National Athletic Trainers’ Association Position Statement: Exertional Heat Illnesses

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Objective: To present recommendations for the prevention, recognition, and treatment of exertional heat illnesses and to describe the relevant physiology of thermoregulation.

Background: Certified athletic trainers evaluate and treat heat-related injuries during athletic activity in “safe” and high-risk environments. While the recognition of heat illness has improved, the subtle signs and symptoms associated with heat illness are often overlooked, resulting in more serious problems for affected athletes. The recommendations presented here provide athletic trainers and allied health providers with an integrated scientific and practical approach to the prevention, recognition, and treatment of heat illnesses. These recommendations can be modified based on the environmental conditions of the site, the specific sport, and individual considerations to maximize safety and performance.

Recommendations: Certified athletic trainers and other allied health providers should use these recommendations to establish on-site emergency plans for their venues and athletes. The primary goal of athlete safety is addressed through the prevention and recognition of heat-related illnesses and a well-developed plan to evaluate and treat affected athletes. Even with a heat-illness prevention plan that includes medical screening, acclimatization, conditioning, environmental monitoring, and suitable practice adjustments, heat illness can and does occur. Athletic trainers and other allied health providers must be prepared to respond in an expedient manner to alleviate symptoms and minimize morbidity and mortality.

Key Words: heat cramps, heat syncope, heat exhaustion, heat stroke, hyponatremia, dehydration, exercise, heat tolerance

Heat illness is inherent to physical activity and its incidence increases with rising ambient temperature and relative humidity. Athletes who begin training in the late summer (e.g., football, soccer, and cross-country athletes) experience exertional heat-related illness more often than athletes who begin training during the winter and spring.1–5 Although the hot conditions associated with late summer provide a simple explanation for this difference, we need to understand what makes certain athletes more susceptible and how these illnesses can be prevented.

PURPOSE

This position statement provides recommendations that will enable certified athletic trainers (ATCs) and other allied health providers to (1) identify and implement preventive strategies that can reduce heat-related illnesses in sports, (2) characterize factors associated with the early detection of heat illness, (3) provide on-site first aid and emergency management of athletes with heat illnesses, (4) determine appropriate return-to-play procedures, (5) understand thermoregulation and physiologic responses to heat, and (6) recognize groups with special concerns related to heat exposure.

ORGANIZATION

This position statement is organized as follows:

1. Definitions of exertional heat illnesses, including exercise-associated muscle (heat) cramps, heat syncope, exercise (heat) exhaustion, exertional heat stroke, and exertional hyponatremia;
2. Recommendations for the prevention, recognition, and treatment of exertional heat illnesses;
3. Background and literature review of the diagnosis of exertional heat illnesses; risk factors; predisposing medical conditions; environmental risk factors; thermoregulation, heat acclimatization, cumulative dehydration, and cooling therapies;
Table 1. Signs and Symptoms of Exertional Heat Illnesses

<table>
<thead>
<tr>
<th>Condition</th>
<th>Sign or Symptom*</th>
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<tbody>
<tr>
<td>Exercise-associated muscle (heat) cramps</td>
<td>Dehydration</td>
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<tr>
<td></td>
<td>Thirst</td>
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<td></td>
<td>Sweating</td>
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<td></td>
<td>Transient muscle cramps</td>
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<td>Fatigue</td>
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<tr>
<td>Heat syncope</td>
<td>Dehydration</td>
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<td>Fatigue</td>
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<td></td>
<td>Tunnel vision</td>
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<td>Pale or sweaty skin</td>
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<td>Decreased pulse rate</td>
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<td>Dizziness</td>
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<td></td>
<td>Lightheadedness</td>
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<tr>
<td>Fainting</td>
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<tr>
<td>Exercise (heat) exhaustion</td>
<td>Normal or elevated body-core temperature</td>
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<tr>
<td></td>
<td>Dehydration</td>
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<td></td>
<td>Dizziness</td>
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<td>Lightheadedness</td>
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<td></td>
<td>Syncope</td>
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<td>Headache</td>
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<td></td>
<td>Nausea</td>
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<td></td>
<td>Anorexia</td>
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<td>Diarrhea</td>
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<td></td>
<td>Decreased urine output</td>
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<td></td>
<td>Persistent muscle cramps</td>
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<td>Pallor</td>
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<td>Profuse sweating</td>
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<td>Chills</td>
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<td>Cool, clammy skin</td>
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<td></td>
<td>Intestinal cramps</td>
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<td></td>
<td>Urge to defecate</td>
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<td></td>
<td>Weakness</td>
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<td></td>
<td>Hyperventilation</td>
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<tr>
<td>Exertional heat stroke</td>
<td>High body-core temperature (&gt;40°C [104°F])</td>
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<tr>
<td></td>
<td>Central nervous system changes</td>
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<tr>
<td></td>
<td>Dizziness</td>
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<td>Drowsiness</td>
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<td></td>
<td>Irrational behavior</td>
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<td>Confusion</td>
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<td>Irritability</td>
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<td>Emotional instability</td>
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<td>Hysteria</td>
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<td>Apathy</td>
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<td>Aggressiveness</td>
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<td>Delirium</td>
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<td>Disorientation</td>
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<td>Staggering</td>
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<td>Seizures</td>
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<td>Loss of consciousness</td>
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<td>Coma</td>
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<td>Dehydration</td>
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<td></td>
<td>Weakness</td>
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<td>Hot and wet or dry skin</td>
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<td></td>
<td>Tachycardia (100 to 120 beats per minute)</td>
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<td>Hypotension</td>
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<td></td>
<td>Hyperventilation</td>
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<tr>
<td></td>
<td>Vomiting</td>
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<tr>
<td></td>
<td>Diarrhea</td>
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<tr>
<td>Exertional hyponatremia</td>
<td>Body-core temperature &lt;40°C (104°F)</td>
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<tr>
<td></td>
<td>Nausea</td>
</tr>
<tr>
<td></td>
<td>Vomiting</td>
</tr>
</tbody>
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Table 1. Continued

<table>
<thead>
<tr>
<th>Condition</th>
<th>Sign or Symptom*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extremity (hands and feet) swelling</td>
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<tr>
<td>Low blood-sodium level</td>
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<tr>
<td>Progressive headache</td>
<td></td>
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<tr>
<td>Confusion</td>
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<td>Significant mental compromise</td>
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<td>Lethargy</td>
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<td>Altered consciousness</td>
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<td>Apathy</td>
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<td>Pulmonary edema</td>
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<tr>
<td>Cerebral edema</td>
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<tr>
<td>Seizures</td>
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<tr>
<td>Coma</td>
<td></td>
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</tbody>
</table>

*Not every patient will present with all the signs and symptoms for the suspected condition.

4. Special concerns regarding exertional heat illnesses in prepubescent athletes, older athletes, and athletes with spinal cord injuries;
5. Hospitalization and recovery from exertional heat stroke and resumption of activity after heat-related collapse; and
6. Conclusions.

DEFINITIONS OF EXERTIONAL HEAT ILLNESSES

The traditional classification of heat illness defines 3 categories: heat cramps, heat exhaustion, and heat stroke. However, this classification scheme omits several other heat- and activity-related illnesses, including heat syncope and exertional hyponatremia. The signs and symptoms of the exertional heat illnesses are listed in Table 1.

Heat illness is more likely in hot, humid weather but can occur in the absence of hot and humid conditions.

Exercise-Associated Muscle (Heat) Cramps

Exercise-associated muscle (heat) cramps represent a condition that presents during or after intense exercise sessions as an acute, painful, involuntary muscle contraction. Proposed causes include fluid deficiencies (dehydration), electrolyte imbalances, neuromuscular fatigue, or any combination of these factors.

Heat Syncope

Heat syncope, or orthostatic dizziness, can occur when a person is exposed to high environmental temperatures. This condition is attributed to peripheral vasodilation, postural pooling of blood, diminished venous return, dehydration, reduction in cardiac output, and cerebral ischemia. Heat syncope usually occurs during the first 5 days of acclimatization, before the blood volume expands, or in persons with heart disease or those taking diuretics. It often occurs after standing for long periods of time, immediately after cessation of activity, or after rapid assumption of upright posture after resting or being seated.

Exercise (Heat) Exhaustion

Exercise (heat) exhaustion is the inability to continue exercise associated with any combination of heavy sweating, dehydra-
Exertional heat stroke is an elevated core temperature (usually \( >40^\circ C \) \[104^\circ F \]) associated with signs of organ system failure due to hyperthermia. The central nervous system neurologic changes are often the first marker of exertional heat stroke. Exertional heat stroke occurs when the temperature regulation system is overwhelmed due to excessive endogenous heat production or inhibited heat loss in challenging environmental conditions and can progress to complete thermoregulatory system failure.19,21 This condition is life threatening and can be fatal unless promptly recognized and treated. Signs and symptoms include tachycardia, hypotension, sweating (although skin may be wet or dry at the time of collapse), hyperventilation, altered mental status, vomiting, diarrhea, seizures, and coma.5,6,10,14 The risk of morbidity and mortality is greater the longer an athlete’s body temperature remains above \( 41^\circ C \) \[106^\circ F \] and is significantly reduced if body temperature is lowered rapidly.22–24

Unlike classic heat stroke, which typically involves prolonged heat exposure in infants, elderly persons, or unhealthy, sedentary adults in whom body-heat-regulation mechanisms are inefficient,25–27 exertional heat stroke occurs during physical activity.28 The pathophysiology of exertional heat stroke is due to the overheating of organ tissues that may induce malfunction of the temperature-control center in the brain, circulatory failure, or endotoxicosis (or a combination of these).29,30 Severe lactic acidosis (accumulation of lactic acid in the blood), hyperkalemia (excessive potassium in the blood), acute renal failure, rhabdomyolysis (destruction of skeletal muscle that may be associated with strenuous exercise), and disseminated intravascular coagulation (a bleeding disorder characterized by diffuse blood coagulation), among other medical conditions, may result from exertional heat stroke and often cause death.25

Exertional hyponatremia is a relatively rare condition defined as a serum-sodium level less than 130 mmol/L. Low serum-sodium levels usually occur when activity exceeds 4 hours.19 Two, often-additive mechanisms are proposed: an athlete ingests water or low-solute beverages well beyond sweat losses (also known as water intoxication), or an athlete’s sweat sodium losses are not adequately replaced.15–18 The low blood-sodium levels are the result of a combination of excessive fluid intake and inappropriate body water retention in the water-intoxication model and insufficient fluid intake and inadequate sodium replacement in the latter. Ultimately, the intravascular and extracellular fluid has a lower solute load than the intracellular fluids, and water flows into the cells, producing intracellular swelling that causes potentially fatal neurologic and physiologic dysfunction. Affected athletes present with a combination of disorientation, altered mental status, headache, vomiting, lethargy, and swelling of the extremities (hands and feet), pulmonary edema, cerebral edema, and seizures. Exertional hyponatremia can result in death if not treated properly. This condition can be prevented by matching fluid intake with sweat and urine losses and by rehydrating with fluids that contain sufficient sodium.31,32

RECOMMENDATIONS

The National Athletic Trainers’ Association (NATA) advocates the following prevention, recognition, and treatment strategies for exertional heat illnesses. These recommendations are presented to help ATCs and other allied health providers maximize health, safety, and sport performance as they relate to these illnesses. Athletes’ individual responses to physiologic stimuli and environmental conditions vary widely. These recommendations do not guarantee full protection from heat-related illness but should decrease the risk during athletic participation. These recommendations should be considered by ATCs and allied health providers who work with athletes at risk for exertional heat illnesses to improve prevention strategies and ensure proper treatment.

Prevention

1. Ensure that appropriate medical care is available and that rescue personnel are familiar with exertional heat illness prevention, recognition, and treatment. Table 2 provides general guidelines that should be considered.7 Ensure that ATCs and other health care providers attending practices or events are allowed to evaluate and examine any athlete who displays signs or symptoms of heat illness33,34 and have the authority to restrict the athlete from participating if heat illness is present.

2. Conduct a thorough, physician-supervised, preparticipation medical screening before the season starts to identify athletes predisposed to heat illness on the basis of risk factors34–36 and those who have a history of exertional heat illness.

3. Adapt athletes to exercise in the heat (acclimatization) gradually over 10 to 14 days. Progressively increase the intensity and duration of work in the heat with a combination of strenuous interval training and continuous exercise.6,9,14,33,37–44 Well-acclimatized athletes should train for 1 to 2 hours under the same heat conditions that will be present for their event.6,45,46 In a cooler environment, an athlete can wear additional clothing during training to induce or maintain heat acclimatization. Athletes should maintain proper hydration during the heat-acclimatization process.47

4. Educate athletes and coaches regarding the prevention, recognition, and treatment of heat illnesses33,38,39,42,48–51 and the risks associated with exercising in hot, humid environmental conditions.

5. Educate athletes to match fluid intake with sweat and urine losses to maintain adequate hydration.8 (See the “National Athletic Trainers’ Association Position Statement: Fluid Replacement in Athletes.”52) Instruct athletes to drink sodium-containing fluids to keep their urine clear to light yellow to improve hydration3,34,52–55 and to replace fluids between practices on the same day and on successive days to maintain less than 2% body-weight change. These strategies will lessen the risk of acute and chronic dehydration and decrease the risk of heat-related events.

*References 9, 29, 37, 38, 40, 41, 43, 52–66.
Lack of Acclimatization to Heat. An athlete with no or minimal physiologic acclimatization to hot conditions is at an increased risk of heat-related illness.\textsuperscript{8,37,83,124} 

Medications and Drugs. Athletes who take certain medications or drugs, particularly medications with a dehydrating effect, are at an increased risk for a heat illness.\textsuperscript{101–106,125–136} Alcohol, caffeine, and theophylline at certain doses are mild diuretics.\textsuperscript{106,137,138} Caffeine is found in coffee, tea, soft drinks, chocolate, and several over-the-counter and prescription medications.\textsuperscript{139} Theophylline is found mostly in tea and anti-asthma medications.\textsuperscript{140} 

Electrolyte Imbalance. Electrolyte imbalances do not usually occur in trained, acclimatized individuals who engage in physical activity and eat a normal diet.\textsuperscript{141} Most sodium and chloride losses in athletes occur through the urine, but athletes who sweat heavily, are salty sweaters, or are not heat acclimatized can lose significant amounts of sodium during activity.\textsuperscript{142} Electrolyte imbalances often contribute to heat illness in older athletes who use diuretics.\textsuperscript{143,144} 

Predisposing Medical Conditions 

The following predisposing medical conditions add to the risk of heat illness. 

Malignant Hyperthermia. Malignant hyperthermia is caused by an autosomal dominant trait that causes muscle rigidity, resulting in elevation of body temperature due to the accelerated metabolic rate in the skeletal muscle.\textsuperscript{145–147} 

Neuroleptic Malignant Syndrome. Neuroleptic malignant syndrome is associated with the use of neuroleptic agents and antipsychotic drugs and an unexpected idiopathic increase in core temperature during exercise.\textsuperscript{148–151} 

Arteriosclerotic Vascular Disease. Arteriosclerotic vascular disease compromises cardiac output and blood flow through the vascular system by thickening the arterial walls.\textsuperscript{115,152} 

Scleroderma. Scleroderma is a skin disorder that decreases sweat production, thereby decreasing heat transfer.\textsuperscript{149,153} 

Cystic Fibrosis. Cystic fibrosis causes increased salt loss in sweat and can increase the risk for hyponatremia.\textsuperscript{154,155} 

Sickle Cell Trait. Sickle cell trait limits blood-flow distribution and decreases oxygen-carrying capacity. The condition is exacerbated by exercise at higher altitudes.\textsuperscript{156,157} 

Environmental Risk Factors 

When the environmental temperature is above skin temperature, athletes begin to absorb heat from the environment and depend entirely on evaporation for heat loss.\textsuperscript{113,158,159} High relative humidity inhibits heat loss from the body through evaporation.\textsuperscript{61} 

The environmental factors that influence the risk of heat illness include the ambient air temperature, relative humidity (amount of water vapor in the air), air motion, and the amount of radiant heat from the sun or other sources.\textsuperscript{2,9,41} The relative risk of heat illness can be calculated using the WBGT equation.\textsuperscript{2,43,50,69,77,160,161} Using the WBGT index to modify activity in high-risk settings has virtually eliminated heat-stroke deaths in United States Marine Corps recruits.\textsuperscript{159} Wet-bulb globe temperature is calculated using the wet-bulb (wb), dry-bulb (db), and black-globe (bg) temperature with the following equation\textsuperscript{99,82,85,162,163}:

$$WBGT = 0.7T_{wb} + 0.2T_{bg} + 0.1T_{db}$$

When there is no radiant heat load, $T_{db} = T_{bg}$, and the equation is reduced\textsuperscript{62} to

$$WBGT = 0.7T_{wb} + 0.3T_{db}$$

This equation is used to estimate risk as outlined in Table 3.13,40,50,61,85 This index was determined for athletes wearing a T-shirt and light pants.\textsuperscript{158} The WBGT calculation can be performed using information obtained from electronic devices\textsuperscript{42} or the local meteorologic service, but conversion tables for relative humidity and $T_{db}$ are needed to calculate the wet-bulb temperature.\textsuperscript{50,162} The predictive value from the meteorologic service is not as accurate as site-specific data for representing local heat load but will suffice in most situations. When WBGT measures are not possible, environmental heat stress can be estimated using a sling psychrometer (see Figures 1, 2).

Several recommendations have been published for distance running, but these can also be applied to other continuous activity sports. The Canadian Track and Field Association recommended that a distance race should be cancelled if the WBGT is greater than 26.7°C (80°F).\textsuperscript{39} The American College of Sports Medicine guidelines from 1996 recommended that a race should be delayed or rescheduled when the WBGT is greater than 27.8°C (82°F).\textsuperscript{31,72,73} In some instances, the event will go on regardless of the WBGT; ATCs should then have an increased level of suspicion for heat stroke and focus on hydration, emergency supplies, and detection of exertional heat illnesses.

Thermoregulation 

Thermoregulation is a complex interaction among the central nervous system (CNS), the cardiovascular system, and the skin to maintain a body-core temperature of 37°C.\textsuperscript{9,43,51,164} The CNS temperature-regulation center is located in the hypothalamus and is the site where the core temperature setpoint is determined.\textsuperscript{9,43,82,158,164–166} The hypothalamus receives information regarding body-core and shell temperatures from peripheral skin receptors and the circulating blood; body-core temperature is regulated through an open-ended feedback loop similar to that in a home thermostat system.\textsuperscript{158,165,167,168} Body responses for heat regulation include cutaneous vasodilation, increased sweating, increased heart rate, and increased respiratory rate.\textsuperscript{38,43,51,164,165}

Body-core temperature is determined by metabolic heat production and the transfer of body heat to and from the surrounding environment using the following heat-production and heat-storage equation\textsuperscript{166,167}:

$$S = M \pm R \pm K \pm CV - E$$

where $S$ is the amount of stored heat, $M$ is the metabolic heat production, $R$ is the heat gained or lost by radiation, $K$ is the convective heat lost or gained, $CV$ is the convective heat lost or gained, and $E$ is the evaporative heat lost.

Basal metabolic heat production fasting and at absolute rest is approximately 60 to 70 kcal/h for an average adult, with 50% of the heat produced by the internal organs. Metabolic heat produced by intense exercise may approach 1000 kcal/h.\textsuperscript{51,164} with greater than 90% of the heat resulting from muscle metabolism.\textsuperscript{9,40,42,166} Heat is gained or lost from the body by one or more of the following mechanisms: \textsuperscript{9,85}:
Radiation. The energy is transferred to or from an object or body via electromagnetic radiation from higher to lower energy surfaces.9,43,51,85,166

Conduction. Heat transfers from warmer to cooler objects through direct physical contact.9,43,51,85,166 Ice packs and cold-water baths are examples of conductive heat exchange.

Convection. Heat transfers to or from the body to surrounding moving fluid (including air).9,43,51,85,166 Moving air from a fan, cycling, or windy day produces convective heat exchange.

Evaporation. Heat transfers via the vaporization of sweat§ and is the most efficient means of heat loss.51,158,169 The evaporation of sweat from the skin depends on the water saturation of the air and the velocity of the moving air.170–172 The effectiveness of this evaporation for heat loss from the body diminishes rapidly when the relative humidity is greater than 60%.9,20,164

Cognitive performance and associated CNS functions deteriorate when brain temperature rises. Signs and symptoms include dizziness, confusion, behavior changes, coordination difficulties, decreased physical performance, and collapse due to hyperthermia.168,173 The residual effects of elevated brain temperature depend on the duration of the hyperthermia. Heat stroke rarely leads to permanent neurologic deficits51; however, some sporadic symptoms of frontal headache and sleep disturbances have been noted for up to 4 months.168,174,175 When permanent CNS damage occurs, it is associated with cerebellar changes, including ataxia, marked dysarthria, and dysmetria.174

Heat Acclimatization

Heat acclimatization is the physiologic response produced by repeated exposures to hot environments in which the capacity to withstand heat stress is improved.14,43,75,176,177 Physiologic responses to heat stress are summarized in Table 4. Exercise heat exposure produces progressive changes in thermoregulation that involve sweating, skin circulation, thermoregulatory setpoint, cardiovascular alterations, and endocrine adjustments.29,43,178 Individual differences affect the onset and decay of acclimatization.29,45,179 The rate of acclimatization is related to aerobic conditioning and fitness; more conditioned athletes acclimatize more quickly.43,45,180 The acclimatization process begins with heat exposure and is reasonably protective after 7 to 14 days, but maximum acclimatization may take 2 to 3 months.45,181,182 Heat acclimatization diminishes by day 6 when heat stress is no longer present.180,183 Fluid replacement improves the induction and effect of heat acclimatization.184–187 Extra salt in the diet during the first few days of heat exposure also improves acclimatization; this can be accomplished by encouraging the athlete to eat salty foods and to use the salt shaker liberally during meals.

Cumulative Dehydration

Cumulative dehydration develops insidiously over several days and is typically observed during the first few days of a season during practice sessions or in tournament competition. Cumulative dehydration can be detected by monitoring daily prepractice and postpractice weights. Even though a small decrease in body weight (less than 1%) may not have a detrimental effect on the individual, the cumulative effect of a 1% fluid loss per day occurring over several days will create an increased risk for heat illness and a decrease in performance.110

During intense exercise in the heat, sweat rates can be 1 to 2.5 L/h (about 1 to 2.25 kilograms [2 to 5 pounds] of body weight per hour) or more, resulting in dehydration. Unfortunately, the volume of fluid that most athletes drink voluntarily during exercise replaces only about 50% of body-fluid losses.188 Ideally, rehydration involves drinking at a rate sufficient to replace all of the water lost through sweating and urination.60,77 If the athlete is not able to drink at this rate, he or she should drink the maximum tolerated. Use caution to ensure that athletes do not overhydrate and put themselves at risk for the development of hyponatremia. However, hydration before an event is essential to help decrease the incidence of heat illnesses. For more information on this topic, see the “National Athletic Trainers’ Association Position Statement: Fluid Replacement in Athletes.”52

Cooling Therapies

The fastest way to decrease body-core temperature is immersion of the trunk and extremities into a pool or tub filled with cold water (between 1°C [35°F] and 15°C [59°F]).39,88,91,97 Conditions that have been associated with immersion therapy include shivering and peripheral vasoconstriction; however, the potential for these should not deter the medical staff from using immersion therapy for rapid cooling. Shivering can be prevented if the athlete is removed from the water once rectal temperature reaches 38.3°C to 38.9°C (101°F to 102°F). Peripheral vasoconstriction may occur, but the powerful cooling potential of immersion outweighs any potential concerns. Cardiogenic shock has also been a proposed consequence of immersion therapy, but this connection has not been proven in cooling heat-stroke patients.39 Cold-water immersion therapy was associated with a zero percent fatality rate in 252 cases of exertional heat stroke in the military.89 Other forms of cooling (water spray; ice packs covering the body; ice packs on axillae, groin, and neck; or blowing air) decrease body-core temperature at a slower rate compared with cold-water im-

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Table 4. Physiologic Responses After Heat Acclimatization Relative to Nonacclimatized State

<table>
<thead>
<tr>
<th>Physiologic Variable</th>
<th>After Acclimatization (10–14 Days’ Exposure)</th>
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<tbody>
<tr>
<td>Heart rate</td>
<td>Decreases9,146</td>
</tr>
<tr>
<td>Stroke volume</td>
<td>Increases146,147</td>
</tr>
<tr>
<td>Body-core temperature</td>
<td>Decreases145</td>
</tr>
<tr>
<td>Skin temperature</td>
<td>Decreases152</td>
</tr>
<tr>
<td>Sweat output/rate</td>
<td>Increases46,47,149</td>
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<tr>
<td>Onset of sweat</td>
<td>Earlier in training9,145</td>
</tr>
<tr>
<td>Evaporation of sweat</td>
<td>Increases146,152</td>
</tr>
<tr>
<td>Salt in sweat</td>
<td>Decreases9,50</td>
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<tr>
<td>Work output</td>
<td>Increases46,50</td>
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<tr>
<td>Subjective discomfort (rating of perceived exertion [RPE])</td>
<td>Decreases51,146</td>
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<tr>
<td>Fatigue</td>
<td>Decreases9,90</td>
</tr>
<tr>
<td>Capacity for work</td>
<td>Increases20,50</td>
</tr>
<tr>
<td>Mental disturbance</td>
<td>Decreases9,50</td>
</tr>
<tr>
<td>Syncopal response</td>
<td>Decreases9,50</td>
</tr>
<tr>
<td>Extracellular fluid volume</td>
<td>Increases9,50</td>
</tr>
<tr>
<td>Plasma volume</td>
<td>Increases20,150</td>
</tr>
</tbody>
</table>

§References 9, 40, 43, 50, 51, 85, 159, 165, 166.

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mersion.\textsuperscript{97} If immersion cooling is not being used, cooling with ice bags should be directed to as much of the body as possible, especially the major vessels in the armpit, groin, and neck regions (and likely the hands and feet), and cold towels may be applied to the head and trunk because these areas have been demonstrated on thermography.\textsuperscript{173,189} to have the most rapid heat loss.

**SPECIAL CONCERNS**

Most research related to heat illness has been performed on normal, healthy adults. Child athletes, older athletes, and athletes with spinal-cord injuries have been studied less frequently. The following are suggestions for special populations or those with special conditions.

**Children (Prepubescents)**

Exercise in hot environments and heat tolerance are affected by many physiologic factors in children. These include decreased sweat gland activity,\textsuperscript{190} higher skin temperatures,\textsuperscript{191±193} by many physiologic factors in children. These include decreased cardiac output (increased heart rate and lower stroke volume) due to increased peripheral circulation,\textsuperscript{194} decreased exercise economy,\textsuperscript{195} decreased ability to acclimatize to heat (slower and takes longer),\textsuperscript{196} smaller body size (issues related to body-surface-to-mass ratio), maturational differences,\textsuperscript{197} and predisposing conditions (obesity, hypohydration, childhood illnesses, and other disease states).\textsuperscript{190,192,196}

- Decrease the intensity of activities that last longer than 30 minutes,\textsuperscript{197} and have the athlete take brief rests\textsuperscript{50} if the WBGT is between 22.8°C and 27.8°C (73°F and 82°F); cancel or modify the activity if the WBGT is greater than 27.8°C (82°F).\textsuperscript{31,69–73} Modification could involve longer and more frequent rest breaks than are usually permitted within the rules of the sport (eg, insert a rest break before halftime).
- Encourage children to ingest some fluids at least every 15 to 30 minutes during activity to maintain hydration, even if they are not thirsty.\textsuperscript{197}
- Use similar precautions as listed earlier for adults.

**Older Athletes (≥50 Years Old)**

The ability of the older athlete to adapt is partly a function of age and also depends on functional capacity and physiologic health status.\textsuperscript{198–206}

- The athlete should be evaluated by a physician before exercise, with the potential consequences of predisposing medical conditions and illnesses addressed.\textsuperscript{9,34–36} An increase has been shown in the exercise heart rate of 1 heat per minute for each 1°C (1.8°F) increase in ambient temperature above neutral (23.9°C [75°F]).\textsuperscript{207} Athletes with known or suspected heart disease should curtail activities at lower temperatures than healthy athletes and should have cardiovascular stress testing before participating in hot environments.
- Older athletes have a decreased ability to maintain an adequate plasma volume and osmolality during exercise,\textsuperscript{198,208} which may predispose them to dehydration. Regular fluid intake is critical to avoid hyperthermia.

**Athletes with Spinal-Cord Injuries**

As sport participation for athletes with spinal-cord injuries increases from beginner to elite levels, understanding the disorder,\textsuperscript{209,210} training methods, and causes of heat injury will be critical to prevent heat illness.\textsuperscript{211} For example, the abilities to regulate heart rate, circulate the blood volume, produce sweat, and transfer heat to the surface may vary with the level and severity of the spinal-cord lesion.\textsuperscript{208,212–218}

- Monitor these athletes closely for heat-related problems. One technique for determining hyperthermia is to feel the skin under the arms of the distressed athlete.\textsuperscript{211} Rectal temperature may not be as accurate for measuring core temperature as in other athletes due to decreased ability to regulate blood flow beneath the spinal-cord lesion.\textsuperscript{218–220}
- If the athlete is hyperthermic, provide more water, lighter clothing, or cooling of the trunk,\textsuperscript{211,213} legs,\textsuperscript{211} and head.\textsuperscript{213}

**HOSPITALIZATION AND RECOVERY**

After an episode of heat stroke, the athlete may experience impaired thermoregulation, persistent CNS dysfunction,\textsuperscript{221,222} hepatic insufficiency, and renal insufficiency.\textsuperscript{39,223} For persons with exertional heat stroke and associated multisystem tissue damage, the rate of recovery is highly individualized, ranging up to more than 1 year.\textsuperscript{8,86,221} In one study, 9 of 10 patients exhibited normal heat-acclimatization responses, thermoregulation, whole-body sodium and potassium balance, sweat gland function, and blood values about 2 months after the heat stroke.\textsuperscript{8} Transient or persistent heat intolerance was found in a small percentage of patients.\textsuperscript{83} For some athletes, a history of exertional heat stroke increases the chance of experiencing subsequent episodes.\textsuperscript{39}

After recovery from an episode of heat stroke or hyponatremia, an athlete’s physical activity should be restricted\textsuperscript{8,86} and the gradual return to sport individualized by his or her physician. The athlete should be monitored on a daily basis by the ATC during exercise.\textsuperscript{86} During the return-to-exercise phase, an athlete may experience some detaining and deconditioning not directly related to the heat exposure.\textsuperscript{8,86} Evaluate the athlete over time to determine whether there has been a complete recovery of exercise and heat tolerance.\textsuperscript{8,86}

**CONCLUSIONS**

Athletic trainers and other allied health providers must be able to differentiate exercise-associated muscle (heat) cramps, heat syncope, exercise (heat) exhaustion, exertional heat stroke, and exertional hyponatremia in athletes.

This position statement outlines the NATA’s current recommendations to reduce the incidence, improve the recognition, and optimize treatment of heat illness in athletes. Education and increased awareness will help to reduce both the frequency and the severity of heat illness in athletes.

**ACKNOWLEDGMENTS**

This pronouncement was reviewed for the NATA by the Pronouncements Committee, Edward R. Eichner, MD, FACSM, and Wil-
liam O. Roberts, MD, MS, FACSM, T. Kyle Eubanks, MA, ATC, and Paul C. Miller, PhD, provided assistance in the preparation of the manuscript.

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