Cold Weather Issues in Sideline and Event Management

J. Andrew McMahon, DO and Allyson Howe, MD

Abstract

Exercise in cold environments exerts a unique physiologic stress on the human body, which, under certain conditions, may result in a cold-related injury. Environmental factors are the most important risk factors for the development of hypothermia in athletes. Frostbite occurs as a result of direct cold injury to peripheral tissues. The biggest risk for frostbite is temperature. Trench foot is a result of repeated and constant immersion in cold water. Chilblains are local erythematous or cyanotic skin lesions that develop at ambient air temperatures of 32°F to 60°F after an exposure time of about 1 to 5 h. Cold urticaria is, essentially, an allergic reaction to a cold exposure and can be controlled with avoidance of the cold. There are a number of risk factors and conditions that predispose athletes to cold injury, but exercise in the cold can be done safely with proper education and planning.

Introduction

With the growing popularity of winter sports and increased participation in endurance sports such as the triathlon, more athletes are being exposed to cold environments during activity. Exercise in cold environments exerts a unique physiologic stress on the human body, which, under certain conditions, may result in a cold-related injury. Some of these injuries result in direct tissue injury while others result in systemic manifestations as a result of a drop in core body temperature. There are a number of risk factors and conditions that predispose athletes to cold injury, but exercise in the cold can be done safely with proper education and planning.

Cooling and the Normal Physiology of Cold Exposure

Cooling occurs through several mechanisms. The most important mechanisms in sports medicine are evaporative, conductive, and convective cooling. Evaporative cooling occurs when water is vaporized directly from the body surface, a phase change that requires heat to occur. The rate of cooling is very rapid with this phase change and is accentuated in humid or wet environments. Conductive cooling results when body heat is transferred to a cooler object in direct physical contact with the person. This typically occurs when body heat is transferred into wet clothing or when an athlete lies on the ground.

"Convective loss" is a result of direct transfer of heat from an object to moving fluid currents, most commonly the wind. "Wind chill" is an example of this phenomenon and demonstrates the direct relationship between wind speed and heat loss. Despite the fact that wind chill temperature values are lower, wind does not cool the skin below the ambient temperature. However, cool air rapidly soaks up heat from tissues that are rich in vasodilated capillaries in response to the potential for cold injury. This accelerated cooling predisposes to frostbite as well as hypothermia if an athlete does not wear appropriate wind-breaking layers (5,7).

When exposed to cold, the human body undergoes a number of adaptive and protective changes. First, vasoconstriction shunts blood flow from peripheral structures to the core, reducing heat loss to the environment. Initially, hypoperfused surface layers function as insulation for the vital organs of the core. However, these peripheral structures continue to cool predisposing athletes to direct cold injuries like frostbite (3).

To help compensate, another vasomotor phenomenon, cold-induced vasodilatation, helps modulate perfusion to areas that are affected by vasoconstriction. Aiming to preserve the health of peripheral structures, periodic episodes of vasodilatation provide blood flow to oxygen-starved peripheral tissues (10). With continued exposure to the cold, this protective mechanism subsequently is lost or blunted as hypothermia progresses (4).

On the larger scale, the body makes several different efforts to generate heat. Thermogenesis happens voluntarily when an athlete chooses to increase physical activity, *e.g.*, jumping jacks or sideline sprints. It also occurs involuntarily through shivering. Shivering begins in the moments after cold exposure and typically affects girdle and core muscles first, before spreading to the extremities. Shivering can

Maine Medical Center, Portland, Maine.

Address for correspondence: Allyson Howe, MD, Maine Medical Center, 5 Bucknam Road, Falmouth, ME 04105; E-mail: howea1@mmc.org

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increase the basal metabolic rate by as much as six times (6). Some individuals with frequent exposure to cold environments develop an exaggerated shivering response to aid in cold acclimatization (3).

Hypothermia

Hypothermia occurs when the body's ability to generate and preserve heat is surmounted by heat loss to the environment. Technically, hypothermia is defined as a decline in body temperature of 2°C from normal core body temperature and is categorized based on the degree of departure from normal (3).

Environmental factors are the most important risk factors for the development of hypothermia in athletes. Water is extremely effective at conductive and convective heat transfer, and athletes exercising in or inadvertently exposed to it are at a risk for rapid heat loss (3). Exposure to wind, which can be a by-product of the activity itself as in downhill skiing, results in convective heat loss. Heat loss seems to be greater in women than in men secondary to a larger surface area-to-mass ratio (12). Although physical fitness does not seem to alter the likelihood of cold injury, being overtrained, fatigued, and undernourished are clear risks for hypothermia. Impaired autoregulation as a result of central nervous system or endocrine diseases and drug and alcohol abuse is also an important risk factor (3).

In the field, diagnosis is based upon clinical assessment and measurement of body temperature. Many thermometers only measure as low as 34.4°F and are thus inadequate in all but the mildest of cases of hypothermia. A special low-reading rectal thermometer or rectal thermistor probe is essential to assessing core temperature. If available, multiple simultaneous methods are ideal as body temperature is often heterogeneous, especially during rewarming.

"Mild hypothermia" is present when body core temperature is between 32°C and 35°C. Other than obvious shivering, early mild hypothermia is sometimes vague and difficult to identify. Athletes may be irritable or apathetic, but this can progress to ataxia, dysarthria, and confusion as temperature continues to drop. A reactive sympathetic drive results in hypertension, tachycardia, and hyperglycemia as means to produce heat and restore homeostasis. Peripheral vasoconstriction shunts blood to the core resulting in a pale cool complexion and extremities. In response to this increased systemic vascular resistance and increase in central volume, the kidney excretes large volumes of dilute urine, and dehydration results (5).

An athlete's electrocardiogram (ECG) may show prolonged PR, QRS, and QTc intervals, and shivering may complicate interpretation given a wandering baseline. Osborn or J waves occur at temperatures below 32°C and are hypothesized to represent cold-induced ion fluxes (Fig. 1). Coagulopathy and thrombocytopenias are common in hypothermia and become more severe as the degree of hypothermia worsens. They are typically inconsequential unless trauma occurs in the athlete (5).

"Moderate hypothermia" is defined as core body temperatures between 28°C and 32°C. These individuals may progress to stupor, and pupils dilate. Shivering tends to cease in moderate disease, essentially eliminating any intrinsic means to recover homeostasis. Bradycardia or hypotension may develop, and the risk of arrhythmia increases (5).

"Severe hypothermia" results when the core temperature drops below 28°C. The resultant hypopnea, bradycardia or asystole, hypotension, and coma make these individuals seem dead. Patients are areflexic, lack corneal reflex, and have profound acid-base disturbances, which predispose to significant dysrhythmia (5).

Treatment

There are a number of rewarming techniques employed in treating hypothermic athletes; the modality chosen is largely a function of the degree of hypothermia.

"Passive external rewarming" is the treatment of choice for mild hypothermia and is used as an adjunctive therapy in more severe cases. Ideally, the athlete should be moved indoors where wet clothing is removed, and the patient subsequently is wrapped in blankets. With the opportunity for heat loss eliminated, intrinsic thermogenesis reestablishes homeostasis.

Those with severe glycogen depletion or major medical problems at baseline, specifically the elderly, likely will be unable to mount enough of a metabolic response to correct hypothermia exclusively through passive means. If a mildly hypothermic athlete fails to rewarm, other medical issues like adrenal crisis or myxedematous coma should be considered. From the time of diagnosis through completion of rewarming, attention to airway, breathing, and circulation are crucial, and blood glucose levels should be monitored. The metabolic demands of shivering may dictate the use of supplemental oxygen, and sympathetically driven bronchorrhea may necessitate a definitive airway in less alert athletes (5).

"Active external rewarming" (AER) is used for moderate and severe hypothermia. Great care is needed in handling a patient with this degree of hypothermia. The patient should be transported horizontally, and any use of peripheral muscles must be avoided. Once removed from the cold wet environment, rewarming occurs through a combination of warm blankets and additional sources of heat that may include heating pads or blankets, warm baths, Bair Hugger, or forced warm air.

The big concern with AER is core temperature after drop, which occurs when the hypothermic extremities are perfused before the core has warmed adequately. In the severely hypothermic individual, the extremities are full of cold, stagnant peripheral blood, a result of vasoconstriction. When this cold and acidotic blood is returned prematurely to a heart struggling to compensate, the potential for cardiovascular collapse and fatal arrhythmia is very real. Core temperature after drop can be precipitated simply by the vasodilatation that accompanies the patient's transition to a warmer environment. It is the reason that moderate to severely hypothermic patients should not be allowed to engage peripheral muscles, which may result in vasodilatation and mobilization of the lactic acid. With AER, an effort to warm the core first is imperative, and active warming modalities should be focused there first (5).

The cold myocardium is extremely irritable, and arrhythmia is a common consequence of the rewarming process,

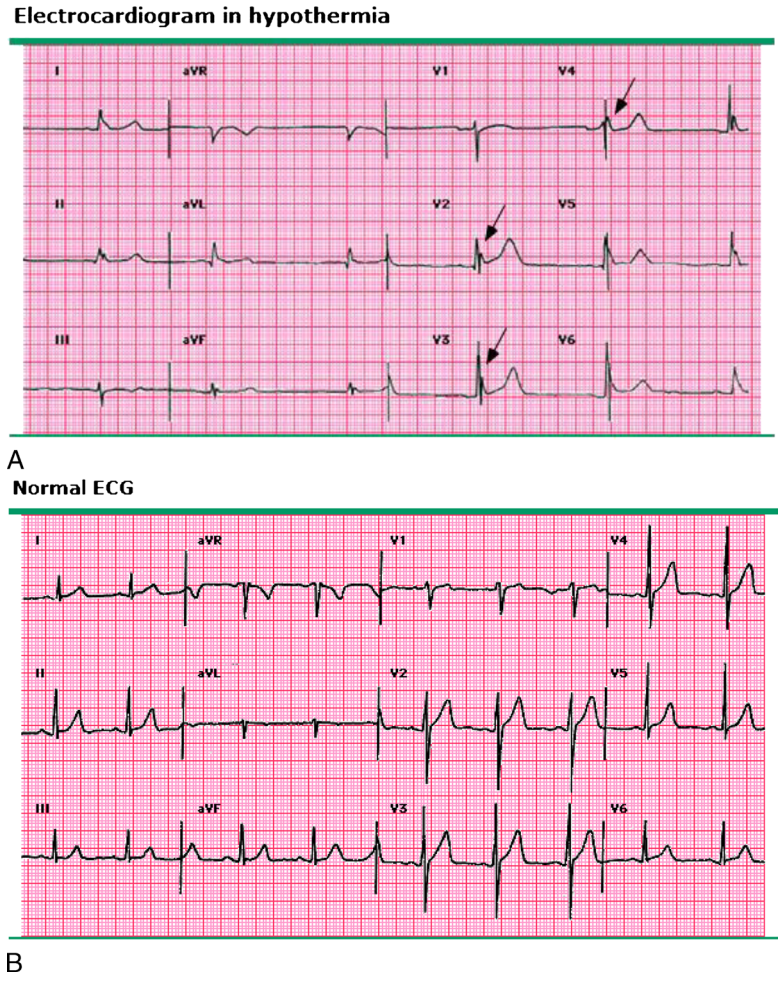

Figure 1: A. The ECG reveals marked sinus bradycardia (about 40 beats·min^{-1}) with first-degree atrioventricular block (PR interval = 0.23 s). The slow heart rate in this patient is due to hypothermia (90°F, 32.2°C), which also produces prominent convex deflections at the J point (the junction of the QRS and ST segments) that are best seen in the precordial leads. The J waves or Osborn waves (*arrows*) are characteristic of severe hypothermia and resolve with rewarming; how they occur is not known. (Reprinted from Mechem CC, Danzl DF. Accidental hypothermia in adults. In: Basow DS, editor. *UpToDate*. Waltham (MA): UpToDate; 2011. Copyright © 2011 UpToDate, Inc. Used with permission. For more information visit www.uptodate.com.) B. Normal ECG showing normal sinus rhythm at a rate of 75 beats·min^{-1}, a PR interval of 0.14 s, a QRS interval of 0.10 s, and a QRS axis of approximately 75°. (Reprinted from Mechem CC, Danzl DF. Accidental hypothermia in adults. In: Basow DS, editor. *UpToDate*. Waltham (MA): UpToDate; 2011. Copyright © 2011 UpToDate, Inc. Used with permission. For more information visit www.uptodate.com.)

acidosis, and electrolyte shifts that accompany hypothermia. Atrial fibrillation and flutter, typically without a rapid ventricular response, are common and typically resolve once rewarming is complete. Bradycardia may be physiologic during hypothermia, and pacing or atropine is usually unnecessary unless persistent beyond the rewarming period. Ventricular arrhythmias and asystole are life-threatening consequences of a cold and acidotic myocardium and may be refractory to the conventional approach to advanced cardiac life support (*5*). American Heart Association guidelines state that hypothermic patients in a pulseless ventricular arrhythmia should receive initial attempts at defibrillation and chemical cardioversion. However, if unsuccessful, cardiopulmonary resuscitation and rewarming should replace additional attempts at cardioversion until the core temperature has been brought above 30°C (*8*).

"Active internal rewarming" is the most aggressive approach to reversing severe hypothermia. It is largely a hospital-based approach to rewarming and may be as simple as an infusion of warm crystalloid solutions or as complex and invasive as peritoneal irrigation or extracorporeal blood rewarming with hemodialysis (*5*).

Direct Cold Injuries

Frostbite

Frostbite occurs as a result of direct cold injury to peripheral tissues. When tissue temperatures drop below 0°C, superficial layers of skin freeze. It commonly occurs in exposed areas with limited blood flow, *e.g.*, nose, ears, or wrists, but also commonly affects hands and feet when tissue temperatures are dropped by peripheral vasoconstriction. Contact or instant frostbite can occur through

contact with highly conductive substances like rock or metal (3).

The biggest risk for frostbite is temperature. The ambient air temperature must be less than 0°C for frostbite to occur. Like hypothermia, fatigue, malnutrition, and many medical comorbidities, including peripheral vascular disease and diabetes, also are risk factors for frostbite. Alcohol abuse commonly is involved in those with frostbite as its use results in poor decision making and alcohol's vasodilatory properties causes hypothermia and subsequent peripheral vasoconstriction (3).

The injury begins with superficial cooling of peripheral tissues. As skin temperatures drop below 10°C, damage to cell membranes results in numbness, pain, and edema. The skin itself initially may look red and then progress to a dull waxy white color while finger dexterity is lost. As cold exposure continues, ice crystals form in the intra- and extracellular compartments distorting normal cell architecture. Increased vascular permeability allows plasma to leak and subsequently freeze. Tissues then become ischemic and hypercoagulable, and vasospasm may potentiate the localized circulatory failure (13).

Treatment

First and foremost, the patient should be removed from the cold environment, and wet clothes should be replaced with loose-fitting dry garments. Hypothermia should be expected in anyone with frostbite as peripheral temperatures low enough to permit frostbite are indicative of low overall body temperatures. Rapid rewarming is essential, but this should be reserved for an appropriate environment where there is no risk of refreezing. Extended freeze is preferable to refreezing, as refreezing carries significant morbidity. If prehospital warming is a necessity, the area can be placed in warm water or can be warmed with body heat, *e.g.*, in the axilla. Fires and stoves should not be used given the risk for burns in the insensate area. The frostbitten area should not be rubbed, and blisters should not be disturbed. The clinician may opt to pad or splint the effected area for transport to help prevent further injury. Ambulating on a frozen foot may result in avulsion fractures (13).

Optimally, an athlete with a frostbitten appendage should be evaluated in a proper medical facility. There, rapid rewarming is accomplished in a warm water bath or whirlpool, at 37°C to 39°C, taking approximately 20 min. Temperatures higher than 39° are often more painful and do not seem to decrease thaw times. Skin will become red or purple, and the process may be very painful, requiring analgesia. Reviewing the literature, there does not seem to be a clear consensus regarding management of frostbite-associated blisters. Some large bullae, however, may require subsequent debridement. Given the potential for short- and long-term wound care as well as the potential bony injury, an orthopedic consult is warranted in many cases. Tetanus prophylaxis should be provided if vaccination is not current, but there does not seem to be a role for prophylactic antibiotics. Topical aloe vera and nonsteroidal anti-inflammatory drugs are reasonable means of supportive care in the absence of contraindications (13).

Moving forward, athletes need to be especially cautious of cold exposure. For those with recent frostbite injury, cold exposure is contraindicated for 6 to 12 months, depending on the extent of the primary injury. Special equipment may need to be used during cold weather activity, often indefinitely, to avoid further damage to the area. Vaseline does not seem to offer any demonstrable benefit in prevention of frostbite. Chronic pain, scarring, arthritis, and neuropathy are all long-term sequelae of frostbite. Frostbite in children may result in necrosis of the bony epiphyses resulting in growth abnormalities (13).

Trench Foot

Trench foot is a result of repeated and constant immersion in cold water and has been reported in soldiers with as little as 12 h of exposure to cold wet environments but typically develops over longer periods of time. The pathophysiology is the same as that in early frostbite where there is increased vascular permeability and subsequent plasma leakage. Ice crystals do not form as this process largely thrives in 32°C to 50°C temperatures. Given the duration of time that it takes for trench foot to develop, it is unlikely to occur in most cold weather events with the exception of extended endurance events (13).

After persistent exposure, the extremity becomes hyperemic and eventually mottled, painful, and edematous. In mild cases, this process lasts 24 h while more severe cases may progress for days and produce areas of blistering and subsequent hypoperfusion. Skin breakdown may progress to gangrene if left untreated. The treatment involves removing the area from wet clothing and exposing it to dry warm air and, in severe cases, may require wound care. Milder cases of trench foot subside slowly over 1 to 4 wk and frequently are accompanied by a variably scarring exfoliation. Athletes with trench foot should not return to play until these issues have subsided (13).

Chilblains

Chilblains are local erythematous or cyanotic skin lesions that develop at ambient air temperatures of 32°F to 60°F after exposure time of about 1 to 5 h. The lesions may be vesicles, bullae, or plaques that typically occur on the hands and feet and are the result of the same pathophysiologic cascade of events at play in both frostbite and trench foot. The process is self-limited, and management again revolves around removing the individual from the cold environment. In extreme cases, basic wound care with dry bandages may be necessary (2) (Fig. 2).

Raynauds Phenomenon

Raynauds phenomenon is a reaction to cold or emotional stress resulting in hyperactivation of the sympathetic system causing extreme vasoconstriction of the peripheral blood vessels, leading to tissue hypoxia. Chronic recurrent cases of Raynauds phenomenon can result in atrophy of the skin, subcutaneous tissues, and muscle. In rare cases, it can cause ulceration and ischemic gangrene. It may be an isolated entity, or it can be secondary to systemic inflammatory diseases like scleroderma. According to the Framingham Study, it is felt to be more prevalent in women, occurring in 8% of females and 5% of males (20).

These ischemic events are episodic, and when exposed to cold, an athlete's fingers or toes will turn white and woody.

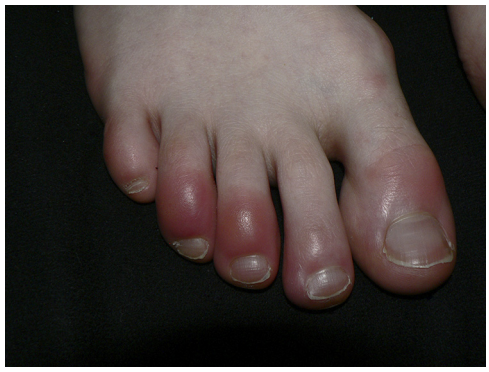

Figure 2: Chilblains are an inflammatory reaction initiated by cold exposure that results in damage to distal capillary beds. Athletes present with redness, itching, blisters, and, occasionally, ulcerations.

When the area is warmed and the blood flow returns, the skin becomes hyperemic before normal color is restored. Frequently, there is a period of swelling and dysesthesia. These ischemic events predispose an athlete to frostbite and other cold injuries.

Treatment involves removing the cold or stress and subsequent rewarming of the extremity. Some believe that increased hydrostatic pressure experienced while "windmilling" the arms can overcome the vasoconstriction facilitating the rewarming process. Decreasing caffeine and tobacco cessation are paramount to controlling symptoms. Choosing oral contraceptive pills that are low in estrogen also seems to help (20).

Cold Urticaria

Cold urticaria is, essentially, an allergic reaction to a cold exposure. The result is mast cell activation and degranulation resulting in hives, angioedema, and, in extreme cases, anaphylaxis. Some also experience respiratory, gastrointestinal, or cardiovascular symptoms following cold exposure. While hives are a nuisance, anaphylaxis and angioedema are life-threatening. Athletes with extreme cold urticaria are at risk with cold-weather sports, but those who swim in cold water are at risk of drowning. Avoidance of cold is the only true solution to the condition, but awareness of their temperature threshold may allow them to live a more normal life. A cold stimulation test is used to diagnose the disease. An antihistamine can be used to help control localized skin symptoms and itching. Those with anaphylaxis and angioedema should carry an EpiPen and consider having oral corticosteroids on hand for symptoms requiring treatment that are not severe enough to warrant epinephrine (11) (Fig. 3).

Prevention of Cold Injury

There are a number of factors that predispose athletes to hypothermia and cold injury. Obviously, it is always good to assess weather conditions before competing or training in cold environments. This allows proper preparation for the weather and may result in adjusting or cancelling the training agenda if extraordinarily harsh weather conditions are predicted. Avoiding stimulants, like coffee and tobacco, which alter the vasoactive responses to cold exposure, as well as drugs and alcohol, which can predispose the athlete to heat loss, are other ways to prevent hypothermia. The sports medicine physician should review all medications with cold-weather athletes on a regular basis. Athletes should be well hydrated and increase their intake of carbohydrates, the primary fuel of thermogenesis (3).

The single most important modifiable risk factor for cold injury prevention is appropriate clothing. The specifics of the number and thickness of layers are dictated by the activity and by other environmental factors, but the fundamentals of layering are universal. The base layer should be made of a thin, moisture-wicking material such as polypropylene or polyester. These fabrics transfer moisture away from the skin, preventing conductive heat loss, and permit it to move through the clothing system to the surface where it evaporates.

The middle layers should provide insulation, the amount required dictated by the ambient temperature and the athlete's degree of exertion. Synthetic fleeces or wool provide insulation while allowing moisture to migrate to the exterior. Dressing in multiple layers is superior to one thick layer as it traps heat between layers and allows the athlete to fine tune layer selection to minimize overheating. Overheating predisposes the athlete to both dehydration and excessive heat loss from increased sweating. Down is sometimes used as insulation in extreme cold but should be avoided in wet conditions as it loses its insulative properties when wet and matted. Synthetic downs, *e.g.*, PrimaLoft, are effective when wet.

The outer shell must have a wind- and water-resistant membrane, *e.g.*, Gore-Tex, which allows moisture from the inner layers to cross the membrane and evaporate into the environment. Appropriate head and hand wear is essential to the prevention of large amounts of heat loss, and in fast-paced sports like ski racing or in windy conditions, a hat and gloves with a wind-stopping membrane may be needed. Hats and face gators also should be able to protect ear lobes, noses, and cheeks from direct cold injury. Eyewear should be sport specific and is necessary to prevent eye injuries in windy or fast-paced sports like downhill skiing. Proper fitting footwear is essential to the prevention of frostbite, and socks should be changed frequently when wet to prevent conductive heat loss or direct cold injuries.

Exercise-Induced Bronchoconstriction

Exercise-induced bronchoconstriction (EIB) is defined as a transient and reversible narrowing of airways that occurs

Figure 3: Cold urticaria is an allergic reaction to a cold exposure. Athletes typically present with hives, as seen above, but should be watched for signs of anaphylaxis and angioedema.

as a result of exercise. Exercise is recognized widely as a precipitant of bronchoconstriction in individuals with asthma affecting as many as 80% with the diagnosis. However, the bulk of athletes experiencing EIB do not carry the diagnosis of asthma. Athletes most at risk for bronchoconstriction include those performing high-ventilation or endurance sports (*e.g.*, Nordic skiers and runners) and those who exercise in the cold (*e.g.*, figure skaters) (1).

Currently, the pathogenesis of EIB is understood poorly and somewhat controversial. Some believe that airway drying, as a result of the humidification of large volumes of cold dry air, secondary to the increased minute ventilation that accompanies exercise, stimulates the inflammatory cascade resulting in bronchoconstriction. Others feel that a similar process results from the heat loss that occurs during the warming and humidification process (16). In EIB, there are increased levels of bronchoconstriction mediators such as leukotrienes and histamine. Activation of lymphocytes and B cells results in expression of immunoglobulin E and the increased presence of eosinophils (15).

Clinically, athletes show signs and symptoms of bronchoconstriction that can begin after just 3 min of exercise and peaks after 10 to 15 min. Athletes with EIB will complain of cough, chest tightness, and shortness of breath. Stridor or hoarseness should raise the question of vocal cord dysfunction (16).

Diagnosis of EIB in the nonasthmatic athlete is challenging and is somewhat controversial. According to the American College of Chest Physicians, the initial work should include a detailed history and physical and spirometry with pre-/postbronchodilator responses. The history also should include information from coaches, teammates, and family as athletes are historically inaccurate at reporting symptoms. The differential diagnosis of exertional dyspnea includes a number of life-threatening cardiopulmonary conditions that should be considered and explored in any athlete with an abnormal physical examination or atypical symptoms. Often, more advanced tests like eucapnic hyperventilation testing or bronchoprovocation are needed to establish the diagnosis of EIB (16).

The management of EIB has been studied extensively, but most studies have included a large number of true asthmatic subjects. The accepted pharmacotherapy, for EIB in nonasthmatic subjects, is prophylactic β-2 agonist dosing 15 min prior to exercise producing peak bronchodilatation at 1 h and is effective for up to 3 h (16). Athletes should have their inhalers on the bench, and trainers should be facile in administration and in the evaluation of a person with asthma and his/her respiratory condition.

Long-acting β agonists are used in those with underlying asthma but are not recommended as monotherapy for EIB (15). When asthma control is poor, inhaled corticosteroids (ICS) are a necessity. In athletes with EIB and no underlying diagnosis of asthma, some benefit has been shown for the long-term use of ICS (9). Antileukotrienes (*e.g.*, montelukast) have been shown to provide significant protection against EIB as soon as 2 h after dosing and evidence of continual benefit for up to 24 h in athletes with a diagnosis of mild asthma (17,18). There is also limited data suggesting that mast cell stabilizers used before exercise reduce the severity and duration of EIB (19).

There are some behavioral modifications that may help athletes cope with EIB. Avoiding triggers and a dedicated warm-up routine have been shown to reduce episodes of EIB (16). Understanding that increased minute ventilation is a trigger for bronchoconstriction, athletes should be counseled that improved fitness (and thus reduced minute ventilation to achieve the same work) will help control symptoms. When exercising in cold air, breathing through a thin scarf may help reduce the work of warming and humidifying the air (15). There are data that suggest that diets rich in omega-3 fatty acids may reduce the levels of the inflammatory mediators implicated in EIB. After 3 wk, athletes supplemented with fish oil had improved pulmonary function testing and used a bronchodilator less (14).

Conclusions

As participation in cold weather and endurance sports increases, preventing, recognizing, and treating cold-induced injuries will become more prominent. Realizing that all cold injuries are preventable, sports medicine physicians have the opportunity to educate athletes, trainers, and coaches on injury prevention. Through discussion of proper dress, hydration, nutrition, and assessment of weather conditions, the physician can empower athletes to train and compete safely in cold environments. Through recognition of the signs and symptoms of early cold injury, team physicians will be able to evaluate and treat athletes before severe injury occurs allowing a more rapid return to play.

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