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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 (eg, 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

Condition Sign or Symptom*
Exercise-associated muscle (heat) cramps[6,9–11]
Dehydration
Thirst
Sweating
Transient muscle cramps
Fatigue
Heat syncope[10,12]
Dehydration
Fatigue
Tunnel vision
Pale or sweaty skin
Decreased pulse rate
Dizziness
Lightheadedness
Fainting
Exercise (heat) exhaustion[6,9,10,13]
Normal or elevated body-core temperature
Dehydration
Dizziness
Lightheadedness
Syncope
Headache
Nausea
Anorexia
Diarrhea
Decreased urine output
Persistent muscle cramps
Pallor
Profuse sweating
Chills
Cool, clammy skin
Intestinal cramps
Urge to defecate
Weakness
Hyperventilation
Exertional heat stroke[6,9,10,14]
High body-core temperature (>40°C [104°F])
Central nervous system changes
Dizziness
Drowsiness
Irrational behavior
Confusion
Irritability
Emotional instability
Hysteria
Apathy
Aggressiveness
Delirium
Disorientation
Staggering
Seizures
Loss of consciousness
Coma
Dehydration
Weakness
Hot and wet or dry skin
Tachycardia (100 to 120 beats per minute)
Hypotension
Hyperventilation
Vomiting
Diarrhea
Exertional hyponatremia[15–18]
Body-core temperature <40°C (104°F)
Nausea
Vomiting
Extremity (hands and feet) swelling
Low blood-sodium level
Progressive headache
Confusion
Significant mental compromise
Lethargy
Altered consciousness
Apathy
Pulmonary edema
Cerebral edema
Seizures
Coma

*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.[6–8] 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.[6,9–11,19]

Heat Syncope

Heat syncope, or orthostatic dizziness, can occur when a person is exposed to high environmental temperatures.[19] This condition is attributed to peripheral vasodilation, postural pooling of blood, diminished venous return, dehydration, reduction in cardiac output, and cerebral ischemia.[10,19] Heat syncope usually occurs during the first 5 days of acclimatization, before the blood volume expands,[12] or in persons with heart disease or those taking diuretics.[10] 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-

tion, sodium loss, and energy depletion. It occurs most frequently in hot, humid conditions. At its worst, it is difficult to distinguish from exertional heat stroke without measuring rectal temperature. Other signs and symptoms include pallor, persistent muscular cramps, urge to defecate, weakness, fainting, dizziness, headache, hyperventilation, nausea, anorexia, diarrhea, decreased urine output, and a body-core temperature that generally ranges between 36°C (97°F) and 40°C (104°F).[6,9,10,13,19]

Exertional Heat Stroke

Exertional heat stroke is an elevated core temperature (usually >40°C [104°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[20] 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.[6,10,14] The risk of morbidity and mortality is greater the longer an athlete's body temperature remains above 41°C (106°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 endotoxemia (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

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 illness[33,34] 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 factors[34–36] 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 illnesses[9,33,38,39,42,48–51] 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.* (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 hydration[33,34,52–55] 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.

Table 2. Prevention Checklist for the Certified Athletic Trainer*

1. Pre-event preparation
   - _____Am I challenging unsafe rules (eg, ability to receive fluids, modify game and practice times)?
   - _____Am I encouraging athletes to drink before the onset of thirst and to be well hydrated at the start of activity?
   - _____Am I familiar with which athletes have a history of a heat illness?
   - _____Am I discouraging alcohol, caffeine, and drug use?
   - _____Am I encouraging proper conditioning and acclimatization procedures?
2. Checking hydration status
   - _____Do I know the preexercise weight of the athletes (especially those at high risk) with whom I work, particularly during hot and humid conditions?
   - _____Are the athletes familiar with how to assess urine color? Is a urine color chart accessible?
   - _____Do the athletes know their sweat rates and, therefore, know how much to drink during exercise?
   - _____Is a refractometer or urine color chart present to provide additional information regarding hydration status in high-risk athletes when baseline body weights are checked?
3. Environmental assessment
   - _____Am I regularly checking the wet-bulb globe temperature or temperature and humidity during the day?
   - _____Am I knowledgeable about the risk categories of a heat illness based on the environmental conditions?
   - _____Are alternate plans made in case risky conditions force rescheduling of events or practices?
4. Coaches' and athletes' responsibilities
   - _____Are coaches and athletes educated about the signs and symptoms of heat illnesses?
   - _____Am I double checking to make sure coaches are allowing ample rest and rehydration breaks?
   - _____Are modifications being made to reduce risk in the heat (eg, decrease intensity, change practice times, allow more frequent breaks, eliminate double sessions, reduce or change equipment or clothing requirements, etc)?
   - _____Are rapid weight-loss practices in weight-class sports adamantly disallowed?
5. Event management
   - _____Have I checked to make sure proper amounts of fluids will be available and accessible?
   - _____Are carbohydrate-electrolyte drinks available at events and practices (especially during twice-a-day practices and those that last longer than 50 to 60 minutes or are extremely intense in nature)?
   - _____Am I aware of the factors that may increase the likelihood of a heat illness?
   - _____Am I promptly rehydrating athletes to preexercise weight after an exercise session?
   - _____Are shaded or indoor areas used for practices or breaks when possible to minimize thermal strain?
6. Treatment considerations
   - _____Am I familiar with the most common early signs and symptoms of heat illnesses?
   - _____Do I have the proper field equipment and skills to assess a heat illness?
   - _____Is an emergency plan in place in case an immediate evacuation is needed?
   - _____Is a kiddy pool available in situations of high risk to initiate immediate cold-water immersion of heat-stroke patients?
   - _____Are ice bags available for immediate cooling when cold-water immersion is not possible?
   - _____Have shaded, air-conditioned, and cool areas been identified to use when athletes need to cool down, recover, or receive treatment?
   - _____Are fans available to assist evaporation when cooling?
   - _____Am I properly equipped to assess high core temperature (ie, rectal thermometer)?
7. Other situation-specific considerations

*Adapted with permission from Casa.[7]

Table 3. Wet-Bulb Globe Temperature Risk Chart[62–67]*

WBGT	Flag Color	Level of Risk	Comments
<18°C (<65°F)	Green	Low	Risk low but still exists on the basis of risk factors
18–23°C (65–73°F)	Yellow	Moderate	Risk level increases as event progresses through the day
23–28°C (73–82°F)	Red	High	Everyone should be aware of injury potential; individuals at risk should not compete
>28°C (82°F)	Black	Extreme or hazardous	Consider rescheduling or delaying the event until safer conditions prevail; if the event must take place, be on high alert

*Adapted with permission from Roberts.[67]

6. Encourage athletes to sleep at least 6 to 8 hours at night in a cool environment,[41,35,50] eat a well-balanced diet that follows the Food Guide Pyramid and United States Dietary Guidelines,[56–58] and maintain proper hydration status. Athletes exercising in hot conditions (especially during twice-a-day practices) require extra sodium from the diet or rehydration beverages or both.

7. Develop event and practice guidelines for hot, humid weather that anticipate potential problems encountered based on the wet-bulb globe temperature (WBGT) (Table 3) or heat and humidity as measured by a sling psychrometer (Figure 1), the number of participants, the nature of the activity, and other predisposing risk factors.[14,51] If the WBGT is greater than 28°C (82°F, or ''very high'' as indicated in Table 3, Figure 1), an athletic event should be delayed, rescheduled, or moved into an air-conditioned space, if possible.[69–74] It is important to note that these measures are based on the risk of environmental stress for athletes wearing shorts and a T-shirt; if an

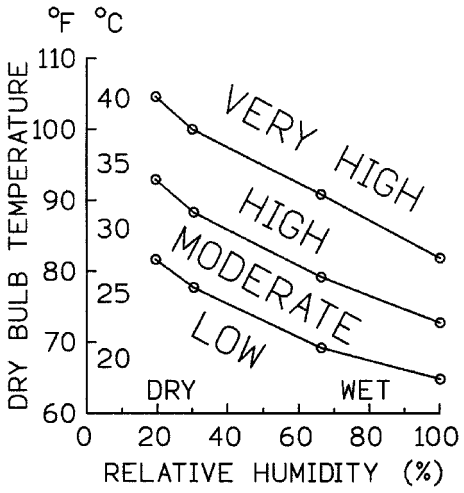

Figure 1. Risk of heat exhaustion or heat stroke while racing in hot environments. However, Figure 2 may be better suited for estimating heat-stroke risk when equipment is worn. Reprinted with permission from Convertino VA, Armstrong LE, Coyle EF, et al. American College of Sports Medicine position stand: exercise and fluid replacement. *Med Sci Sports Exerc.* 1996;28:i–vii.[31]

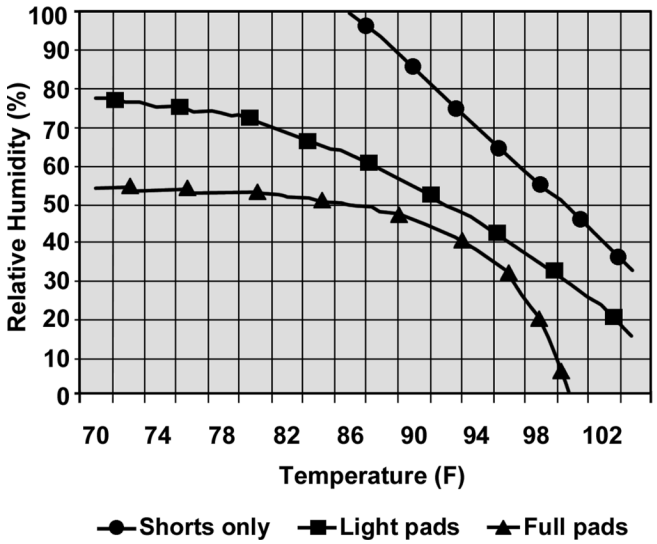

Figure 2. Heat stress risk temperature and humidity graph. Heat-stroke risk rises with increasing heat and relative humidity. Fluid breaks should be scheduled for all practices and scheduled more frequently as the heat stress rises. Add 5° to temperature between 10 AM and 4 PM from mid May to mid September on bright, sunny days. Practices should be modified for the safety of the athletes to reflect the heat-stress conditions. Regular practices with full practice gear can be conducted for conditions that plot to the left of the triangles. Cancel all practices when the temperature and relative humidity plot is to the right of the circles; practices may be moved into air-conditioned spaces or held as walk-through sessions with no conditioning activities.

Conditions that plot between squares and circles: increase rest-to-work ratio with 5- to 10-minute rest and fluid breaks every 15 to 20 minutes; practice should be in shorts only with all protective equipment removed.

Conditions that plot between triangles and squares: increase rest-to-work ratio with 5- to 10-minute rest and fluid breaks every 20 to 30 minutes; practice should be in shorts with helmets and shoulder pads (not full equipment).

Adapted with permission from Kulka J, Kenney WL. Heat balance limits in football uniforms: how different uniform ensembles alter the equation. *Physician Sportsmed.* 2002;30(7):29–39.[68]

athlete is wearing additional clothing (ie, football uniform, wetsuit, helmet), a lower WBGT value could result in comparable risk of environmental heat stress (Figure 2).[75,76] If the event or practice is conducted in hot, humid conditions, then use extreme caution in monitoring the athletes and be proactive in taking preventive steps. In addition, be sure that emergency supplies and equipment are easily accessible and in good working order. The most important factors are to limit intensity and duration of activity, limit the amount of clothing and equipment worn, increase the number and length of rest breaks, and encourage proper hydration.

Modify activity under high-risk conditions to prevent exertional heat illnesses.[19,21] Identify individuals who are susceptible to heat illnesses. In some athletes, the prodromal signs and symptoms of heat illnesses are not evident before collapse, but in many cases, adept medical supervision will allow early intervention.

8. Check the environmental conditions before and during the activity, and adjust the practice schedule accordingly.[29,38,41,42,60] Schedule training sessions to avoid the hottest part of the day (10 AM to 5 PM) and to avoid radiant heating from direct sunlight, especially in the acclimatization during the first few days of practice sessions.[9,29,33,34,38,40,50,60]

9. Plan rest breaks to match the environmental conditions and the intensity of the activity.[33,34] Exercise intensity and environmental conditions should be the major determinants in deciding the length and frequency of rest breaks. If possible, cancel or postpone the activity or move it indoors (if air conditioned) if the conditions are ''extreme or hazardous'' (see Table 3) or ''very high'' (see Figure 1) or to the right of the circled line (see Figure 2). General guidelines during intense exercise would include a work:rest ratio of 1:1, 2:1, 3:1, and 4:1 for ''extreme or hazardous'' (see Table 3) or ''very high'' (see Figure 1), ''high,'' ''moderate,'' or ''low'' environmental risk, respectively.[41,77] For activities such as football in which equipment must be considered, please refer to Figure 2 for equipment modifications and appropriate work:rest ratios for various environmental conditions. Rest breaks should occur in the shade if possible, and hydration during rest breaks should be encouraged.

10. Implement rest periods at mealtime by allowing 2 to 3 hours for food, fluids, nutrients, and electrolytes (sodium and potassium) to move into the small intestine and bloodstream before the next practice.[34,50,77]

11. Provide an adequate supply of proper fluids (water or sports drinks) to maintain hydration[9,34,38,40,50,60] and institute a hydration protocol that allows the maintenance of hydration status.[34,49] Fluids should be readily available and served in containers that allow adequate volumes to be ingested with ease and with minimal interruption of exercise.[49,52] The goal should be to lose no more than 2% to 3% of body weight during the practice session (due to sweat and urine losses).[78–82] (See the ''National Athletic Trainers' Association Position Statement: Fluid Replacement in Athletes.''[52])

12. Weigh high-risk athletes (in high-risk conditions, weigh all athletes) before and after practice to estimate the amount of body water lost during practice and to ensure a return to prepractice weight before the next practice. Following exercise athletes should consume approximately 1–1.25 L (16 oz) of fluid for each kilogram of body water lost during exercise.†

†*References 6, 9, 29, 33, 38, 40, 49, 60, 77, 83.*

13. Minimize the amount of equipment and clothing worn by the athlete in hot or humid (or both) conditions. For example, a full football uniform prevents sweat evaporation from more than 60% of the body.[29,33,40,51,77] Consult Figure 2 for possible equipment and clothing recommendations. When athletes exercise in the heat, they should wear loose-fitting, absorbent, and light-colored clothing; mesh clothing and new-generation cloth blends have been specially designed to allow more effective cooling.‡

14. Minimize warm-up time when feasible, and conduct warm-up sessions in the shade when possible to minimize the radiant heat load in ''high'' or ''very high'' or ''extreme or hazardous'' (see Table 3, Figure 1) conditions.[77]

15. Allow athletes to practice in shaded areas and use electric or cooling fans to circulate air whenever feasible.[66]

16. Include the following supplies on the field, in the locker room, and at various other stations:

- A supply of cool water or sports drinks or both to meet the participants' needs (see the ''National Athletic Trainers' Association Position Statement: Fluid Replacement in Athletes''[52] for recommendations regarding the appropriate composition of rehydration beverages based on the length and intensity of the activity)[29,34,38]
- Ice for active cooling (ice bags, tub cooling) and to keep beverages cool during exercise[29,38]
- Rectal thermometer to assess body-core temperature[39,74,75,87,88]
- Telephone or 2-way radio to communicate with medical personnel and to summon emergency medical transportation[38,39,48]
- Tub, wading pool, kiddy pool, or whirlpool to cool the trunk and extremities for immersion cooling therapy[35,65]

17. Notify local hospital and emergency personnel before mass participation events to inform them of the event and the increased possibility of heat-related illnesses.[41,89]

18. Mandate a check of hydration status at weigh-in to ensure athletes in sports requiring weight classes (eg, wrestling, judo, rowing) are not dehydrated. Any procedures used to induce dramatic dehydration (eg, diuretics, rubber suits, exercising in a sauna) are strictly prohibited.[52] Dehydrated athletes exercising at the same intensity as euhydrated athletes are at increased risk for thermoregulatory strain (see the ''National Athletic Trainers' Association Position Statement: Fluid Replacement in Athletes''[52]).

Recognition and Treatment

19. Exercise-associated muscle (heat) cramps:

- An athlete showing signs or symptoms including dehydration, thirst, sweating, transient muscle cramps, and fatigue is likely experiencing exercise-associated muscle (heat) cramps.
- To relieve muscle spasms, the athlete should stop activity, replace lost fluids with sodium-containing fluids, and begin mild stretching with massage of the muscle spasm.
- Fluid absorption is enhanced with sports drinks that contain sodium.[52,60,87] A high-sodium sports product may be added to the rehydration beverage to prevent or relieve cramping in athletes who lose large amounts of sodium in their sweat.[19] A simple salted fluid consists of two 10-grain salt tablets dissolved in 1 L (34 oz) of water. Intravenous fluids may be required if nausea or vomiting limits oral fluid intake; these must be ordered by a physician.[6,7,52,90,91]
- A recumbent position may allow more rapid redistribution of blood flow to cramping leg muscles.

20. Heat syncope:

- If an athlete experiences a brief episode of fainting associated with dizziness, tunnel vision, pale or sweaty skin, and a decreased pulse rate but has a normal rectal temperature (for exercise, 36°C to 40°C [97°F to 104°F]), then heat syncope is most likely the cause.[19]
- Move the athlete to a shaded area, monitor vital signs, elevate the legs above the level of the head, and rehydrate.

21. Exercise (heat) exhaustion:

- Cognitive changes are usually minimal, but assess central nervous system function for bizarre behavior, hallucinations, altered mental status, confusion, disorientation, or coma (see Table 1) to rule out more serious conditions.
- If feasible, measure body-core temperature (rectal temperature) and assess cognitive function (see Table 1) and vital signs.[19] Rectal temperature is the most accurate method possible in the field to monitor body-core temperature.[34,74,75,87,88] The ATC should not rely on the oral, tympanic, or axillary temperature for athletes because these are inaccurate and ineffective measures of body-core temperature during and after exercise.[75,89,92]
- If the athlete's temperature is elevated, remove his or her excess clothing to increase the evaporative surface and to facilitate cooling.[6,93]
- Cool the athlete with fans,[94] ice towels,[29,38] or ice bags because these may help the athlete with a temperature of more than 38.8°C (102°F) to feel better faster.
- Remove the athlete to a cool or shaded environment if possible.
- Start fluid replacement.[6,52,93,95]
- Transfer care to a physician if intravenous fluids are needed[6,52,90,91,96] or if recovery is not rapid and uneventful.

22. Exertional heat stroke:

- Measure the rectal temperature if feasible to differentiate between heat exhaustion and heat stroke. With heat stroke, rectal temperature is elevated (generally higher than 40°C [104°F]).[19]
- Assess cognitive function, which is markedly altered in exertional heat stroke (see Table 1).
- Lower the body-core temperature as quickly as possible.[34,70,77] The fastest way to decrease body temperature is to remove clothes and equipment and immerse the body (trunk and extremities) into a pool or tub of cold water (approximately 1°C to 15°C [35°F to 59°F]).[32,91,92,97–99] Aggressive cooling is the most critical factor in the treatment of exertional heat stroke. Circulation of the tub water may enhance cooling.
- Monitor the temperature during the cooling therapy and recovery (every 5 to 10 minutes).[39,87] Once the athlete's rectal temperature reaches approximately 38.3°C to 38.9°C (101°F to 102°F), he or she should be removed from the pool or tub to avoid overcooling.[40,100]
- If a physician is present to manage the athlete's medical care on site, then initial transportation to a medical facility may not be necessary so immersion can continue uninterrupted.

‡*References 8, 9, 29, 33, 38, 40, 53, 59, 84–86.*

If a physician is not present, aggressive first-aid cooling should be initiated on site and continued during emergency medical system transport and at the hospital until the athlete is normothermic.
- Activate the emergency medical system.
- Monitor the athlete's vital signs and other signs and symptoms of heat stroke (see Table 1).[34,95]
- During transport and when immersion is not feasible, other methods can be used to reduce body temperature: removing the clothing; sponging down the athlete with cool water and applying cold towels; applying ice bags to as much of the body as possible, especially the major vessels in the armpit, groin, and neck; providing shade; and fanning the body with air.[39,95]
- In addition to cooling therapies, first-aid emergency procedures for heat stroke may include airway management. Also a physician may decide to begin intravenous fluid replacement.[87]
- Monitor for organ-system complications for at least 24 hours.

23. Exertional hyponatremia:

- Attempt to differentiate between hyponatremia and heat exhaustion. Hyponatremia is characterized by increasing headache, significant mental compromise, altered consciousness, seizures, lethargy, and swelling in the extremities. The athlete may be dehydrated, normally hydrated, or overhydrated.[19]
- Attempt to differentiate between hyponatremia and heat stroke. In hyponatremia, hyperthermia is likely to be less (rectal temperature less than 40°C [104°F]).[19] The plasma-sodium level is less than 130 mEq/L and can be measured with a sodium analyzer on site if the device is available.
- If hyponatremia is suspected, immediate transfer to an emergency medical center via the emergency medical system is indicated. An intravenous line should be placed to administer medication as needed to increase sodium levels, induce diuresis, and control seizures.
- An athlete with suspected hyponatremia should not be administered fluids until a physician is consulted.

24. Return to activity

In cases of exercise-associated muscle (heat) cramps or heat syncope, the ATC should discuss the athlete's case with the supervising physician. The cases of athletes with heat exhaustion who were not transferred to the physician's care should also be discussed with the physician. After exertional heat stroke or exertional hyponatremia, the athlete must be cleared by a physician before returning to athletic participation.[92] The return to full activity should be gradual and monitored.[8,87]

BACKGROUND AND LITERATURE REVIEW

Diagnosis

To differentiate heat illnesses in athletes, ATCs and other on-site health care providers must be familiar with the signs and symptoms of each condition (see Table 1). Other medical conditions (eg, asthma, status epilepticus, drug toxicities) may also present with similar signs and symptoms. It is important to realize, however, that an athlete with a heat illness will not exhibit all the signs and symptoms of a specific condition, increasing the need for diligent observation during athletic activity.

Nonenvironmental Risk Factors

Athletic trainers and other health care providers should be sensitive to the following nonenvironmental risk factors, which could place athletes at risk for heat illness.

Dehydration. Sweating, inadequate fluid intake, vomiting, diarrhea, certain medications,[89,101–103] and alcohol[104,105] or caffeine[106] use can lead to fluid deficit. Body-weight change is the preferred method to monitor for dehydration in the field, but a clinical refractometer is another accurate method (specific gravity should be no more than 1.020).[34,49,107–110] Dehydration can also be identified by monitoring urine color or body-weight changes before, during, and after a practice or an event and across successive days.[53,54]

The signs and symptoms of dehydration are thirst, general discomfort, flushed skin, weariness, cramps, apathy, dizziness, headache, vomiting, nausea, heat sensations on the head or neck, chills, decreased performance, and dyspnea.[52] Water loss that is not regained by the next practice increases the risk for heat illness.[110]

Barriers to Evaporation. Athletic equipment and rubber or plastic suits used for ''weight loss'' do not allow water vapor to pass through and inhibit evaporative, convective, and radiant heat loss.[111,112] Participants who wear equipment that does not allow for heat dissipation are at an increased risk for heat illness.[113] Helmets are also limiting because a significant amount of heat is dissipated through the head.

Illness. Athletes who are currently or were recently ill may be at an increased risk for heat illness because of fever or dehydration.[114–116]

History of Heat Illness. Some individuals with a history of heat illness are at greater risk for recurrent heat illness.[8,117]

Increased Body Mass Index (Thick Fat Layer or Small Surface Area). Obese individuals are at an increased risk for heat illness because the fat layer decreases heat loss.[118] Obese persons are less efficient and have a greater metabolic heat production during exercise. Conversely, muscle-bound individuals have increased metabolic heat production and a lower ratio of surface area to mass, contributing to a decreased ability to dissipate heat.[119–121]

Wet-Bulb Globe Temperature on Previous Day and Night. When the WBGT is high to extreme (see Table 3), the risk of heat-related problems is greater the next day; this appears to be one of the best predictors of heat illness.[121] Athletes who sleep in cool or air-conditioned quarters are at less risk.

Poor Physical Condition. Individuals who are untrained are more susceptible to heat illness than are trained athletes. As the $\dot{V}O_2$max of an individual improves, the ability to withstand heat stress improves independent of acclimatization and heat adaptation.[122] High-intensity work can easily produce 1000 kcal/h and elevate the core temperature of at-risk individuals (those who are unfit, overweight, or unacclimatized) to dangerous levels within 20 to 30 minutes.[123]

Excessive or Dark-Colored Clothing or Equipment. Excessive clothing or equipment decreases the ability to thermoregulate, and dark-colored clothing or equipment may cause a greater absorption of heat from the environment. Both should be avoided.[113]

Overzealousness. Overzealous athletes are at a higher risk for heat illness because they override the normal behavioral adaptations to heat and decrease the likelihood of subtle cues being recognized.

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.[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.[101–106,125–136] Alcohol, caffeine, and theophylline at certain doses are mild diuretics.[106,137,138] Caffeine is found in coffee, tea, soft drinks, chocolate, and several over-the-counter and prescription medications.[139] Theophylline is found mostly in tea and anti-asthma medications.[140]

Electrolyte Imbalance. Electrolyte imbalances do not usually occur in trained, acclimatized individuals who engage in physical activity and eat a normal diet.[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.[142] Electrolyte imbalances often contribute to heat illness in older athletes who use diuretics.[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.[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.[148–151]

Arteriosclerotic Vascular Disease. Arteriosclerotic vascular disease compromises cardiac output and blood flow through the vascular system by thickening the arterial walls.[115,152]

Scleroderma. Scleroderma is a skin disorder that decreases sweat production, thereby decreasing heat transfer.[149,153]

Cystic Fibrosis. Cystic fibrosis causes increased salt loss in sweat and can increase the risk for hyponatremia.[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.[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.[113,158,159] High relative humidity inhibits heat loss from the body through evaporation.[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.[2,9,41] The relative risk of heat illness can be calculated using the WBGT equation.[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.[159] Wet-bulb globe temperature is calculated using the wet-bulb (wb), dry-bulb (db), and black-globe (bg) temperature with the following equation[49,62,85,162,163]:

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

When there is no radiant heat load, $T_{db} = T_{bg}$, and the equation is reduced[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.[158] The WBGT calculation can be performed using information obtained from electronic devices[42] or the local meteorologic service, but conversion tables for relative humidity and T_{db} are needed to calculate the wet-bulb temperature.[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).[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).[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.[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.[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.[158,165,167,168] Body responses for heat regulation include cutaneous vasodilation, increased sweating, increased heart rate, and increased respiratory rate.[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[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 conductive 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,[51,164] with greater than 90% of the heat resulting from muscle metabolism.[9,40,42,166]

Heat is gained or lost from the body by one or more of the following mechanisms[9,85]:

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

Physiologic Variable	After Acclimatization (10–14 Days' Exposure)
Heart rate	Decreases[46,145]
Stroke volume	Increases[145,147]
Body-core temperature	Decreases[145]
Skin temperature	Decreases[152]
Sweat output/rate	Increases[46,47,149]
Onset of sweat	Earlier in training[46,145]
Evaporation of sweat	Increases[47,152]
Salt in sweat	Decreases[9,50]
Work output	Increases[46,50]
Subjective discomfort (rating of perceived exertion [RPE])	Decreases[50,145]
Fatigue	Decreases[50]
Capacity for work	Increases[46,50]
Mental disturbance	Decreases[50]
Syncopal response	Decreases[9,50]
Extracellular fluid volume	Increases[50]
Plasma volume	Increases[50,150]

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 deficits[51]; 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-

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

mersion.[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[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,[190] higher skin temperatures,[191–193] decreased cardiac output (increased heart rate and lower stroke volume) due to increased peripheral circulation,[194] decreased exercise economy,[195] decreased ability to acclimatize to heat (slower and takes longer),[192] smaller body size (issues related to body surface-to-mass ratio), maturational differences,[190] and predisposing conditions (obesity, hypohydration, childhood illnesses, and other disease states).[190,192,196]

- Decrease the intensity of activities that last longer than 30 minutes,[197] and have the athlete take brief rests[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).[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.[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.[198–206]

- The athlete should be evaluated by a physician before exercise, with the potential consequences of predisposing medical conditions and illnesses addressed.[9,34–36] An increase has been shown in the exercise heart rate of 1 beat per minute for each 1°C (1.8°F) increase in ambient temperature above neutral (23.9°C [75°F]).[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,[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 disability,[209,210] training methods, and causes of heat injury will help make competition safer.[211] For example, the abilities to regulate heart rate, circulate the blood volume, produce sweat, and transfer heat to the surface vary with the level and severity of the spinal-cord lesion.[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.[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.[218–220]
- If the athlete is hyperthermic, provide more water, lighter clothing, or cooling of the trunk,[211,213] legs,[211] and head.[213]

HOSPITALIZATION AND RECOVERY

After an episode of heat stroke, the athlete may experience impaired thermoregulation, persistent CNS dysfunction,[221,222] hepatic insufficiency, and renal insufficiency.[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.[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.[8] Transient or persistent heat intolerance was found in a small percentage of patients.[83] For some athletes, a history of exertional heat stroke increases the chance of experiencing subsequent episodes.[39]

An athlete who experiences heat stroke may have compromised heat tolerance and heat acclimatization after physician clearance.[35,224,225] Decreased heat tolerance may affect 15% to 20% of persons after a heat stroke-related collapse,[226,227] and in a few individuals, decreased heat tolerance has persisted up to 5 years.[35,224,228] Additional heat stress may reduce the athlete's ability to train and compete due to impaired cardiovascular and thermoregulatory responses.[115,228–230]

After recovery from an episode of heat stroke or hyponatremia, an athlete's physical activity should be restricted[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.[86] During the return-to-exercise phase, an athlete may experience some detraining and deconditioning not directly related to the heat exposure.[8,86] Evaluate the athlete over time to determine whether there has been a complete recovery of exercise and heat tolerance.[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.

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REFERENCES

1. Hawley DA, Slentz K, Clark MA, Pless JE, Waller BF. Athletic fatalities. *Am J Forensic Med Pathol.* 1990;11:124–129.
2. Mueller FO, Schindler RD. Annual survey of football injury research 1931–1984. *Athl Train J Natl Athl Train Assoc.* 1985;20:213–218.
3. Bijur PE, Trumble A, Harel Y, Overpeck MD, Jones D, Scheidt PC. Sports and recreation injuries in US children and adolescents. *Arch Pediatr Adolesc Med.* 1995;149:1009–1016.
4. Tucker AM. Common soccer injuries: diagnosis, treatment and rehabilitation. *Sports Med.* 1997;23:21–32.
5. Martin DE. Influence of elevated climatic heat stress on athletic competition in Atlanta, 1996. *New Stud Athl.* 1997;12:65–78.
6. Rich B. Environmental concerns: heat. In: Sallis RE, Massimino F, eds. *Essentials of Sports Medicine.* St Louis, MO: Mosby Year Book; 1997: 129–133.
7. Casa DJ. Exercise in the heat, II: critical concepts in rehydration, exertional heat illnesses, and maximizing athletic performance. *J Athl Train.* 1999;34:253–262.
8. Armstrong LE, De Luca JP, Hubbard RW. Time course of recovery and heat acclimation ability of prior exertional heatstroke patients. *Med Sci Sports Exerc.* 1990;22:36–48.
9. Brewster SJ, O'Connor FG, Lillegard WA. Exercise-induced heat injury: diagnosis and management. *Sports Med Arthrosc Rev.* 1995;3:206–266.
10. Knochel JP. Environmental heat illness: an eclectic review. *Arch Intern Med.* 1974;133:841–864.
11. Bergeron MF. Heat cramps during tennis: a case report. *Int J Sport Nutr.* 1996;6:62–68.
12. Hubbard R, Gaffin S, Squire D. Heat-related illness. In: Auerbach PS, ed. *Wilderness Medicine.* 3rd ed. St Louis, MO: Mosby Year Book; 1995:167–212.
13. Armstrong LE, Hubbard RW, Kraemer WJ, DeLuca JP, Christensen EL. Signs and symptoms of heat exhaustion during strenuous exercise. *Ann Sports Med.* 1987;3:182–189.
14. Epstein Y. Exertional heatstroke: lessons we tend to forget. *Am J Med Sports.* 2000;2:143–152.
15. Epstein Y, Armstrong LE. Fluid-electrolyte balance during labor and exercise: concepts and misconceptions. *Int J Sport Nutr.* 1999;9:1–12.
16. Maughan RJ. Optimizing hydration for competitive sport. In: Lamb DR, Murray R, eds. *Optimizing Sport Performance.* Carmel, IN: Cooper Publishing; 1997:139–183.
17. Armstrong LE, Curtis WC, Hubbard RW, Francesconi RP, Moore R, Askew W. Symptomatic hyponatremia during prolonged exercise in the heat. *Med Sci Sports Exerc.* 1993;25:543–549.
18. Garigan T, Ristedt DE. Death from hyponatremia as a result of acute water intoxication in an Army basic trainee. *Mil Med.* 1999;164:234–238.
19. Casa DJ, Roberts WO. Considerations for the medical staff in preventing, identifying and treating exertional heat illnesses. In: Armstrong LE, ed. *Exertional Heat Illnesses.* Champaign, IL: Human Kinetics; 2003. In press.
20. Cabanac M, White MD. Core temperature thresholds of hyperpnea during passive hyperthermia in humans. *Eur J Appl Physiol Occup Physiol.* 1995;71:71–76.
21. Casa DJ, Armstrong LE. Heatstroke: a medical emergency. In: Armstrong LE, ed. *Exertional Heat Illnesses.* Champaign, IL: Human Kinetics: 2003. In press.
22. Vicario SJ, Okabajue R, Haltom T. Rapid cooling in classic heatstroke: effect on mortality rates. *Am J Emerg Med.* 1986;4:394–398.
23. Assia E, Epstein Y, Shapiro Y. Fatal heatstroke after a short march at night: a case report. *Aviat Space Environ Med.* 1985;56:441–442.
24. Graham BS, Lichtenstein MJ, Hinson JM, Theil GB. Nonexertional heatstroke: physiologic management and cooling in 14 patients. *Arch Intern Med.* 1986;146:87–90.
25. Hart GR, Anderson RJ, Crumpler CP, Shulkin A, Reed G, Knochel JP. Epidemic classical heat stroke: clinical characteristics and course of 28 patients. *Medicine (Baltimore).* 1982;61:189–197.
26. Thomas C, ed. *Taber's Cyclopedic Medical Dictionary.* Philadelphia, PA: FA Davis; 1993.
27. Akhtar MJ, Al-Nozha M, al-Harthi S, Nouh MS. Electrocardiographic abnormalities in patients with heat stroke. *Chest.* 1993;104:411–414.
28. Partin N. Internal medicine: exertional heatstroke. *Athl Train J Natl Athl Train Assoc.* 1990;25:192–194.
29. Knochel J. Management of heat conditions. *Athl Ther Today.* 1996;1: 30–34.
30. Hubbard RW, Armstrong LE. Hyperthermia: new thoughts on an old problem. *Physician Sportsmed.* 1989;17(6):97–98,101,104,107–108,111–113.
31. Convertino VA, Armstrong LE, Coyle EF, et al. American College of Sports Medicine position stand: exercise and fluid replacement. *Med Sci Sports Exerc.* 1996;28:i–vii.
32. Armstrong LE, Casa DJ, Watson G. Exertional hyponatremia: unanswered questions and etiological perspectives. *Int J Sport Nutr Exerc Metab.* In press.
33. Francis K, Feinstein R, Brasher J. Optimal practice times for the reduction of the risk of heat illness during fall football practice in the Southeastern United States. *Athl Train J Natl Athl Train Assoc.* 1991;26:76–78,80.
34. Shapiro Y, Seidman DS. Field and clinical observations of exertional heat stroke patients. *Med Sci Sports Exerc.* 1990;22:6–14.
35. Epstein Y, Shapiro Y, Brill S. Role of surface area-to-mass ratio and work efficiency in heat intolerance. *J Appl Physiol.* 1983;54:831–836.
36. Kenney WL. Physiological correlates of heat intolerance. *Sports Med.* 1985;2:279–286.
37. Mitchell D, Senay LC, Wyndham CH, van Rensburg AJ, Rogers GG, Strydom NB. Acclimatization in a hot, humid environment: energy exchange, body temperature, and sweating. *J Appl Physiol.* 1976;40:768–778.
38. Davidson M. Heat illness in athletics. *Athl Train J Natl Athl Train Assoc.* 1985;20:96–101.
39. Brodeur VB, Dennett SR, Griffin LS. Exertional hyperthermia, ice baths, and emergency care at the Falmouth Road Race. *J Emerg Nurs.* 1989; 15:304–312.
40. Allman FL Jr. The effects of heat on the athlete. *J Med Assoc Ga.* 1992; 81:307–310.
41. Bernard TE. Risk management for preventing heat illness in athletes. *Athl Ther Today.* 1996;1:19–21.
42. Delaney KA. Heatstroke: underlying processes and lifesaving management. *Postgrad Med.* 1992;91:379–388.
43. Haymes EM, Wells CL. *Environment and Human Performance.* Champaign, IL: Human Kinetics; 1986:1–41.
44. Gisolfi C, Robinson S. Relations between physical training, acclimatization, and heat tolerance. *J Appl Physiol.* 1969;26:530–534.
45. Armstrong LE, Maresh CM. The induction and decay of heat acclimatisation in trained athletes. *Sports Med.* 1991;12:302–312.
46. Fortney SM, Vroman NB. Exercise, performance and temperature control: temperature regulation during exercise and implications for sports performance and training. *Sports Med.* 1985;2:8–20.
47. Dawson B. Exercise training in sweat clothing in cool conditions to improve heat tolerance. *Sports Med.* 1994;17:233–244.
48. Kleiner DM, Glickman SE. Medical considerations and planning for short distance road races. *J Athl Train.* 1994;29:145–146,149–151.
49. Murray B. Fluid replacement: the American College of Sports Medicine position stand. *Sport Sci Exch.* 1996;9(4S):63.
50. Elias SR, Roberts WO, Thorson DC. Team sports in hot weather: guidelines for modifying youth soccer. *Physician Sportsmed.* 1991;19(5):67–68,72–74,77,80.
51. Knochel JP. Heat stroke and related heat stress disorders. *Dis Month.* 1989;35:301–377.
52. Casa DJ, Armstrong LE, Hillman SK, et al. National Athletic Trainers' Association position statement: fluid replacement for athletes. *J Athl Train.* 2000;35:212–224.
53. Armstrong LE, Maresh CM, Castellani JW, et al. Urinary indices of hydration status. *Int J Sport Nutr.* 1994;4:265–279.
54. Armstrong LE, Soto JA, Hacker FT Jr, Casa DJ, Kavouras SA, Maresh

CM. Urinary indices during dehydration exercise and rehydration. *Int J Sport Nutr.* 1997;8:345–355.
55. Heat and humidity. In: Armstrong LE. *Performing in Extreme Environments.* Champaign, IL: Human Kinetics; 2000:15–70.
56. Nadel ER, Fortney SM, Wenger CB. Effect of hydration state on circulatory and thermal regulations. *J Appl Physiol.* 1980;49:715–721.
57. Keithley JK, Keller A, Vazquez MG. Promoting good nutrition: using the food guide pyramid in clinical practice. *Medsurg Nurs.* 1996;5:397–403.
58. Achterberg C, McDonnell E, Bagby R. How to put the Food Guide Pyramid into practice. *J Am Diet Assoc.* 1994;94:1030–1035.
59. Laywell P. Guidelines for pre-event eating. *Texas Coach.* 1981;25:40–41,59.
60. Terrados N, Maughan RJ. Exercise in the heat: strategies to minimize the adverse effects on performance. *J Sports Sci.* 1995;13(suppl):55–62.
61. Armstrong LE, Hubbard RW, Szlyk PC, Matthew WT, Sils IV. Voluntary dehydration and electrolyte losses during prolonged exercise in the heat. *Aviat Space Environ Med.* 1985;56:765–770.
62. Sandor RP. Heat illness: on-site diagnosis and cooling. *Physician Sportsmed.* 1997;25(6):35–40.
63. Squire DL. Heat illness: fluid and electrolyte issues for pediatric and adolescent athletes. *Pediatr Clin North Am.* 1990;37:1085–1109.
64. Murray R. Fluid needs in hot and cold environments. *Int J Sports Nutr.* 1995;5(suppl):62–73.
65. Gisolfi CV. Fluid balance for optimal performance. *Nutr Rev.* 1996;54(4 Pt 2, suppl):159–168.
66. Sawka MN, Coyle EF. Influence of body water and blood volume on thermoregulation and exercise performance in the heat. *Exerc Sport Sci Rev.* 1999;27:167–218.
67. Roberts WO. Medical management and administration manual for long distance road racing. In: Brown CH, Gudjonsson B, eds. *IAAF Medical Manual for Athletics and Road Racing Competitions: A Practical Guide.* Monaco: International Amateur Athletic Federation Publications; 1998: 39–75.
68. Kulka TJ, Kenney WL. Heat balance limits in football uniforms: how different uniform ensembles alter the equation. *Physician Sportsmed.* 2002;30(7):29–39.
69. Department of the Army. *Prevention Treatment and Control of Heat Injury.* Washington, DC: Department of the Army; 1980. Technical bulletin TBMED 507:1–21.
70. Hughson RL, Staudt LA, Mackie JM. Monitoring road racing in the heat. *Physician Sportsmed.* 1983;11(5):94–102.
71. American College of Sports Medicine. ACSM position statement: prevention of thermal injuries during distance running. *Med Sci Sports Exerc.* 1987;19:529–533.
72. Armstrong LE, Epstein Y, Greenleaf JE, et al. American College of Sports Medicine position stand: heat and cold illnesses during distance running. *Med Sci Sports Exerc.* 1996;28:i–x.
73. Rozycki TJ. Oral and rectal temperatures in runners. *Physician Sportsmed.* 1984;12(6):105–110.
74. Knight JC, Casa DJ, McClung JM, Caldwell KA, Gilmer AM, Meenan PM, Goss PJ. Assessing if two tympanic temperature instruments are valid predictors of core temperature in hyperthermic runners and does drying the ear canal help [abstract]. *J Athl Train.* 2000;35(suppl):S21.
75. Shapiro Y, Pandolf KB, Goldman RF. Predicting sweat loss response to exercise, environment and clothing. *Eur J Appl Physiol Occup Physiol.* 1982;48:83–96.
76. Shvartz E, Saar E, Benor D. Physique and heat tolerance in hot dry and hot humid environments. *J Appl Physiol.* 1973;34:799–803.
77. Murray R. Dehydration, hyperthermia, and athletes: science and practice. *J Athl Train.* 1996;31:248–252.
78. Pichan G, Gauttam RK, Tomar OS, Bajaj AC. Effects of primary hypohydration on physical work capacity. *Int J Biometerorol.* 1988;32: 176–180.
79. Walsh RM, Noakes TD, Hawley JA, Dennis SC. Impaired high-intensity cycling performance time at low levels of dehydration. *Int J Sports Med.* 1994;15:392–398.
80. Cheung SS, McLellan TM. Heat acclimation, aerobic fitness, and hydration effects on tolerance during uncompensable heat stress. *J Appl Physiol.* 1998;84:1731–1739.
81. Bijlani R, Sharma KN. Effect of dehydration and a few regimes of rehydration on human performance. *Indian J Physiol Pharmacol.* 1980; 24:255–266.
82. Nielsen B. Solar heat load: heat balance during exercise in clothed subjects. *Eur J Appl Physiol Occup Physiol.* 1990;60:452–456.
83. Maughan RJ, Shirreffs SM. Preparing athletes for competition in the heat: developing an effective acclimatization strategy. *Sports Sci Exchange.* 1997;10:1–4.
84. Lloyd EL. ABC of sports medicine: temperature and performance—II: heat. *BMJ.* 1994;309:587–589.
85. Pascoe DD, Shanley LA, Smith EW. Clothing and exercise, I: biophysics of heat transfer between the individual clothing and environment. *Sports Med.* 1994;18:38–54.
86. Anderson MK, Hall SJ. *Sports Injury Management.* Philadelphia, PA: Williams & Wilkins; 1995:66–75.
87. Roberts WO. Assessing core temperature in collapsed athletes: what's the best method? *Physician Sportsmed.* 1994;22(8):49–55.
88. Armstrong LE, Maresh CM, Crago AE, Adams R, Roberts RO. Interpretation of aural temperatures during exercise, hyperthermia, and cooling therapy. *Med Exerc Nutr Health.* 1994;3:9–16.
89. Adner MM, Scarlet JJ, Casey J, Robinson W, Jones BH. The Boston Marathon medical care team: ten years of experience. *Physician Sportsmed.* 1988;16(7):99–108.
90. Casa DJ, Maresh CM, Armstrong LE, et al. Intravenous versus oral rehydration during a brief period: responses to subsequent exercise in the heat. *Med Sci Sports Exerc.* 2000;32:124–133.
91. Noakes T. Failure to thermoregulate. In: Sutton J, Thompson M, Torode M, eds. *Exercise and Thermoregulation.* Sydney, Australia: The University of Sydney; 1995:37.
92. Deschamps A, Levy RD, Coslo MG, Marliss EB, Magder S. Tympanic temperature should not be used to assess exercise-induced hyperthermia. *Clin J Sport Med.* 1992;2:27–32.
93. Gonzalez-Alonso J, Mora-Rodriguez R, Coyle EF. Supine exercise restores arterial blood pressure and skin blood flow despite dehydration and hyperthermia. *Am J Physiol.* 1999;277(2 Pt 2):H576–H583.
94. Germain M, Jobin M, Cabanac M. The effect of face fanning during the recovery from exercise hyperthermia. *Can J Physiol Pharmacol.* 1987; 65:87–91.
95. Roberts WO. Exercise-associated collapse in endurance events: a classification system. *Physician Sportsmed.* 1989;17(5):49–55.
96. Matthew CB. Treatment of hyperthermia and dehydration with hypertonic saline in dextran. *Shock.* 1994;2:216–221.
97. Armstrong LE, Crago AE, Adams R, Roberts WO, Maresh CM. Whole-body cooling of hyperthermic runners: comparison to two field therapies. *Am J Emerg Med.* 1996;14:355–358.
98. Marino F, Booth J. Whole body cooling by immersion in water at moderate temperature. *J Sci Med Sport.* 1998;1:73–82.
99. Clements JM, Casa DJ, Knight JC, et al. Ice-water immersion and cold-water immersion provide similar cooling rates in runners with exercise-induced hyperthermia. *J Athl Train.* 2002;37:146–150.
100. Ash CJ, Cook JR, McMurry TA, Auner CR. The use of rectal temperature to monitor heat stroke. *Mo Med.* 1992;89:283–288.
101. Brechue WF, Stager JM. Acetazolamide alters temperature regulation during submaximal exercise. *J Appl Physiol.* 1990;69:1402–1407.
102. Kubica R, Nielsen B, Bonnesen A, Rasmussen IB, Stoklosa J, Wilk B. Relationship between plasma volume reduction and plasma electrolyte changes after prolonged bicycle exercise, passive heating and diuretic dehydration. *Acta Physiol Pol.* 1983;34:569–579.
103. Claremont AD, Costill DL, Fink W, Van Handel P. Heat tolerance following diuretic induced dehydration. *Med Sci Sports.* 1976;8:239–243.
104. Desruelle AV, Boisvert P, Candas V. Alcohol and its variable effect on human thermoregulatory response to exercise in a warm environment. *Eur J Appl Physiol Occup Physiol.* 1996;74:572–574.
105. Kalant H, Le AD. Effect of ethanol on thermoregulation. *Pharmacol Ther.* 1983;23:313–364.
106. Vanakoski J, Seppala T. Heat exposure and drugs: a review of the effects

of hyperthermia on pharmacokinetics. *Clin Pharmacokinet.* 1998;34: 311–322.
107. Shirreffs SM, Maughan RJ. Urine osmolality and conductivity as indices of hydration status in athletes in the heat. *Med Sci Sports Exerc.* 1998; 30:1598–1602.
108. Kaplan A, Szabo LL, Opheim KE. *Clinical Chemistry: Interpretations and Techniques.* 2nd ed. Philadelphia, PA: Lea & Febiger; 1983.
109. Ross D, Neely AE. *Textbook of Urinalysis and Body Fluids.* Norwalk, CT: Appleton-Century-Crofts; 1983.
110. Armstrong L. The impact of hyperthermia and hypohydration on circulation strength endurance and health. *J Appl Sport Sci Res.* 1998;2: 60–65.
111. Montain SJ, Sawka MN, Cadarette BS, Quigley MD, McKay JM. Physiological tolerance to uncompensable heat stress: effects of exercise intensity, protective clothing, and climate. *J Appl Physiol.* 1994;77:216–222.
112. Kenney WL, Hyde DE, Bernard TE. Physiological evaluation of liquid-barrier, vapor-permeable protective clothing ensembles for work in hot environments. *Am Ind Hyg Assoc J.* 1993;54:397–402.
113. Mathews DK, Fox EL, Tanzi D. Physiological responses during exercise and recovery in a football uniform. *J Appl Physiol.* 1969;26:611–615.
114. Armstrong LE. The nature of heatstroke during exercise. *Natl Strength Condition J.* 1992;14:80.
115. Wetterhall SF, Coulombier DM, Herndon JM, Zaza S, Cantwell JD. Medical care delivery at the 1996 Olympic Games: Centers for Disease Control and Prevention Olympics Surveillance Unit. *JAMA.* 1998;279: 1463–1468.
116. Cooper KE. Some responses of the cardiovascular system to heat and fever. *Can J Cardiol.* 1994;10:444–448.
117. Epstein Y. Heat intolerance: predisposing factor or residual injury? *Med Sci Sports Exerc.* 1990;22:29–35.
118. Chung NK, Pin CH. Obesity and the occurrence of heat disorders. *Mil Med.* 1996;161:739–742.
119. Gardner JW, Kark JA, Karnei K, et al. Risk factors predicting exertional heat illness in male Marine Corps recruits. *Med Sci Sports Exerc.* 1996; 28:939–944.
120. Hayward JS, Eckerson JD, Dawson BT. Effect of mesomorphy on hyperthermia during exercise in a warm, humid environment. *Am J Phys Anthropol.* 1986;70:11–17.
121. Kark JA, Burr PQ, Wenger CB, Gastaldo E, Gardner JW. Exertional heat illness in Marine Corps recruit training. *Aviat Space Environ Med.* 1996;67:354–360.
122. Piwonka RW, Robinson S, Gay VL, Manalis RS. Preacclimatization of men to heat by training. *J Appl Physiol.* 1965;20:379–384.
123. Noakes TD, Myburgh KH, du Plessis J, et al. Metabolic rate, not percent dehydration, predicts rectal temperature in marathon runners. *Med Sci Sports Exerc.* 1991;23:443–449.
124. Nadel ER, Pandolf KB, Roberts MF, Stolwijk JA. Mechanisms of thermal acclimation to exercise and heat. *J Appl Physiol.* 1974;37:515–520.
125. Walter FF, Bey TA, Ruschke DS, Benowitz NL. Marijuana and hyperthermia. *J Toxicol Clin Toxicol.* 1996;34:217–221.
126. Watson JD, Ferguson C, Hinds CJ, Skinner R, Coakley JH. Exertional heat stroke induced by amphetamine analogues: does dantrolene have a place? *Anaesthesia.* 1993;48:1057–1060.
127. Epstein Y, Albukrek D, Kalmovitc B, Moran DS, Shapiro Y. Heat intolerance induced by antidepressants. *Ann N Y Acad Sci.* 1997;813:553–558.
128. Stadnyk AN, Glezos JD. Drug-induced heat stroke. *Can Med Assoc J.* 1983;128:957–959.
129. Forester D. Fatal drug-induced heat stroke. *JACEP.* 1978;7:243–244.
130. Sarnquist F, Larson CP Jr. Drug-induced heat stroke. *Anesthesiology.* 1973;39:348–350.
131. Zelman S, Guillan R. Heat stroke in phenothiazine-treated patients: a report of three fatalities. *Am J Psychiatry.* 1970;126:1787–1790.
132. Gordon NF, Duncan JJ. Effect of beta-blockers on exercise physiology: implications for exercise training. *Med Sci Sports Exerc.* 1991;23:668–676.
133. Freund BJ, Joyner MJ, Jilka SM, et al. Thermoregulation during prolonged exercise in heat: alterations with beta-adrenergic blockade. *J Appl Physiol.* 1987;63:930–936.
134. Kew MC, Hopp M, Rothberg A. Fatal heat-stroke in a child taking appetite-suppressant drugs. *S Afr Med J.* 1982;62:905–906.
135. Lomax P, Daniel KA. Cocaine and body temperature: effect of exercise at high ambient temperature. *Pharmacology.* 1993;46:164–172.
136. Chen WL, Huang WS, Lin YF, Shieh SD. Changes in thyroid hormone metabolism in exertional heat stroke with or without acute renal failure. *J Clin Endocrinol Metab.* 1996;81:625–629.
137. Wemple RD, Lamb DR, McKeever KH. Caffeine vs caffeine-free sports drinks: effect on urine production at rest and during prolonged exercise. *Int J Sports Med.* 1997;18:40–46.
138. Odlind B. Site and mechanism of the action of diuretics. *Acta Pharmacol Toxicol (Copenh).* 1984;54(suppl 1):5–15.
139. Stookey JD. The diuretic effects of alcohol and caffeine and total water intake misclassification. *Eur J Epidemiol.* 1999;15:181–188.
140. Schlaeffer F, Engelberg I, Kaplanski J, Danon A. Effect of exercise and environmental heat on theophylline kinetics. *Respiration.* 1984;45:438–442.
141. Armstrong LE, Hubbard RW, Askew EW, et al. Responses to moderate and low sodium diets during exercise-heat acclimation. *Int J Sport Nutr.* 1993;3:207–221.
142. Armstrong LE, Szlyk PC, DeLuca JP, Sils IV, Hubbard RW. Fluid-electrolyte losses in uniforms during prolonged exercise at 30 degrees C. *Aviat Space Environ Med.* 1992;63:351–355.
143. Mendyka BE. Fluid and electrolyte disorders caused by diuretic therapy. *AACN Clin Issues Crit Care Nurs.* 1992;3:672–680.
144. Melby JC. Selected mechanisms of diuretic-induced electrolyte changes. *Am J Cardiol.* 1986;58:1A–4A.
145. Bourdon L, Canini F. On the nature of the link between malignant hyperthermia and exertional heatstroke. *Med Hypotheses.* 1995;45:268–270.
146. Dixit SN, Bushara KO, Brooks BR. Epidemic heat stroke in midwest community: risk factors, neurological complications, and sequelae. *Wis Med J.* 1997;96:39–41.
147. Hunter SL, Rosenberg H, Tuttle GH, DeWalt JL, Smodie R, Martin J. Malignant hyperthermia in a college football player. *Physician Sportsmed.* 1987;15(12):77–81.
148. Lazarus A. Differentiating neuroleptic-related heatstroke from neuroleptic malignant syndrome. *Psychosomatics.* 1989;30:454–456.
149. Rampertaap MP. Neuroleptic malignant syndrome. *South Med J.* 1986; 79:331–336.
150. Addonizio G, Susman V. Neuroleptic malignant syndrome and heat stroke. *Br J Psychiatry.* 1984;145:556–557.
151. Martin ML, Lucid EJ, Walker RW. Neuroleptic malignant syndrome. *Ann Emerg Med.* 1985;14:354–358.
152. Virmani R, Robinowitz M. Cardiac pathology and sports medicine. *Hum Pathol.* 1987;18:493–501.
153. Buchwald I, Davis PJ. Scleroderma with fatal heat stroke. *JAMA.* 1967; 201:270–271.
154. Smith HR, Dhatt GS, Melia WM, Dickinson JG. Cystic fibrosis presenting as hyponatraemic heat exhaustion. *BMJ.* 1995;310:579–580.
155. Andrews C, Mango M, Venuto RC. Cystic fibrosis in adults. *Ann Intern Med.* 1978;88:128–129.
156. Kerle KK, Nishimura KD. Exertional collapse and sudden death associated with sickle cell trait. *Am Fam Physician.* 1996;54:237–240.
157. Gardner JW, Kark JA. Fatal rhabdomyolysis presenting as mild heat illness in military training. *Mil Med.* 1994;159:160–163.
158. Kenney WL. Thermoregulation during exercise in the heat. *Athl Ther Today.* 1996;1:13–16.
159. Tilley RI, Standerwick JM, Long GJ. Ability of the Wet Bulb Globe Temperature Index to predict heat stress in men wearing NBC protective clothing. *Mil Med.* 1987;152:554–556.
160. Rasch W, Cabanac M. Selective brain cooling is affected by wearing headgear during exercise. *J Appl Physiol.* 1993;74:1229–1233.
161. Sheffield-Moore M, Short KR, Kerr CG, Parcell AC, Bolster DR, Costill DL. Thermoregulatory responses to cycling with and without a helmet. *Med Sci Sports Exerc.* 1997;29:755–761.
162. Shapiro Y, Pandolf KB, Avellini BA, Pimental NA, Goldman RF. Phys-

iological responses of men and women to humid and dry heat. *J Appl Physiol.* 1980;49:1–8.
163. Yaglou CP, Minard D. Control of heat casualties at military training centers. *Arch Ind Health.* 1957;16:302–305.
164. Bracker MO. Hyperthermia: man's adaptation to a warm climate. *Sports Med Dig.* 1991;13:1–2.
165. Johnson SC, Ruhling RO. Aspirin in exercise-induced hyperthermia: evidence for and against its role. *Sports Med.* 1985;2:1–7.
166. Werner J. Central regulation of body temperature. In: Gisolfi C, ed. *Exercise, Heat, and Thermoregulation.* Carmel, IN: Cooper Publishing; 1993:7–35.
167. Galaski MJ. Hyperthermia. *J Can Athl Ther.* 1985;12;23–26.
168. Yaqub BA. Neurologic manifestations of heatstroke at the Mecca pilgrimage. *Neurology.* 1987;37:1004–1006.
169. Armstrong LE. *Keeping Your Cool in Barcelona: The Effects of Heat Humidity and Dehydration on Athletic Performance Strength and Endurance.* Colorado Springs, CO: United States Olympic Committee Sports Sciences Division; 1992:1–29.
170. Anderson GS, Meneilly GS, Mekjavic IB. Passive temperature lability in the elderly. *Eur J Appl Physiol Occup Physiol.* 1996;73:278–286.
171. Candas V, Libert JP, Vogt JJ. Influence of air velocity and heat acclimation on human skin wettedness and sweating efficiency. *J Appl Physiol.* 1979;47:1194–2000.
172. Berglund LG, Gonzalez RR. Evaporation of sweat from sedentary man in humid environments. *J Appl Physiol.* 1977;42:767–772.
173. Gabrys J, Pieniazek W, Olejnik I, Pogorzelska T, Karpe J. Effects of local cooling of neck circulatory responses in men subjected to physical exercise in hyperthermia. *Biol Sport.* 1993;10:167–171.
174. Royburt M, Epstein Y, Solomon Z, Shemer J. Long-term psychological and physiological effects of heat stroke. *Physiol Behav.* 1993;54:265–267.
175. Mehta AC, Baker RN. Persistent neurological deficits in heat stroke. *Neurology.* 1970;20:336–340.
176. McArdle WD, Katch FI, Katch VL. *Exercise Physiology*. 3rd ed. Philadelphia, PA: Lea & Febiger; 1991:556–570.
177. Avellini BA, Kamon E, Krajewski JT. Physiological responses of physically fit men and women to acclimation to humid heat. *J Appl Physiol.* 1980;49:254–261.
178. Geor RJ, McCutcheon LJ. Thermoregulatory adaptations associated with training and heat acclimation. *Vet Clin North Am Equine Pract.* 1988; 14:97–120.
179. Nielsen B. Heat stress and acclimation. *Ergonomics.* 1994;37:49–58.
180. Gisolfi CV, Wenger CB. Temperature regulation during exercise: old concepts, new ideas. *Exerc Sport Sci Rev.* 1984;12:339–372.
181. Morimoto T, Miki K, Nose H, Yamada S, Hirakawa K, Matsubara D. Changes in body fluid and its composition during heavy sweating and effect of fluid and electrolyte replacement. *Jpn J Biometeorol.* 1981;18: 31–39.
182. Pandolf KB, Cadarette BS, Sawka MN, Young AJ, Francesconi RP, Gonzalez RR. Thermoregulatory responses of middle-aged and young men during dry-heat acclimation. *J Appl Physiol.* 1998;65:65–71.
183. Pandolf KB, Burse RL, Goldman RF. Role of physical fitness in heat acclimatisation, decay and reinduction. *Ergonomics.* 1977;20:399–408.
184. Cadarette BS, Sawka MN, Toner MM, Pandolf KB. Aerobic fitness and the hypohydration response to exercise-heat stress. *Aviat Space Environ Med.* 1984;55:507–512.
185. Buskirk ER, Iampietro PF, Bass DE. Work performance after dehydration: effects of physical conditioning and heat acclimatization. *J Appl Physiol.* 1958;12:789–794.
186. Adams J, Fox R, Grimby G, Kidd D, Wolff H. Acclimatization to heat and its rate of decay in man. *J Physiol.* 1960;152:26P–27P.
187. Czerkawski JT, Meintod A, Kleiner DM. Exertional heat illness: teaching patients when to cool it. *Your Patient Fitness.* 1996;10:13–20.
188. Wyndham C, Strydom N, Cooks H, et al. Methods of cooling subjects with hyperpyrexia. *J Appl Physiol.* 1959;14:771–776.
189. Hayward JS, Collis M, Eckerson JD. Thermographic evaluation of relative heat loss areas of man during cold water immersion. *Aerosp Med.* 1973;44:708–711.
190. Tsuzuki-Hayakawa K, Tochihara Y, Ohnaka T. Thermoregulation during heat exposure of young children compared to their mothers. *Eur J Appl Physiol Occup Physiol.* 1995;72:12–17.
191. Bar-Or O. Children's responses to exercise in hot climates: implications for performance and health. *Sports Sci Exerc.* 1994;7:1–5.
192. Davies CT. Thermal responses to exercise in children. *Ergonomics.* 1981;24:55–61.
193. Docherty D, Eckerson JD, Hayward JS. Physique and thermoregulation in prepubertal males during exercise in a warm, humid environment. *Am J Phys Anthropol.* 1986;70:19–23.
194. Armstrong LE, Maresh CM. Exercise-heat tolerance of children and adolescents. *Pediatr Exerc Sci.* 1995;7:239–252.
195. Gutierrez GG. Solar injury and heat illness: treatment and prevention in children. *Physician Sportsmed.* 1995;23(7):43–48.
196. Nash HL. Hyperthermia: risks greater in children. *Physician Sportsmed.* 1987;15(2):29.
197. American Academy of Pediatrics Committee on Sports Medicine. Climatic heat stress and the exercising child. *Pediatrics.* 1982;69:808–809.
198. Kenney WL, Hodgson JL. Heat tolerance, thermoregulation, and ageing. *Sports Med.* 1987;4:446–456.
199. Wagner JA, Robinson S, Tzankoff SP, Marino RP. Heat tolerance and acclimatization to work in the heat in relation to age. *J Appl Physiol.* 1972;33:616–622.
200. Pandolf KB. Heat tolerance and aging. *Exp Aging Res.* 1994;20:275–284.
201. Pandolf KB. Aging and human heat tolerance. *Exp Aging Res.* 1997;23: 69–105.
202. Kenney W. The older athlete: exercise in hot environments. *Sports Sci Exerc.* 1993;6:1–4.
203. Inoue Y, Shibasaki M, Hirata K, Araki T. Relationship between skin blood flow and sweating rate and age related regional differences. *Eur J Appl Physiol Occup Physiol.* 1998;79:17–23.
204. Sagawa S, Shiraki K, Yousef MK, Miki K. Sweating and cardiovascular responses of aged men to heat exposure. *J Gerontol.* 1988;43:M1–M8.
205. Inoue Y, Shibasaki M. Regional differences in age-related decrements of the cutaneous vascular and sweating responses to passive heating. *Eur J Appl Physiol Occup Physiol.* 1996;74:78–84.
206. Inoue Y, Shibasaki M, Ueda H, Ishizashi H. Mechanisms underlying the age-related decrement in the human sweating response. *Eur J Appl Physiol Occup Physiol.* 1999;79:121–126.
207. Pandolf KB, Cafarelli E, Noble BJ, Metz KF. Hyperthermia: effect on exercise prescription. *Arch Phys Med Rehabil.* 1975;56:524–526.
208. Zappe DH, Bell GW, Swartzentruber H, Wideman RF, Kenney WL. Age and regulation of fluid and electrolyte balance during repeated exercise sessions. *Am J Physiol.* 1996;207(1 Pt 2):R71–R79.
209. Binkhorst RA, Hopman MT. Heat balance in paraplegic individuals during arm exercise at 10 and 35°C. *Med Sci Sports Exerc.* 1995;27(suppl): 83.
210. Clark MW. The physically challenged athlete. *Adolesc Med.* 1998;9: 491–499.
211. Bloomquist LE. Injuries to athletes with physical disabilities: prevention implications. *Physician Sportsmed.* 1986;14(9):96–100,102,105.
212. Hopman MT, Binkhourst RA. Spinal cord injury and exercise in the heat. *Sports Sci Exerc.* 1997;10:1–4.
213. Armstrong LE, Maresh CM, Riebe D, et al. Local cooling in wheelchair athletes during exercise-heat stress. *Med Sci Sports Exerc.* 1995;27:211–216.
214. Sawka MN, Latzka WA, Pandolf KB. Temperature regulation during upper body exercise: able-bodied and spinal cord injured. *Med Sci Sports Exerc.* 1989;21(5 suppl):132–140.
215. Hopman MT, Oeseburg B, Binkhorst RA. Cardiovascular responses in persons with paraplegia to prolonged arm exercise and thermal stress. *Med Sci Sports Exerc.* 1993;25:577–583.
216. Petrofsky JS. Thermoregulatory stress during rest and exercise in heat in patients with a spinal cord injury. *Eur J Appl Physiol Occup Physiol.* 1992;64:503–507.
217. Bracker MD. Environmental and thermal injury. *Clin Sports Med.* 1992; 11:419–436.
218. Hopman MT. Circulatory responses during arm exercise in individuals with paraplegia. *Int J Sports Med.* 1994;15:126–131.

219. Yamaski M, Kim KT, Choi SW, Muraki S, Shiokawa M, Kurokawa T. Characteristics of body heat balance of paraplegics during exercise in a hot environment. *J Physiol Anthropol Appl Human Sci.* 2001;20:227–232.
220. Gass GC, Camp EM, Nadel ER, Gwinn TH, Engel P. Rectal and rectal vs. esophageal temperatures in paraplegic men during prolonged exercise. *J Appl Physiol.* 1998;64:2265–2271.
221. Yaqub BA, Al-Harthi SS, Al-Orainey IO, Laajam MA, Obeid MT. Heat stroke at the Mekkah pilgrimage: clinical characteristics and course of 30 patients. *Q J Med.* 1986;59:523–530.
222. Hubbard RW. The role of exercise in the etiology of exertional heatstroke. *Med Sci Sports Exerc.* 1990;22:2–5.
223. Holman ND, Schneider AJ. Multi-organ damage in exertional heat stroke. *Neth J Med.* 1989;35:38–43.
224. Shibolet S, Coll R, Gilat T, Sohar E. Heatstroke: its clinical picture and mechanism in 36 cases. *Q J Med.* 1965;36:525–548.
225. Gummaa K, El-Mahrouky S, Mahmoud H, Mustafa K, Khogall M. The metabolic status of heat stroke patients: the Makkah experience. In: Khogali M, Hale JR, eds. *Heat Stroke and Temperature Regulation.* New York, NY: Academic Press; 1983:157–169.
226. Garcia-Rubira JC, Aguilar J, Romero D. Acute myocardial infarction in a young man after heat exhaustion. *Int J Cardiol.* 1995;47:297–300.
227. Senay LC, Kok R. Body fluid responses of heat-tolerant and intolerant men to work in a hot wet environment. *J Appl Physiol.* 1976;40:55–59.
228. Shvartz E, Shibolet S, Merez A, Magazanik A, Shapiro Y. Prediction of heat tolerance from heart rate and rectal temperature in a temperate environment. *J Appl Physiol.* 1977;43:684–688.
229. Strydom NB. Heat intolerance: its detection and elimination in the mining industry. *S Afr J Sci.* 1980;76:154–156.
230. Robergs RA, Roberts SO. *Exercise Physiology: Exercise, Performance, and Clinical Applications.* St Louis, MO: Mosby; 1997:653–662.