Environmental Illness in Athletes

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HEAT-RELATED ILLNESS

Introduction

Heat-related illness represents a spectrum of disorders that continues to be a significant problem in competitive athletes. Although more common in summer months, significant heat illness can occur even in moderate conditions [1]. Heatstroke, the most severe form of heat illness, is the third most common cause of death in high-school athletes, and is responsible for at least 240 deaths a year in the United States [2]. The recent tragic deaths of several high-profile athletes are solemn reminders that we need to continue educating physicians, trainers, coaches, and athletes on heat illness prevention and treatment.

Thermoregulation

The hypothalamus is the primary regulator of heat transfer in the body. At rest or when the ambient temperature is less than 68°F, most of the heat of the body is lost through conduction, convection, and radiation [3]. Conduction is the transfer of heat energy from warmer to cooler objects by direct contact. Convection means heat loss to air circulating around the body. Radiation is heat transfer by electromagnetic waves. During exercise or when the ambient temperature is above 68°F, most heat dissipation occurs at the skin surface by the evaporation of sweat. Evaporation is the most important heat-dissipating mechanism during exercise [3–5].

During maximal exercise, the muscles can produce 15 to 20 times more energy than at rest. Most of this energy is converted to heat, which quickly raises the core body temperature of the athlete. The core temperature will rise 1°C every 5 minutes without the body’s thermoregulatory system [6]. A rise in core temperature is sensed by thermodetectors in the hypothalamus, which initiate sweating and increase cutaneous blood flow. A 70 kg athlete can sweat 1 to 2 liters per hour during intense exercise, with larger athletes sweating considerably more. Each milliliter of evaporated sweat can consume about 0.6 kcal of heat; thus a well-conditioned athlete has the ability to dissipate well over 1000 kcal of heat per hour into the environment [3].

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After the heat-dissipating mechanisms are activated, the body’s core temperature reaches a plateau, where it remains until the exercise demand is past. In elite athletes, the equilibrium between heat production and heat dissipation may be as high as 104°F without diminished performance. If the heat-dissipating mechanisms fail or there is an overwhelming heat stress, the core temperature may continue to rise and produce a heat injury. Because the evaporation of sweat is the major heat-dissipating mechanism in exercising athletes, anything that hinders this process, such as humidity, dehydration, or inappropriate clothing, will increase the likelihood of heat-related illness [3,5].

**Acclimatization**

Acclimatization is the process by which the body adapts to exercising in a warmer environment. This process takes approximately 10 to 14 days, during which the body is exposed to the environment for 60 to 90 minutes per day while performing moderate exercise. When done appropriately, acclimatization will result in physiologic changes that will increase the plasma volume and rate of sweating, as well as allowing sweating to begin earlier. Additionally, the electrolyte content of sweat and urine decreases. Thus, an acclimatized athlete is able to exercise longer, at a higher level of exertion, while maintaining a lower core temperature for a given workload compared with an unacclimatized athlete [3,5,7,8]. Physiologic changes due to acclimatization can be summarized as follows [3]:

- Increased plasma volume
- Increased rate of sweating
- Increased cutaneous vasodilatation
- Decreased threshold for initiation of sweating
- Decreased electrolyte content of sweat
- Decreased heart rate at given work load and stress
- Increased aldosterone production/decreased urinary sodium excretion

**Risk Factors for Heat-Related Illness**

There are a multitude of factors that put an individual at risk for a heat-related illness. These risk factors can be categorized either as intrinsic or extrinsic risk factors. Intrinsic risk factors arise from current medical conditions or medication use, as well as fitness status and level of acclimatization. Extrinsic factors are those related to the environment and the activity being performed. These factors include the intensity and duration of physical activity, the environmental conditions (temperature and humidity), and the availability of water, rest and shade. Both intrinsic and extrinsic factors must be taken into account to accurately assess the risk of a heat-related illness [4,6,9]. Risk factors are outlined in Box 1.

**Spectrum of Heat-Related Illness**

Heat-related illness represents a wide spectrum of disease, from the mild annoyance of “prickly heat” to life-threatening exertional heatstroke (EHS). Although minor heat illness is relatively easy to treat, its occurrence should serve as a warning sign that conditions are favorable for more severe heat-related illness [7].
### Box 1: Risk factors for heat-related illness

**Intrinsic factors**

**General**
- History of previous heat illness
- Poor fitness
- Lack of acclimatization
- Obesity
- Inappropriate clothing
- Extremes of age (young and old)
- Sleep deprivation
- Mentality (desire to push self)

**Medications**
- Stimulants (increase heat production): amphetamines, methylphenidate (Ritalin), ephedra, alpha agonists, thyroid agonists
- Anticholinergic (decrease sweating): antihistamines, antidepressants, antipsychotics
- Cardiovascular (alter cardiovascular response): calcium channel blockers, beta blockers, diuretics, monoamine oxidase inhibitors (MAOI)
- Illegal drugs: cocaine, heroin, lysergic acid diethylamide (LSD), phencyclidine (PCP)
- Others: alcohol, laxatives

**Medical conditions**
- Febrile illness or upper respiratory infection (URI)
- Gastrointestinal (GI) illness
- Skin conditions (sunburn, rash)
- Dehydration or overhydration
- Sweat gland dysfunction
- Uncontrolled diabetes or hypertension
- Cardiac disease

**Extrinsic factors**
- Environmental conditions (temperature, humidity, wind, sun exposure)
- Intensity and duration of activity
- Lack of education and awareness of heat illness among coaches and athletes
- Poor access to shade and water
- Inappropriate work/rest ratios based on conditions
- Lack of emergency plan for treating heat-related illness
- Delay in recognition of early warning signs of heat-related illness
Minor Heat-Related Illness

*Miliaria rubra*

Miliaria rubra, or “prickly heat” is a pruritic papulovesicular skin eruption that occurs with exposure to high heat and humidity. It occurs as a result of occluded sweat glands on skin that is covered by clothing. The condition worsens as sweating continues. It is treated with cooling and drying the skin to avoid further sweating. It may take 7 to 10 days for the condition to resolve [7,10].

*Heat edema*

Heat edema represents mild dependent edema in the nonacclimatized individual. It is due to the increasing plasma volume that occurs with acclimatization. It resolves spontaneously as acclimatization occurs, but can be treated symptomatically with rest and elevation of the effected extremities [5,7,10].

*Sunburn*

Sunburn is thermal damage to the skin caused by ultraviolet rays. It is a significant risk factor for further heat-related illness because it decreases the cutaneous skin’s ability to transfer heat away from the body. It is best prevented by frequent and liberal application of a sunscreen (at least sun protection factor [SPF] 30) to all sun-exposed skin [3].

*Heat tetany*

Heat tetany is carpopedal spasm due to hyperventilation in response to acute exposure to overwhelming heat stress. It is treated by removing the athlete from the heat stress, cooling down, resting, and hydration. Breathing into a paper bag can also be useful [3].

*Heat cramps*

Heat cramps are muscle cramps that occur in the large muscles such as the quadriceps, hamstrings, gastrocnemius, and abdominals because of fluid and electrolyte depletion. Individuals who lose a great deal of sodium in their sweat (“salty sweaters”) are at higher risk. Heat cramps are treated with rest, cooling, stretching, and massage, along with rehydration using an electrolyte solution (sports drink). For athletes who have recurrent cramps (“crampers”), it may be helpful to encourage liberal use of salt in their diets and adding salty foods to their diet, such as pretzels and soups high in sodium, including chicken broth or soup [5,7,10].

*Heat syncope*

Heat syncope represents an orthostatic syncopal event due to peripheral vaso-dilatation and venous pooling that results from prolonged standing or rising suddenly from a seated position. It is treated with moving the individual to a cool area, elevating the legs, and oral rehydration [5,7,10].

Moderate Heat-Related Illness

Heat exhaustion is the most common form of heat-related illness [5]. It occurs in exercising athletes who have become dehydrated and thus are unable to appropriately dissipate their heat. Their core body temp is usually lower than
104°F. Affected individuals present with profuse sweating, weakness, malaise, headache, nausea, and at times vomiting, along with tachycardia and orthostatic hypotension. Mental status is relatively intact; however, emotional lability may be seen. Because heat-related illness is a spectrum of disease, it can be very difficult to differentiate between heat exhaustion and heatstroke on its initial presentation. Therefore, it is recommended to treat any significant heat-related illness as a heatstroke in order not to miss this very serious condition, which requires immediate cooling to decrease mortality [5,7,11].

Severe Heat-Related Illness

Exertional heatstroke

EHS is the most serious form of heat-related illness in athletes. The hallmark features of EHS are a core temperature higher than 104°F or 40.5°C and severe mental status impairment. EHS represents a condition of total thermoregulatory failure that will not spontaneously reverse itself without external cooling measures. It is a true medical emergency that requires immediate and urgent institution of external cooling measures in order to decrease morbidity and mortality. Mortality rates may exceed 10%, and are directly related to the length of time the core temperature remains elevated. Prompt recognition and immediate initiation of cooling are key factors for improving outcomes and decreasing mortality. The signs and symptoms are similar to those seen in heat exhaustion, with the addition of significant mental status impairment. Mental status changes may include ataxia, confusion, disorientation, psychotic behavior, seizures, and coma. Frequently these individuals have physically collapsed. Measurement of rectal temperature is essential to making the diagnosis in an athlete who collapses while exercising in the heat [5,7,11].

Pathophysiology of heat-related illness

Heat stress causes damage to the body by three different mechanisms: (1) it causes direct damage to cells by protein denaturation and interrupting cellular functions—exposure to temperatures above 41.6°C to 42°C (the critical thermal maximum), even for a few hours, can cause damage to the cells; (2) in response to significant heat stress, the body releases a number of inflammatory cytokines that cause systemic damage; and (3) significant temperature elevation can injure the vascular endothelium, increasing vascular permeability, and resulting in the activation of the coagulation cascade and subsequent development of disseminated intravascular coagulopathy [5]. When untreated, heatstroke can cause cardiac cellular damage, hepatic necrosis, rhabdomyolysis, disseminated intravascular coagulation, adult respiratory distress syndrome, and renal failure [1]. The key factors affecting morbidity and mortality of heatstroke are directly related to the degree of temperature elevation and the duration of exposure. Fortunately, current rates of heatstroke survival are 90% to 100% [1].

Treatment of exertional heatstroke

Treatment for EHS involves immediate cooling measures by whatever means possible while evaluating and stabilizing the airway, breathing and circulation...
Clothing should be removed and the patient moved to a cool or shaded area. Cold or ice water immersion is a cooling method that can lower core temperature rapidly. In the field, a small plastic portable swimming pool filled with water and ice can be used for this purpose [12]. If immersion is unavailable, then dousing with tepid or cool water and fanning with a towel or fan to speed evaporative cooling has also been shown to be very effective at decreasing core temperature. Using ice water-soaked towels and applying ice to the neck, axilla, and groin are other methods of cooling. The airway must be secured by appropriate means, to include endotracheal intubation if necessary. Breathing, circulation and core temperature should be continuously monitored, and intravenous (IV) access obtained to administer fluids and medications. Active cooling should continue until the core temperature reaches 38.8°C (101.8°F), then cooling measures should be slowed to avoid hypothermia. Transportation to a medical facility should be arranged as soon as possible.

Evaporative cooling and cold-water submersion methods have been compared and are likely equally effective. Due to availability and ease of application, evaporative cooling is more commonly used [7]. Medications to accelerate cooling have not been found to be helpful in treating heatstroke. Antipyretics have not been studied for use in heatstroke, but may be a topic of future study because pyrogenic cytokines have been shown to play a role in heat-stress injury [13].

Return to Play Following a Heat-Related Illness

Following an episode of EHS, an athlete’s ability to tolerate the heat may be temporarily or permanently compromised. Thus, it is important to have a graduated and monitored strategy for safely returning an athlete to play. This should be through the coordinated efforts of the athlete’s physician and an athletic trainer or qualified health professional. The Inter-Association Task Force on Exertional Heat Illness Consensus Statement has guidelines to assist with returning an athlete to play following EHS [9]. The guidelines include the following recommendations:

1. Physician clearance before returning to exercise
2. No exercise until the athlete is completely asymptomatic and all laboratory tests have normalized.
3. The severity of illness should dictate the length of recovery time.
4. The athlete should avoid exercise for a minimum of 1 week after release from medical care.

When athletes are ready to return to play, they should then cautiously begin a gradual return to physical activity under the supervision of an certified athletic trainer (ATC) or other qualified health professional. An example of schedule for returning to activity is

1. Easy to moderate exercise in a climate-controlled environment (several days), then increase to strenuous activity in the same conditions (several days)
2. Easy to moderate exercise in heat (several days), then increase to strenuous activity in the same conditions (several days)
3. Easy to moderate exercise in heat with equipment (several days), then increase to strenuous activity in the same conditions (several days)

Athletes are allowed to advance through each stage as they are able to tolerate the new level of activity and heat stress. Though this has not been well-studied, the ability to regain heat tolerance may take anywhere from several weeks to several months.

Prevention of Heat-Related Illness—Adapting to Exercise in the Heat

Preventing heat-related illness in athletes involves addressing several different risk factors related to heat illness. The most important modifiable factors for athletes include addressing issues of adequate hydration, accurate assessment of heat stress, appropriate clothing, and acclimatization [4].

Appropriate hydration

Mild dehydration is extremely common in athletes, and is often unavoidable because fluid replacement may not match fluids lost due to sweating during exercise. Even mild dehydration (<2%), however, can affect athletic performance and thermoregulatory function. Mild dehydration of 2% to 3% decreases work capacity by 15% to 20%. Optimal fluid replacement should match the fluid and electrolytes lost for each athlete on an individual basis during exercise. This is best accomplished by pre/post-exercise weighing. In this manner, the athlete will be able to estimate his/her fluid deficit based on weight loss following exercise. The athlete should consume 16 oz of fluid (preferably a carbohydrate/electrolyte drink) for each pound of weight loss. The athlete should not be allowed to return to exercising until appropriately rehydrated (baseline weight is restored). Replacing sodium after exercise is best accomplished by consuming salty foods in combination with a sports drink. This is especially important to those athletes who are salty sweaters and have recurrent muscle cramps while exercising in the heat [14].

Exertional hyponatremia. Hyponatremia (blood sodium level <130 mmol/L) can occur when athletes consume relatively more free water than necessary, or when sodium loss in the sweat is not adequately replaced. This condition was initially described in ultradistance runners and triathletes, but more recently has been reported in marathon runners as well [15–18]. Athletes at risk for this condition typically consume more fluid (usually water) than they lose in sweat, and typically gain weight over the course of an endurance race. These athletes are typically smaller in stature and have slower race times. The signs and symptoms of hyponatremia are similar to those of heat exhaustion, including malaise, fatigue, nausea, and vomiting. Vital signs including temperature are usually within normal limits, producing a baffling clinical picture. As the condition progresses, central nervous system (CNS) abnormalities become more significant, with altered levels of consciousness, confusion, coma, seizures and, in severe cases, death resulting from cerebral edema. The treatment for hyponatremia is prompt recognition and administration of sodium by mouth, or judicious IV
administration of 0.9% or 3% saline. Urgent transfer to the nearest medical facility should then take place for further monitoring and supportive care [18–20].

**Hydration guidelines.** Guidelines for optimizing hydration have been published in an effort to improve athletic performance and decrease the chance of heat-related illness. Optimal hydration must follow guidelines for hydration pre-exercise, during exercise, and post-exercise [14].

Current research supports the following recommendations:

1. **Pre-exercise hydration:** 500–600 ml of fluid (preferably carbohydrate/electrolyte sports drink) consumed 2–3 hours before exercise; then consume 200–300 ml of fluid 10–20 minutes before exercise.
2. **Hydration during exercise:** 200–300 ml of fluid (water or sports drink) should be consumed approximately every 10–20 minutes of exercise to minimize fluid losses.
3. **Post-exercise hydration:** approximately 500 ml of fluid (sports drink) should be consumed for every pound of weight loss during exercise.

These guidelines assume that athletes are hydrated at baseline and that they weigh themselves before exercising and after exercising.

**Recognizing and addressing heat stress**

Accurate measurement of environmental heat stress is essential for assessing the risk of heat-related illness. The gold standard for measuring heat stress is the wet bulb globe temperature (WBGT), which can be measured using commercially available devices. The WBGT measurement takes into account the contribution of ambient temperature, radiant heat, and humidity to determine heat stress using the following formula [1,6]:

\[
WBGT = 0.7 \text{ (wet bulb)} + 0.2 \text{ (dry bulb)} + 0.1 \text{ (globe)}.
\]

The WBGT measurement uses a chart to help determine the category of risk for heat-related illness. Changes in the scheduling of athletic activities can then take place based on the category of risk. If the athletic venue cannot be postponed, then medical personnel can proactively prepare by stocking up on supplies needed to treat and manage heat illness victims. These should include water, ice, towels, IV fluids, fans, and medical cots. Other ways of minimizing heat stress during athletic events are to provide shade or air-conditioned areas, provide frequent breaks and adequate access to water, and avoid exercise or activities during the hottest times of the day (10:00 AM–6:00 PM) [1,6,9]. WBGT risk categories are

- **Low risk** = <64°F or <18°C
- **Moderate risk** = 64°F–73°F or 18°C–23°C
- **High risk** = 73°F–82°F or 23°C–28°C
- **Hazardous** = >82°F or >28°C

**Appropriate clothing**

Clothing should be light in color, loose-fitting, and made from a lightweight, open-weave material that will maximize evaporation [6]. Because the face and
scalp account for 50% of the total sweat production, toweling off the face to renew the evaporative surface and removing headgear whenever possible are important adjuncts to effective heat dissipation [10].

**Acclimatization and conditioning**

The process of acclimatization results in physiologic changes that allow the athlete to exercise in the heat at lower physiologic stress. Acclimatization does not guarantee immunity from heat-related illness, but can improve the body’s ability to withstand a given heat load. It should be remembered that the hydration requirements of an athlete will actually increase with acclimatization because of the increased rate of sweating. Thus, meticulous attention to rehydration will continue to be extremely important [5,7,8].

**Discussion**

Heat-related illness is a spectrum of disorders that can be prevented with ongoing educational efforts. Coaches, trainers, athletes, and parents need to be continuously educated on key risk factors of heat illness in athletes. This includes identifying individual risk factors in an athlete, adhering to hydration guidelines, calculating the heat stress category, dressing accordingly, and making changes in activities when appropriate. These efforts will allow athletes to participate in sports more safely in hot environments [3].

**COLD INJURIES**

**Introduction**

Accompanying an increase in outdoor activities is the rising risk for cold injury. Unfortunately, the rising popularity of outdoor activities brings with it a naiveté about the threat of cold injuries [21]. Certain sports are more prone to cold injuries than others: those commonly performed in cold environments, such as skiing, bobsledding, dog sledding, and hiking; those involving water immersion, such as swimming, scuba diving, boating, and wind surfing; and those that require prolonged activity, such as running and biking.

Thoughtless preparation for cold conditions can result in significant harm and even death. Hypothermia affects athletes who remain in a cold environment for too long, either because of accidental injury or because of unexpected weather changes [22]. Prevention of cold injuries in sports is the responsibility of trainers, coaches, team physicians, and athletes, and requires careful planning or appropriate cancellation and rescheduling of events. All should be educated regarding the risks, prevention, and treatment of cold injuries and hypothermia.

**Hypothermia**

Hypothermia is defined as the cooling of the body’s core temperature to below 35°C or 95°F. It occurs when the body loses more heat than it generates. Hypothermia usually occurs in the sports arena due to an injury or accident that prevents the athlete from returning to warm shelter. Although uncommon, hypothermia can occur quickly after the finish of an endurance race, when an athlete has low serum glucose, depleted intravascular volume, and is dilated...
peripherally. More commonly, hypothermia affects inexperienced mountain hikers in situations where unpredictable changes in weather such as wind, rain, and snow can exhaust the novice and allow his core body temperature to spiral downwards to dangerous levels.

**Thermoregulation**
The human body needs to remain within 34°C to 40.5°C (95°F to 105°F) to retain normal organ function [23,24]. The hypothalamus is the main thermoregulatory center of the body for maintenance of temperature homeostasis. When there is a decrease in core temperature, the hypothalamus activates different mechanisms within the body to produce and conserve heat. Shivering produces heat and can raise the basal metabolic rate to two to five times normal. Heat production can also occur through nonshivering thermogenesis by increasing levels of thyroxine and epinephrine. Heat conservation occurs with peripheral vasoconstriction to keep warmed blood near the body’s core, and with the behavioral response of layering clothing for insulation [25,26].

**Heat Loss**
We lose heat through four basic physical methods: radiation, evaporation, convection, and conduction. Insensible heat loss primarily is through the skin, though feces and urine account for other forms of heat loss. Our body attempts to reduce heat loss by adjusting the circulation in the form of peripheral vasoconstriction in order to maintain homeostasis of crucial organs at the body’s core; however, vasoconstriction of the surface of the head is minimal. The amount of insulation an athlete wears greatly reduces heat loss. Thinner athletes are more prone to heat loss because they have less fat insulation. Adolescents are also more prone to heat loss because they are less likely to conserve their energy, and are more likely to dress inappropriately [21,25].

**Radiation**
Radiation involves the dispersal of heat to nearby objects. It accounts for greater than 50% of heat loss. This heat loss can be significantly reduced with appropriate clothing.

**Evaporation**
Evaporation of sweat from exposed skin is another mechanism of heat loss. This mechanism accounts for insensible heat loss, and can cause 20% to 30% heat loss to the environment [27]. Dehydration can occur if insensible water loss is not taken into account. In a cold environment, evaporative heat loss increases in the presence of wind chill and wet clothing, both of which can occur frequently in mountaineering at high altitudes.

**Convection**
Convection is a mechanism by which heat is transferred from the body to the air flowing around it. Generally, heat transfer is more significant when the air flow around the body is rapid. Convection is a significant factor in sports such as wind surfing, cycling, and running.
Conduction
Conduction is the transfer of heat from one object to another through physical contact. This type of heat loss is most important in water sports or immersion accidents, because of the water’s ability to act as a strong conductor. The conductivity of water is approximately 23 times that of air [28]. Thus, an immersion injury reduces the core body temperature more rapidly than convective loss in air. Conduction is a significant source of heat loss for persons lying or sitting on frozen surfaces for an extensive period of time.

Measuring Body Temperature
Measuring core body temperature is challenging. In high-risk colder conditions, a first aid kit should always have a low temperature-reading thermometer (one which reads below 32°C [90°F]). An oral temperature is unreliable because it is often affected by external factors; it is not accurate when food or liquid is ingested less than 30 minutes before measurement and it is only of value to rule out hypothermia when the oral temperature is greater than 35°C. The most reliable site for core temperature is the rectum. For accuracy, the rectal thermometer should be left in place for 3 minutes at a depth of 10 cm. If a thermometer is not available, the best but admittedly challenging way to assess core body temperature is to feel for cutaneous areas that are normally not cold, but it is important to remember that skin temperatures can be much lower than actual core temperature [28].

Determining the Severity of Hypothermia
Recognizing the signs and symptoms of hypothermia is critical in sports medicine. The clinical manifestations of an athlete who has hypothermia will differ depending on the degree of core temperature reduction. Table 1 summarizes the signs and symptoms of the varying degrees of hypothermia [29]. Be aware that there will be individual variation and overlap of signs and symptoms in hypothermic situations.

The Physiologic Response to Hypothermia
A multitude of physiologic processes become impaired or ineffective as the core temperature drops. As the body’s core temperature decreases, nerve conduction becomes delayed, resulting in lack of coordination and muscle stiffness. Shivering attempts to increase metabolic rate, but also decreases coordination and increases oxygen demand. The catecholamine response becomes significantly impaired, resulting in fatigue and decreased heart rate, respiratory rate, reflexes, and muscle coordination [27] Ultimately, metabolic acidosis occurs because of a combination of respiratory depression, anaerobic metabolism in the peripheral tissues, and renal impairment caused by dehydration and hypoperfusion [30]. Blood viscosity increases and platelets become hyper-reactive to fluid shear stress, increasing platelet aggregation [31]. Atrial dysrhythmias are common, but are less serious than ventricular dysrhythmias, which occur at colder temperatures. The most recognizable, but not pathognomonic, ECG feature of hypothermia is the J wave. The J wave is a positive deflection between the
QRS complex and the ST segment. The J wave is found in almost 80% of hypothermic individuals whose core temperature is less than 33°C (91.4°F) [32].

Managing Hypothermia
All athletes who feel cold both during and after exercise or who have altered sensorium due to colder conditions should be assessed for hypothermia. Initial assessment and determination of the severity of hypothermia are the most important steps toward patient recovery. The relative severity should be based on clinical characteristics. A rectal temperature should be measured as soon as practical. The first priority is to reduce further heat loss. A suspected hypo-

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Signs and symptoms of the varying degrees of hypothermia</th>
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<tbody>
<tr>
<td>Type of hypothermia</td>
<td>Mild</td>
</tr>
<tr>
<td>Temperature</td>
<td>33°C–35°C (91°F–95°F)</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Tachycardia, hypertension, vasoconstriction</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Tachypnea</td>
</tr>
<tr>
<td>Neurological</td>
<td>Lethargy, slight incoordination, mild dysarthria</td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>Shivering (sometimes uncontrollably), cold extremities, unable to perform fine movements of the hand.</td>
</tr>
<tr>
<td>Patient appearance</td>
<td>Pale, cool</td>
</tr>
<tr>
<td>Other</td>
<td>Increased urinary frequency (cold diuresis)</td>
</tr>
</tbody>
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*Abbreviation:* ECG, electrocardiogram.
thermic individual should be sheltered from a wet, cold, or windy environment. Wet clothes should be replaced with dry clothes, and the individual should be insulated with added garments and blankets. Exercise to generate heat is only helpful in mild hypothermia. Minimal handling is important for all hypothermic individuals who have altered mental status, in order to avoid causing cardiac arrhythmias. In these individuals, clothes should not be removed; insulating blankets should be placed over them instead. If possible, warm fluid support should be provided orally or IV. It is important to recognize that even comatose, asystolic individuals have been resuscitated successfully [21].

The Process of Rewarming
Rewarming hypothermic individuals occurs in three ways depending on the level of intervention. The least invasive rewarming method is passive rewarming. More invasive forms include external active rewarming and internal active rewarming. The choice of method will be based on the severity of hypothermia.

Passive rewarming
Passive rewarming is applicable to mild, moderate, and severely hypothermic individuals, and involves warming the patient by insulating him with clothing and blankets. Wet clothing should be removed to reduce evaporation and convective heat loss. If wet clothing cannot be removed, plastic should be used to cover an individual from the neck down. The whole body should be covered with insulating material, including the head. Passive rewarming is thought to be the safest, albeit slowest, way to rewarm in the field until a patient can be brought to an intensive care unit [33].

External active rewarming
Active external rewarming involves applying heat to the skin, and is most appropriate for treating mild hypothermia. It can be dangerous in moderate and severe hypothermia. Nonetheless, extremely cautious external active rewarming can be used to treat moderate hypothermia. Methods of active external rewarming include immersion of the torso in warmed water, a hot-water circulating blanket, forced-air warming systems, hot packs, and heat lamps. The newly developing arteriovenous anastomoses opening by immersion of the hands or feet into 45°C(113°F) water carries promise, but is still being investigated [34]. Active external rewarming can be complicated by an “afterdrop” in body temperature caused by the return of cold peripheral blood into the circulation [35–37]. This complication can be minimized by using passive rewarming before active rewarming [25].

Internal active rewarming
Internal active rewarming can only occur in a hospital setting. This form of rewarming is necessary for severely hypothermic individuals, and requires the attention of an intensive care unit with experienced staff. Methods of internal active rewarming include cardiopulmonary femoral bypass, hemodialysis, heated intravenous fluids, extracorporeal blood warming, peritoneal dialysis, and humidified oxygen respiratory warming. Thoracic cavity lavage, and
GI, colonic, or bladder lavage with warm fluids have also been used with limited success [30].

**General Measures for Treating Hypothermia**

The treatment of hypothermia is guided by the severity of hypothermia. It is important to realize that hypothermia may result in death in 2 hours. General measures include establishing the ABCs of basic life support. Intubate only when absolutely necessary, because intubation can bring about reflexive bradycardia and can further exacerbate cardiac instability. A patient should be supplied with warm humidified oxygen for respiratory rewarming. The rate of warming should be 0.5°C to 2°C/hour, because more rapid rewarming can cause ventricular fibrillation [38]. Monitoring vital signs and core body temperature is of utmost importance. In moderate to severe cases, cardiac rhythm should be monitored as well.

**Treating Immersion Hypothermia**

Athletes who slowly develop hypothermia after extended immersion begin to lag in their performance and start to show signs of hypothermia. Immersion accidents, however, usually cool the victim rapidly and minimize the ability for slow adaptation to the cold environment. Patients suspected of immersion hypothermia should be removed from the water in the horizontal position, with special attention being paid to cardiovascular stability. The change in hydrostatic pressure upon removal from water can further exacerbate cardiovascular instability and can cause arrhythmias. In patients whose rectal temperature is greater than 32°C and for whom immersion occurred for less than 3 hours, rapid rewarming can begin, because significant fluid shifts have not occurred. Those patients who have rectal temperatures below 31°C should be treated for severe hypothermia [28].

**Treating Mild Hypothermia**

Mild hypothermia (33°C–35°C or 91°F–95°F) is treated to a large degree with common sense in the form of passive rewarming. The patient should be removed from the cold environment, warmed, dried, insulated, and given warm sweet beverages. Alcohol is counterproductive in all settings of hypothermia. Heat should be applied to the torso to reduce the risk of hypovolemia due to peripheral vasodilation. Administration of heated IV fluids to 40°C can be helpful [39].

**Treating Moderate Hypothermia**

Active external rewarming is used to treat moderate hypothermia (31°C–32°C or 88°F–90°F), with caution to prevent skin burns; however, active rewarming should not begin until core temperature is greater than 34°C (93°F) [28]. The same principles apply as in mild hypothermia: remove the patient from the environment and insulate him. Until the core temperature exceeds 34°C, a patient should only be treated with passive rewarming. When possible, the patient should be closely monitored for cardiovascular decompensation. Though moderate hypothermia is not thought to be life-threatening, the treating
physician should be aware that the progression to severe hypothermia with risk of death can occur.

**Treating Severe Hypothermia**

The treatment of severe hypothermia (<31°C or <88°F) needs to be both aggressive and extremely cautious. On the aggressive side, physicians need to monitor patients diligently in an intensive care setting with frequent blood sampling, and they should use active internal core rewarming methods; however, caution must be exercised in moving the patient as little as possible to avoid precipitating ventricular fibrillation, which is resistant to pharmacologic or electrical cardioversion without prior core rewarming [39]. Rewarming should never exceed 2°C per hour, because a higher rate can cause ventricular fibrillation and hypovolemic shock [38]. Recent evidence suggests that bretylium tosylate is the drug of choice for conversion if the patient is warmed [40]. Cardiopulmonary resuscitation (CPR) should be used only with extreme caution, because external cardiac massage can cause ventricular fibrillation. Indications for CPR are that: (1) no carotid pulse is detectable for at least 1 minute, or cardiac arrest is observed whereby a pulse disappears or cardiac arrest occurred within 2 hours, and (2) that CPR will be able to be continued with only brief periods of interruption [41].

Broad-spectrum antibiotics are not necessary unless infection appears to be present and a patient fails to warm or has infiltrates on lung films [27]. Corticosteroids should not be delivered unless a patient is determined to have underlying corticosteroid deficiency. Blood pH, PCO₂, glucose, and potassium should be followed during rewarming. The return of peripherally pooled lactic acid into the circulation and the increased solubility of carbon dioxide can cause metabolic acidosis and subsequent electrolyte abnormalities. Fluid shifts can also cause electrolyte abnormalities. Positive pressure ventilation can be helpful in a severely hypothermic patient, but artificial ventilation can cause reflex bradycardia, and should be used only when absolutely necessary.

**Frostbite**

Frostbite occurs when tissue freezes and crystals form in the extracellular space between cells. Frostbite occurs at ambient temperature below 32°F (0°C) [42,43]. With dehydration, vasoconstriction, and low epidermal temperature, circulation is limited as blood viscosity increases [44], and water hydrostatically pulled out of cells begins to freeze. Frostbite usually affects exposed areas and the distal extremities, including the nose, ears, face, fingers, and toes. In runners and skaters, the scrotum and penis may be involved because of inadequate insulation in these areas. Corneal freezing can affect skiers and speed skaters. Close to 60% of frostbite injuries involve the lower extremities, in particular the great toe and feet [45]. Predisposing diseases can include Raynaud’s disease, peripheral vascular disease, and diabetes mellitus. Other factors which can increase the likelihood of developing frostbite include tobacco smoking and constrictive clothing [46].
Box 2: Cold injury prevention

Plan

Plan for the threat of hypothermia, especially in cold, windy, and wet conditions; and bring appropriate equipment, such as a low-reading thermometer, wool blankets, changes of dry clothing, and warmed beverages. Alcoholic beverages are discouraged.

Check weather forecasts, keeping in mind rain and wind-chill factors.

Athletes should never be alone, and should carry on activities in nonisolated areas. If athletes are in an isolated area, they should make sure others are with them.

If an athlete cannot prevent being alone, she should appoint a contact person to be called when an activity is complete. The contact person should be aware of the athlete’s whereabouts during the activity before the athlete begins; this allows an athlete to be found if injured.

Schedule events in appropriate seasons and be familiar with cancellation policies. For example, the American College of Sports Medicine recommends that if the dry ambient temperature is < 20°C (< 4°F), race directors should consider rescheduling a race [44].

Communicate

Always alert athletes about weather forecasts so they can dress appropriately.

Athletes should communicate plans to others, especially if venturing out in isolated areas.

Athletes should carry communication devices such as cell phones or walkie-talkies whenever possible.

Limit heat loss by insulating and dressing appropriately [28]

Many layers are better than one thick layer.

When competition in cold conditions is anticipated, increase subcutaneous fat if low reserves are present. Waterproofed outer clothing is essential. Clothing materials should be wool, wool blends, or polypropylene. Cotton is not recommended.

All extremities and the head should be covered. The face should be covered, especially with high wind chill. Goggles should be worn by skiers to prevent corneal freezing.

The feet should be protected with two layers of socks when running in cold climates. The first layer should be made of polypropylene and the second layer made of wool [28].

Promote hydration by encouraging consumption of fluids, even when athletes are not thirsty

Prevent exhaustion

Ensure appropriate fitness for level of activity.

Be aware of increased risk of hypothermia with fatigue.

Maximize heat production by maximizing glycogen stores when preparing for distance events
Clinical features of frostbite

“Frostnip” can initially appear as frostbite, but without irreversible tissue damage. In frostnip, exposed painful skin blanches and gradually loses sensation but remains pliable. With gentle rewarming, the frostnip-affected area becomes hyperemic, and the sensation of pain returns rapidly. In frostbite, the tissues are hard, insensitive, and white or mottled [41]. Frostbite can be superficial, involving only epidermal and surface tissues, or deep, involving full-thickness damage to cartilage, bone, and nerves.

Frostbite treatment

Never begin to thaw an area of frostbite if there is any chance of refreezing, because refreezing will cause more damage to the tissue [47]. Also, never rewarm a frostbitten area by rubbing, because friction also increases the risk of further damage and can result in extensive tissue loss. The best way to rewarm is in a 40°C whirlpool. If this is not available, spontaneous passive rewarming can be successful. During rewarming, sensation returns with hyperemia and can be quite painful. Blisters can evolve over many affected areas and require tetanus prophylaxis, analgesics, and appropriate antibiotic therapy [48]. Demarcation of the edges of the affected area can be difficult, because deep injury can occur under normal-appearing skin. For this reason, debridement or amputation should be delayed for up to 90 days, at which point mummification and demarcation are complete [21]. Injury from frostbite after healing can result in cold hypersensitivity, decreased sensation, hyperhydrosis, and general autonomic dysfunction in affected extremities [49,50]. Frostbite pathophysiology has been recently reviewed by Sallis and Chassay [39].

Prevention of Cold Injuries

Cold injuries in sports are largely preventable. General guidelines are provided by numerous references and are summarized in Box 2.
Discussion
Taking appropriate measures to prevent hypothermia and frostbite in colder conditions is a necessity. Proper preparation can reduce the risk of cold injury, whether or not accidental injury or immersion occurs. One should never forget that moderate and severe hypothermia are medical emergencies, and that mild hypothermia can progress to these levels easily. Prevention of cold injuries in sports is the responsibility of trainers, coaches, team physicians, and athletes.

HIGH-ALTITUDE ILLNESS
Introduction
High altitude presents unique environmental challenges to athletes and non-athletes alike. With increasing altitude, barometric pressure falls in a logarithmic fashion. Falling pressure is accompanied by decreases in the partial pressure of oxygen. At 20,000 feet, the barometric pressure is one half that at sea level; on the summit of Mount Everest (29,029 feet) the partial pressure of oxygen is only 28% of that at sea level [51].

Although decreased barometric pressure and hypoxia are the primary challenges posed by high altitude, they are not the only ones. Temperature also decreases with altitude, leading to a significant risk of cold injuries. Ultraviolet light exposure increases as the atmosphere thins, so climbers must be attentive to the potential for sunburn, skin cancer, and snowblindness. Finally, increased water losses combined with difficulty obtaining water in the so-called “high-altitude desert” can result in serious dehydration.

Given these physiologic challenges, it is not surprising that high-altitude illness is fairly common. The World Health Organization (WHO) estimates that 140 million people live permanently at altitudes greater than 8000 feet [52]. Many people travel to high-altitude regions for recreation and competitive sports. Military personnel are frequently deployed to high-altitude countries around the world. Study of acute change in altitude found that 22% of the studied population developed acute mountain sickness at altitudes of 7000 to 9000 feet, and 42% at altitudes of 10,000 feet [53].

Risk factors for the development of high-altitude illness include rate of ascent, altitude reached, and sleeping altitude. A prior history of high-altitude illness and permanent residence at low altitude also increase risk [52]. Persons more than 50 years of age seem to be somewhat less susceptible, but the incidence in children is similar to that in young adults [54]. Notably, exertion increases the risk of high-altitude illness; unfortunately, physical fitness is not protective [52].

Strictly defined, high-altitude syndromes include illnesses directly attributable to hypobaric hypoxia [51]. Considerable overlap exists between the various syndromes, but it is useful conceptually to divide them into two categories: neurologic syndromes and pulmonary syndromes. The neurologic syndromes include high-altitude headache, acute mountain sickness, and high-altitude cerebral edema (HACE). Pulmonary syndromes include acute hypoxia and high-altitude pulmonary edema (HAPE).
Neurologic Syndromes

Definitions and diagnosis

The neurologic high-altitude syndromes exist in a continuum from isolated high-altitude headache to HACE. In the middle lies acute mountain sickness. High-altitude headache is the most common form of high-altitude illness, and presents as headache in an unacclimatized person who has recently traveled to a high-altitude area. It may or may not progress to acute mountain sickness. The cause of high-altitude headache remains unclear, but it appears to be multifactorial, with a variety of mechanical and chemical pathways contributing [52].

Acute mountain sickness (AMS) is defined as the presence of high-altitude headache plus one or more additional symptoms. Common symptoms include GI symptoms (anorexia, nausea, vomiting), dizziness, fatigue, and sleep disturbance [54]. Symptoms typically begin 6 to 12 hours after arrival at high altitude. Importantly, these symptoms are very nonspecific and can be easily confused with many other conditions, such as gastroenteritis, alcohol hangover, migraine, and dehydration. By definition, the physical examination is normal. Any abnormal neurologic findings point toward the development of cerebral edema.

HACE is the end stage of acute mountain sickness. It is a clinical diagnosis defined by the onset of altered consciousness or ataxia in someone who has acute mountain sickness or HAPE [54]. Drowsiness is common and may progress to stupor. Physical examination often reveals papilledema or retinal hemorrhages. Cranial nerve palsies may occasionally be seen, but global encephalopathy is more common than focal findings. The natural course of HACE is rapid progression to death due to cerebral herniation. The time course can vary from hours to days.

Treatment and prophylaxis

High-altitude headache has historically been treated with nonsteroidal anti-inflammatory drugs (NSAIDs). The most commonly used regimen is ibuprofen 400 to 600 mg once, to be repeated as needed [54]. Recently, a randomized, controlled trial was conducted at Mount Everest base camp (altitude 13,580 feet) that found that 1000 mg of acetaminophen is as effective as 400 mg of ibuprofen in reducing pain from high-altitude headache [55]. This provides a safe option for patients who have a contraindication to NSAID therapy. Some studies have found sumatriptan (Imitrex) to be effective for the acute treatment of high-altitude headache, but other studies have failed to show this same benefit [51]. Interestingly, oxygen is often immediately effective for high-altitude headache.

Aspirin is the most common drug used for prophylaxis of high-altitude headache. The typical regimen is 325 mg every 4 hours for a total of three doses, starting 1 hour before arrival at high altitude. A randomized, controlled trial of 29 volunteers was performed in 1998 to evaluate the efficacy of this regimen, and those who took aspirin had fewer headaches.
Moreover, the headaches in the aspirin group developed at lower levels of arterial oxygen saturation than those in the control group, suggesting that aspirin raises the headache threshold by increasing tolerance to hypoxemia [56].

AMS requires additional measures for treatment and prophylaxis. Once it develops, ascent must be stopped. Mild cases may improve with rest and acclimatization at the current altitude, but the cornerstone of treatment for moderate or severe AMS is descent. Even a small descent of 400 to 500 meters can be sufficient to relieve symptoms [52]. If descent is impossible, it may be simulated using a portable hyperbaric chamber. Oxygen is the other traditional treatment, typically administered at low flow rates of 1 to 2 L/minute.

Pharmacotherapy can also be used to treat AMS, especially if descent is impossible or oxygen is unavailable. Acetazolamide is administered in doses of 125 to 250 mg twice daily, although little evidence exists as to the optimal dose. Dexamethasone can also be given (4 mg orally or intramuscularly every 6 hours). No studies exist to guide choice of medication, and combinations of descent, oxygen, and the above medications are typically used. If the patient develops signs of HACE, immediate descent becomes crucial, and emergency evacuation may become necessary. Oxygen should be given at higher flow rates of 2 to 4 L/minute. Dexamethasone is considered standard treatment, and doses may be increased to 8 mg intramuscularly or intravenously [54]. If descent is going to be delayed, acetazolamide should also be given.

Given the potential severity of AMS and HACE, considerable research has gone into prophylaxis. It has been well-demonstrated that slowing the rate of ascent prevents many cases of AMS. Guidelines suggest that above an altitude of 8000 feet, the altitude at which one sleeps should not be increased by more than 2000 feet in 24 hours [54]. If direct transport is necessary to high altitudes, spending a night at an intermediate altitude can be beneficial.

Pharmacotherapy is also commonly employed in the prevention of AMS and HACE. A systematic review of the literature was performed in 2000 that identified 33 relevant trials [57] and found evidence of efficacy for both acetazolamide and dexamethasone. Prophylactic dexamethasone in daily doses of 8 to 16 mg is very effective; fewer than three subjects need to be treated to prevent one case of acute mountain sickness. Interestingly, the most common daily dose of acetazolamide (500 mg) is no more effective than placebo, but higher doses (750 mg/day) are as efficacious as dexamethasone.

Neither dexamethasone nor acetazolamide is without side effects. Two trials of dexamethasone were stopped early due to reports of depression in the dexamethasone group [57]. Acetazolamide can cause increased diuresis and paresthesias. These concerns have led to a search for natural alternatives. Recently, several small trials showed gingko biloba to be beneficial for prophylaxis. Unfortunately, a large randomized, controlled trial of 614 Himalayan trekkers has subsequently found gingko to be no better than placebo for the prevention of acute mountain sickness [58]. This same trial confirmed the efficacy of acetazolamide prophylaxis.
Pulmonary Syndromes

Definitions and diagnosis

Although acute hypoxia is possible when climbers ascend extremely rapidly, it is more commonly encountered in aviation medicine [51]. The most well-defined high-altitude pulmonary syndrome is HAPE. Importantly, it accounts for most of the deaths from high-altitude illness. In addition to the general risk factors for high-altitude illness, cold is a specific risk factor for HAPE, because it increases pulmonary-artery pressure through sympathetic stimulation [54]. Pre-existing cardiopulmonary disease is also a specific risk factor.

HAPE usually begins with a dry cough and decreased exercise tolerance. Because these symptoms can be mild, a high degree of suspicion is needed for early diagnosis. Symptoms typically begin the second night at a new altitude and rarely occur after 4 days at a given altitude, due to adaptation of the pulmonary vasculature [54]. As the illness progresses, resting tachycardia and tachypnea become more prominent. Late in the illness, pink or bloody sputum can be seen, and respiratory distress develops. Fever is common, as are CNS symptoms, because there is a significant overlap with acute mountain sickness and HACE (50% of those who have HAPE have acute mountain sickness, and 14% have HACE) [54].

Physical examination reveals rales, often originating in the right axilla but spreading bilaterally as the disease progresses. EKG shows sinus tachycardia, and may also show right ventricular strain. Chest radiograph typically reveals a normal-sized heart, but prominent pulmonary arteries and patchy infiltrates. If arterial blood gas is obtained, severe hypoxemia is usually found.

Treatment and prophylaxis

Correcting hypoxemia is the most important treatment in patients who have HAPE. This is accomplished with supplemental oxygen, which also acts to decrease pulmonary-artery pressure, reversing the influx of edema into the lungs. Descent is the other mainstay of treatment. When medical care is available, mild-to-moderate HAPE can be treated successfully with rest and 48 to 72 hours of supplemental oxygen [54]. Endotracheal intubation can be required in severe cases. If death does occur, it is usually in the context of additional exertion after the development of edema or failure to obtain prompt medical care.

If supplemental oxygen is not available and descent is impossible, medication can be used to treat HAPE. Nifedipine has been shown to decrease pulmonary-artery pressure and is commonly recommended for this condition [59]. It is given in oral doses of 10 to 30 mg, and patients must be monitored for hypotension and reflex tachycardia. Nitric oxide has also generated interest as a potential treatment for HAPE. Nitric oxide has been shown to improve arterial oxygenation and decrease pulmonary artery pressure [60], but its clinical applications are not yet clear.

The prevention of HAPE depends again on gradual ascent and avoidance of overexertion. Climbers who have developed HAPE in the past may be...
candidates for prophylaxis with nifedipine (20 to 30 mg of extended release preparation every 12 hours) [54]. Most recently, a randomized controlled trial of 37 patients susceptible to HAPE [61] looked at salmeterol for prophylaxis. Subjects used the inhaler twice daily, and the incidence of HAPE was reduced from 74% in the control group to 33% with salmeterol. Thus, salmeterol is a safe and effective option for the prevention of HAPE, although it is not yet clear who should be offered prophylaxis.

References