50:95-97.
45. Sarkany I, Gaylarde PM. Negative reactions to ice in cold urticaria. *Br J Dermatol*. 1971;85:46-48.
46. Wanderer AA, Ellis EF. Treatment of cold urticaria with cyproheptadine. *J Allergy Clin Immunol*. 1971;48: 366-371.
47. Leigh IM, Ramsay CA, Calnan CD. Cold urticaria-"desensitization". *Trans St Johns Hosp Dermatol Soc*. 1974;60:40-42.
48. Kobza-Black A, Sibbald RG, Greaves MW. Cold urticaria treated by induction of tolerance [letter]. *Lancet*. 1979;2:964.
49. Seckmeyer G, McKenzie RL. Increased ultraviolet radiation in New Zealand (45°S) relative to Germany (48°N). *Nature*. 1992;359:135-137.
50. Blumthaler M, Webb AR, Seckmeyer G, et al. Simultaneous spectroradiometry: a study of solar UV irradiance at two altitudes. *Geophys Res Lett*. 1994;21:2805-2808.
51. Allen M, McKenzie R. Enhanced UV exposure on a ski-field compared with exposures at sea level. *Photochem Photobiol Sci*. 2005;4:429-437.
52. Blumthaler M. Solar UV measurements. In: Tevini M, ed. *UV-B Radiation and Ozone Depletion: Effects on Humans, Animals, Plants, Microorganisms and Materials*. Boca Raton, FL: Lewis Publishers; 1993:71-94.
53. Sunscreens. *Med Lett Drugs Ther*. 1979;21:46-48.
54. Rigel DS, Rigel EG, Rigel AC. Effects of altitude and latitude on ambient UVB radiation. *J Am Acad Dermatol*. 1999;40:114-116.
55. Rigel EG, Lebwohl MG, Rigel AC, et al. Ultraviolet radiation in alpine skiing: magnitude of exposure and importance of regular protection. *Arch Dermatol*. 2003;139:60-62.
56. Buller DB, Andersen PA, Walkosz B. Sun safety behaviours of alpine skiers and snowboarders in the western United States. *Cancer Prev Control*. 1998;2:133-139.
57. Whitmore ES, Morison WL. Prevention of UVB-induced immunosuppression in humans by high sun protection factor sunscreen. *Arch Dermatol*. 1995;131:1128-1133.
58. Diffey BL. When should sunscreen be reapplied? *J Am Acad Dermatol*. 2001;45:882-885.
59. Moehrle M, Koehle W, Dietz K, et al. Reduction of minimal erythema dose by sweating. *Photodermatol Photoimmunol Photomed*. 2000;16:260-262.
60. Gambichler T, Schropl F. Changes of minimal erythema dose after water and salt water baths. *Photodermatol Photoimmunol Photomed*. 1998;14:109-111.
61. Maier H, Schauberger G, Brunnhofer K, et al. Assessment of thickness of photoprotective lipsticks and frequency of reapplication: results from a laboratory test and a field experiment. *Br J Dermatol*. 2003;148:763-769.
62. Blyth WA, Hill TJ, Field HJ, et al. Reactivation of herpes simplex virus infection by ultraviolet light and possible involvement of prostaglandins. *J Gen Virol*. 1976;33:547-555.
63. Spruance SL, Pathogenesis of herpes simplex labialis: experimental induction of lesions with UV light. *J Clin Microbiol*. 1985;22:366-368.
64. Spruance SL. The natural history of recurrent oral-facial herpes simplex virus infection. *Semin Dermatol*. 1992;11:200-206.
65. Rooney JF, Bryson Y. Prevention of ultraviolet-light–induced herpes labialis by sunscreen. *Lancet*. 1991;338:1419-1422.
66. Duteil L, Queille-Roussel C, Loesche C, et al. Assessment of the effect of a sunblock stick in the prevention of solar-stimulating ultraviolet light-induced herpes labialis. *J Dermatol Treat*. 1998;9:11-14.

67. Spruance SL, Hamill ML, Hoge WS, et al. Acyclovir prevents reactivation of herpes simplex labialis in skiers. *JAMA*. 1988;260:1597-1599.
68. Baker D, Eisen D. Valacyclovir for prevention of recurrent herpes labialis: 2 double-blind, placebo-controlled studies. *Cutis*. 2003;71:239-242.
69. Swinehart JM. Mogul skier's palm: traumatic hypothenar ecchymosis. *Cutis*. 1992;50:117-118.
70. Izumi AK. Pigmented palmar petechiae (black palm)[letter]. *Arch Dermatol*. 1974;109:261.
71. Houston SD, Knox JM. Skin problems related to sports and recreational activities. *Cutis*. 1977;19:487-491.
72. Adams BB. Jogger's toenail. *J Am Acad Dermatol*. 2003;48(suppl 5):S58-S59.
73. Scher JK. Jogger's toe. *Int J Dermatol*. 1978;17:719-720.
74. Adams BB. Running-related toenail abnormality. *Phys Sportsmed*. 1999;27:85-87.
75. Kantor GR, Bergfeld WF. Common and uncommon dermatologic diseases related to sports activities. *Exerc Sport Sci Rev*. 1988;16:215-253.
76. Adams BB. Dermatologic disorders of the athlete. *Sports Med*. 2002;32:309-312.
77. Mailler EA, Adams BB. The wear and tear of 26.2: dermatological injuries reported on marathon day. *Br J Sports Med*. 2004;38:498-501.
78. Baran R, Badillet G. Primary onycholysis of the big toenails: a review of 113 cases. *Br J Dermatol*. 1982;106:529-534.
79. Conklin RJ. Common cutaneous disorders in athletes. *Sports Med*. 1990;9:100-119.
80. Fisher AA. Sports-related cutaneous reactions, part III. sports identification marks. *Cutis*. 1999;63:256-258.
81. Adams BB. More on jogger's toe [letter]. *Phys Sportsmed*. 2000;28:20.
82. Adams BB. Sports dermatology. *Dermatol Nurs*. 2001;13:347-363.
83. Tobogganing injuries. *BMJ*. 1991;302:849-850.
84. Long CC, Holt PJ. Tobogganers thighs. *Clin Exp Dermatol*. 1992;17:466-467.
85. Nissen ER, Melchert PJ, Lewis EJ. A case of bullous frostbite following recreational snowmobiling. *Cutis*. 1999;63:21-23.
86. Christenson C, Stewart C. Frostbite. *Am J Fam Pract*. 1984;30:111-122.
87. Vivokannas H, Anttonen H. Combined effects of cold, vibration, and smoking particularly in snowmobile users. *Arctic Med Res*. 1994;53(suppl 3):29-34.
88. Friden J. Vibration damage to the hand: clinical presentation, prognosis and length and severity of vibration required. *J Hand Surg*. 2001;75:55-57.
89. Helm TN, Bergfeld WF. Sports dermatology. *Clin Dermatol*. 1998;16:159-165.
90. Cohen PR, Eliezri YD, Silvers DN. Athlete's nodules. *Sports Med*. 1990;10:198-203.
91. Cohen PR, Eliezri YD, Silvers DN. Athlete's nodules: sports-related connective tissue nevi of the collagen type (collagenomas). *Cutis*. 1992;50:131-135.