



**AMERICAN COLLEGE  
of SPORTS MEDICINE®**

POSITION STAND

# Exertional Heat Illness during Training and Competition

This pronouncement was written for the American College of Sports Medicine by Lawrence E. Armstrong, Ph.D., FACSM (Chair); Douglas J. Casa, Ph.D., ATC, FACSM; Mindy Millard-Stafford, Ph.D., FACSM, Daniel S. Moran, Ph.D., FACSM; Scott W. Pyne, M.D., FACSM; and William O. Roberts, M.D., FACSM.

## SUMMARY

Exertional heat illness can affect athletes during high-intensity or long-duration exercise and result in withdrawal from activity or collapse during or soon after activity. These maladies include exercise associated muscle cramping, heat exhaustion, or exertional heatstroke. While certain individuals are more prone to collapse from exhaustion in the heat (i.e., not acclimatized, using certain medications, dehydrated, or recently ill), exertional heatstroke (EHS) can affect seemingly healthy athletes even when the environment is relatively cool. EHS is defined as a rectal temperature greater than 40°C accompanied by symptoms or signs of organ system failure, most frequently central nervous system dysfunction. Early recognition and rapid cooling can reduce both the morbidity and mortality associated with EHS. The clinical changes associated with EHS can be subtle and easy to miss if coaches, medical personnel, and athletes do not maintain a high level of awareness and monitor at-risk athletes closely. Fatigue and exhaustion during exercise occur more rapidly as heat stress increases and are the most common causes of withdrawal from activity in hot conditions. When athletes collapse from exhaustion in hot conditions, the term heat exhaustion is often applied. In some cases, rectal temperature is the only discernable difference between severe heat exhaustion and EHS in on-site evaluations. Heat exhaustion will generally resolve with symptomatic care and oral fluid support. Exercise associated muscle cramping can occur with exhaustive work in any temperature range, but appears to be more prevalent in hot and humid conditions. Muscle cramping usually responds to rest and replacement of fluid and salt (sodium). Prevention strategies are essential to reducing the incidence of EHS, heat exhaustion, and exercise associated muscle cramping.

## INTRODUCTION

This document replaces, in part, the 1996 Position Stand titled "Heat and Cold Illnesses during Distance Running" (9) and considers selected heat related medical conditions (EHS, heat exhaustion, and exercise associated muscle cramping) that may affect active people in warm or hot environments. These recommendations are intended to

reduce the morbidity and mortality of exertional heat-related illness during physical activity, but individual physiologic responses to exercise and daily health status are variable, so compliance with these recommendations will not guarantee protection.

Heat illness occurs world wide with prolonged intense activity in almost every venue (e.g., cycling, running races, American football, soccer). EHS (1,27,62,64,65,109,132, 154,160,164) and heat exhaustion (54,71,149,150) occur most frequently in hot-humid conditions, but can occur in cool conditions, during intense or prolonged exercise (133). Heat exhaustion and exercise related muscle cramps do not typically involve excessive hyperthermia, but rather are a result of fatigue, body water and/or electrolyte depletion, and/or central regulatory changes that fail in the face of exhaustion.

This document will address recognition, treatment, and incidence reduction for heat exhaustion, EHS, and exercise associated muscle cramping, but does not include anesthesia-induced malignant hyperthermia, sunburn, anhidrotic heat exhaustion, or sweat gland disorders that are classified in other disease categories, because these disorders may or may not involve exercise or be solely related to heat exposure. Hyponatremia also occurs more frequently during prolonged activity in hot conditions, but is usually associated with excessive fluid intake and is addressed in the ACSM Exercise and Fluid Replacement Position Stand.

Evidence statements in this document are based on the strength of scientific evidence with regard to clinical outcomes. Because research ethics preclude the use of human subjects in the study of EHS and other exertional heat illnesses, this document employs the following criteria: A, recommendation based on consistent and good-quality patient- or subject-oriented evidence; B, recommendation based on inconsistent or limited-quality patient- or subject-oriented evidence; C, recommendation based on consensus, usual practice, opinion, disease-oriented evidence, or a case series for studies of diagnosis, treatment, prevention, or screening.

0195-9131/07/3903-0557/0

MEDICINE & SCIENCE IN SPORTS & EXERCISE®

Copyright © 2007 by the American College of Sports Medicine

DOI: 10.1249/MSS.0b013e31802fa199

## General Background: Exhaustion, Hyperthermia, and Dehydration

Exhaustion is a physiologic response to work defined as the inability to continue exercise and occurs with heavy exertion in all temperature ranges. As ambient temperature increases beyond 20°C (68°F) and heat stress rises, the time to exhaustion decreases (58). From a clinical perspective it is difficult to distinguish athletes with exhaustion in cool conditions from those who collapse in hot conditions. Exercise that must be stopped due to exhaustion is likely triggered by some combination of hyperthermia-induced reduction of peripheral muscle activation due to decreased central activation (brain fatigue) (110,118), hydration level, peripheral effect of hyperthermia on muscle fatigue, depletion of energy stores, electrolyte imbalance, and/or other factors. Some combination of central, spinal cord, and peripheral responses to hyperthermia factor into the etiology of withdrawal or collapse from exhaustion during activity; the exact mechanisms have yet to be explained (90,114–116,171). The exercise-related exhaustion that occurs in hot conditions may be an extension of this phenomenon, but it is more pronounced, because depletion of energy stores occurs faster in hotter conditions, especially when athletes are not acclimatized to exercise in the heat (71). When physiologic exhaustion results in collapse, the clinical syndrome is often referred to as heat exhaustion. In both hot and cool environments, postexercise collapse also may be due to postural hypotension rather than heat exhaustion and postural changes usually resolve with leg elevation and rest in less than 30 min.

There are several variables that affect exhaustion in athletes including duration and intensity of exercise, environmental conditions, acclimatization to exercise-heat stress, innate work capacity ( $\dot{V}O_{2max}$ ), physical conditioning, hydration status, and personal factors like medications, supplements, sleep, and recent illness. In human studies of exercise time to exhaustion at a fixed exercise load, both individuals and groups show a decrease in exercise capacity (time to exhaustion) and an increase in perceived exertion as environmental temperature and/or relative humidity increase and/or as total body water decreases. The combined effects of heat stress and dehydration reduce exercise capacity and performance to a greater degree than either alone. Compared to more moderate conditions, an athlete in hot conditions must either slow the pace to avoid collapse or maintain the pace and risk collapse before the task is completed.

**Evidence statement.** Dehydration reduces endurance exercise performance, decreases time to exhaustion, increases heat storage (11,12,16,41,57,141). *Evidence category A.*

Exertional hyperthermia, defined as a core body temperature above 40°C (104°F) (71,85,86,149,150), occurs during athletic or recreational activity and is influenced by exercise intensity, environmental conditions, clothing, equipment, and individual factors. Hyperthermia occurs during exercise

when muscle-generated heat accumulates faster than heat dissipates via increased sweating and skin blood flow (3). Heat production during intense exercise is 15–20 times greater than at rest, and can raise core body temperature by 1°C (1.8°F) every 5 min if no heat is removed from the body (105). Prolonged hyperthermia may lead to EHS, a life-threatening condition with a high mortality rate if not promptly recognized and treated with body cooling.

The removal of body heat is controlled by central nervous system (CNS) centers in the hypothalamus and spinal cord, and peripheral centers in the skin and organs. Heat flow to maintain a functional core temperature requires a temperature gradient from the body core to the body shell. If the skin temperature remains constant, the gradient increases as the core temperature increases during exercise, augmenting heat removal. If the shell or skin temperature also rises during exercise, as a result of either the environment or internal heat production, the core to skin gradient may be lost (i.e., reducing heat dissipation) and the core temperature increases.

Wide variations of heat tolerance exist among athletes. The extent to which elevated body temperature below 40°C diminishes exercise performance and contributes to heat exhaustion (110) is unknown, but there is considerable attrition from exercise when rectal temperatures reach 39–40°C (144). In controlled laboratory studies, precooling the body will extend the time to exhaustion and preheating will shorten the time to exhaustion, but in both circumstances athletes tend to terminate exercise due to fatigue at a rectal temperature of about 40°C (104°F) (61).

In recent years, the importance of hyperthermia in fatigue and collapse has been investigated. These studies have shown that the brain temperature is always higher than core temperature and heat removal is decreased in the hyperthermic brain compared to control (119). Also, as brain temperature increases from 37 to 40°C during exercise, cerebral blood flow and maximal voluntary muscular force output decrease with concurrent changes in brain wave activity and perceived exertion (110,118). Brain hyperthermia may explain why some exercising individuals collapse with exhaustion, while others are able to override central nervous system controls and push themselves to continue exercising strenuously and develop life-threatening EHS.

It is not unusual for some athletes to experience prolonged hyperthermia without noticeable medical impairment, especially during competition. Elevated rectal temperatures up to 41.9°C (107.4°F) have been noted in soccer players, American football lineman, road runners, and marathoners who show no symptoms or signs of heat related physical changes (21,42,46,98,125,129,130,132, 161,165,176). This is significant because some athletes tolerate rectal temperatures well above the widely accepted threshold for EHS of >40°C without obvious clinical sequelae (71,85,86,104,149,150).

Dehydration occurs during prolonged exercise, more rapidly in hot environments when participants lose

considerably more sweat than can be replaced by fluid intake (3,72,126). When fluid deficits exceed 3–5% of body weight, sweat production and skin blood flow begin to decline (19) reducing heat dissipation. Water deficits of 6–10% of body weight occur in hot weather, with or without clinically significant losses of sodium ( $\text{Na}^+$ ) and chloride ( $\text{Cl}^-$ ) (25,45,71,100,102,155,173) and reduce exercise tolerance by decreasing cardiac output, sweat production, and skin and muscle blood flow (12,41,57,71,101,141,142). Dehydration may be either a direct (i.e., heat exhaustion, exercise associated muscle cramps) or indirect (i.e., heatstroke) factor in heat illness (10). Excessive sweating also results in salt loss, which has been implicated in exercise associated muscle cramps and in salt loss hyponatremia during long-duration (>8 h) endurance events in the heat.

In one study illustrating the cumulative affects of heat stress, a male soldier (32 yr, 180 cm, 110.47 kg, 41.4  $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) participating in monitored, multiday, high-intensity exercise regimen at 41.2°C (106.0°F), 39% RH was asymptomatic with a postexercise rectal temperature of 38.3–38.9°C on days 3–7 (16). From the morning of day 5 to day 8, he lost 5.4 kg of body weight (4.8%) and had an increase of baseline heart rate, skin temperature and rectal temperature during days 6 and 7. On day 8, he developed heat exhaustion with unusual fatigue, muscular weakness, abdominal cramps, and vomiting with a rectal temperature of 39.6°C (103.3°F). His blood endorphin and cortisol levels were 6 and 2 times greater, respectively, than the other study subjects on day 8, indicating severe exercise-heat intolerance. Thirteen other males who maintained body weight near their prestudy baseline completed this protocol without incident. Because day-to-day dehydration affects heat tolerance, physical signs and hydration status should be monitored to reduce the incidence of heat exhaustion in hot environments.

When humans exercise near maximal levels, splanchnic and skin blood flow decrease as skeletal muscle blood flow increases to provide plasma glucose, remove heat, and remove metabolic products from working muscles (70). As the central controls for blood flow distribution fatigue due to a core temperature increase, the loss of compensatory splanchnic and skin vasoconstriction results in reduction of the total vascular resistance and worsens cardiac insufficiency (71,84). The loss of splanchnic vasoconstriction during exhaustion has been reproduced in a laboratory rat model and supports the assertion that loss of splanchnic vasoconstriction plays a role in heat exhaustion in athletes (70,73,84). This mechanism partially explains why exertional collapse is less likely to occur in cool environments, where cool, vasoconstricted skin helps maintain both cardiac filling and mean arterial pressure, and prolongs the time to exhaustion.

How EHS and heat exhaustion evolve, and in what sequence, are not completely understood (106). Some athletes tolerate hot conditions, dehydration, and hyper-

thermia well and are seemingly unaffected, while others discontinue activity in relatively less stressful conditions. The path that leads to EHS has been assumed to pass through heat exhaustion, however anecdotal and case study data seem to refute that notion as EHS can occur in relatively fresh athletes who develop symptomatic hyperthermia in 30–60 min of road racing in hot, humid conditions with no real signs of dehydration or heat exhaustion. If these athletes have heat exhaustion, then the duration and transition must be very short. Heat exhaustion should be protective for athletes in that, once exercise is stopped, the risk of developing exertional heat stroke is reduced because exercise-induced metabolic heat production decreases and heat dissipation to the environment increases. A program of prudent exercise in the heat along with acclimatization, improved cardiorespiratory physical fitness, and reasonable fluid replacement during exercise reduce the risk and incidence of both problems.

**Evidence statement.** Exertional heatstroke (EHS) is defined in the field by rectal temperature >40°C at collapse and by central nervous system changes. *Evidence category B.*

## EXERTIONAL HEAT ILLNESSES

### Exertional Heatstroke

**Etiology.** Exertional heatstroke (EHS) is defined by hyperthermia (core body temperature >40°C) associated with central nervous system disturbances and multiple organ system failure. When the metabolic heat produced by muscle during activity outpaces body heat transfer to the surroundings, the core temperature rises to levels that disrupt organ function. Almost all EHS patients exhibit sweat-soaked and pale skin at the time of collapse, as opposed to the dry, hot, and flushed skin that is described in the presentation of non-exertion-related (classic) heatstroke (162).

**Predisposing factors.** Although strenuous exercise in a hot-humid environment, lack of heat acclimatization, and poor physical fitness are widely accepted as the primary factors leading to EHS, even highly trained and heat-acclimatized athletes develop EHS while exercising at a high intensity if heat dissipation is inadequate relative to metabolic heat production (18,34,71). The greatest risk for EHS exists when the wet bulb globe temperature (WBGT) exceeds 28°C (82°F) (20,81,156) during high-intensity exercise (>75%  $\dot{V}\text{O}_{2\text{max}}$ ) and/or strenuous exercise that lasts longer than 1 h as outlined below in “Monitoring the Environment.” EHS also can occur in cool (8–18°C [45–65°F]) to moderate (18–28°C [65–82°F]) environments (14,56,132,133), suggesting that individual variations in susceptibility (14,22,55,56,66) may be due to inadequate physical fitness, incomplete heat acclimatization, or other temporary factors like viral illness or medications (81,133).

**Evidence statement.** Ten to 14 days of exercise training in the heat will improve heat acclimatization and reduce the risk of EHS. *Evidence category C.*

The risk of EHS rises substantially when athletes experience multiple stressors such as a sudden increase in physical training, lengthy initial exposure to heat, vapor barrier protective clothing, sleep deprivation (14), inadequate hydration, and poor nutrition. The cumulative effect of heat exposure on previous days raises the risk of EHS, especially if the ambient temperature remains elevated overnight (14,168). Over-the-counter drugs and nutritional supplements containing ephedrine, synephrine, ma huang and other sympathomimetic compounds may increase heat production (23,121), but require verification as a cause of hyperthermia by controlled laboratory studies or field trials.

Appropriate fluid ingestion before and during exercise minimizes dehydration and reduces the rate at which core body temperature rises (46,60). However, hyperthermia may occur in the absence of significant dehydration when a fast pace or high-intensity exercise generates more metabolic heat than the body can remove (18,34,165). Skin disease (i.e., *miliaria rubra*), sunburn, alcohol use, drug abuse (i.e., ecstasy), antidepressant medications (69), obesity, age >40 yr, genetic predisposition to malignant hyperthermia, and a history of heat illness also have been linked to an increased risk of EHS in athletes (14,55, 85,150). Athletes should not exercise in a hot environment if they have a fever, respiratory infection, diarrhea, or vomiting (14,81). A study of 179 heat casualties at a 14-km race over 9 yr showed that 23% reported a recent gastrointestinal or respiratory illness (128). A similar study of 10 military patients with EHS reported that three had a fever and six recalled at least one warning sign of impending illness prior to collapse (14).

In American football, EHS usually occurs during the initial 4 d of preseason practice, which for most players takes place during the hottest and most humid time of the summer when athletes are the least fit. This emphasizes the importance of gradually introducing activity to induce acclimatization, carefully monitoring changes in behavior or performance during practices, and selectively modifying exercise (i.e., intensity, duration, rest periods) in high-risk conditions. Three factors may influence the early season EHS risk in American football players: (a) failure of coaches to adjust the intensity of the practice to the current environmental conditions, following the advice of the sports medicine staff; (b) unfit and unacclimatized players practicing intensely in the heat; and (c) vapor barrier equipment introduced before acclimatization.

One study of 10 EHS cases (14) reported that eight incidents occurred during group running at a 12.1–13.8 km·h<sup>-1</sup> pace in environmental temperatures of ≥25°C (77°F), suggesting that some host factor altered exercise-heat tolerance on the day that EHS occurred. Heat tolerance is often less in individuals who have the lowest maximal aerobic power (i.e.,  $\dot{V}O_{2max} \leq 40 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) (14,64,96). To maintain pace when running in a group, these less fit individuals must function at higher exercise

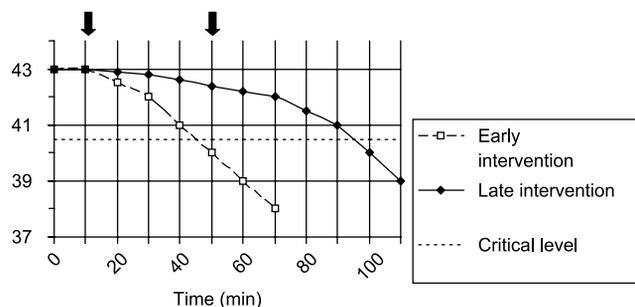
intensities to maintain the group's pace and are likely to have higher rectal temperatures at the end of a run compared to individuals with a higher  $\dot{V}O_{2max}$ . Air flow and heat dissipation also are reduced for runners in a pack.

More clinical and scientific reports of EHS involve males, and some hypotheses have been advanced (14). First, men may simply be in more EHS prone situations (i.e., military combat and American football). Second, men may be predisposed because of gender-specific hormonal, physiological, psychological, or morphological (i.e., muscle mass, body surface area-to-mass ratio) differences. Women, however, are not immune to the disorder, and the number of women who experience EHS may rise with the increased participation of women in strenuous sports.

**Evidence statement.** The following conditions increase the risk of EHS: obesity, low physical fitness level, lack of heat acclimatization, dehydration, a previous history of EHS, sleep deprivation, sweat gland dysfunction, sunburn, viral illness, diarrhea, or certain medications. *Evidence category B.* Physical training, cardiorespiratory fitness, and heat acclimatization reduce the risk of EHS. *Evidence category C.*

**Pathophysiology.** The underlying pathophysiology of EHS occurs when internal organ tissue temperatures rise above critical levels, cell membranes are damaged, and cell energy systems are disrupted, giving rise to a characteristic clinical syndrome (56,149). As a cell is heated beyond its thermal threshold (i.e., about 40°C), a cascade of events occurs that disrupts cell volume, metabolism, acid–base balance, and membrane permeability leading initially to cell and organ dysfunction and finally to cell death and organ failure (71,91,175). This complex cascade of events explains the variable onset of brain, cardiac, renal, gastrointestinal, hematologic, and muscle dysfunction among EHS patients.

The extent of multisystem tissue morbidity and the mortality rate are directly related to the area in degree-minutes under the body core temperature vs. time graph and



**FIGURE 1**—Cooling curves for early and late cooling interventions. The area under the early intervention curve above 40.5°C (the dashed line) in degree-minutes is approximately 60 while the area under the late intervention curve (cooling at 50 min) is >145. The prognosis based on area under the cooling curve for the late intervention is poor. Cooling can be delayed when heat stroke is not recognized early in the evaluation or if the athlete is transported before cooling is initiated. The arrow marks the start of cooling at 10 min for early intervention and 50 min for late intervention.

the length of time required to cool central organs to  $<40^{\circ}\text{C}$  (14,20,47,48). Tissue thresholds and the duration of temperature elevation, rather than the peak core body temperature, determine the degree of injury (72). When cooling is rapidly initiated and both the body temperature and cognitive function return to the normal range within an hour of onset of symptoms, most EHS patients recover fully (47,48). EHS victims who are recognized and cooled immediately theoretically tolerate about  $60^{\circ}\text{C}\cdot\text{min}$  ( $120^{\circ}\text{F}\cdot\text{min}$ ; area under the cooling curve) above  $40.5^{\circ}\text{C}$  without lasting sequelae (see Fig. 1). Conversely, athletes with EHS who go unrecognized or are not cooled quickly, and have more than  $60^{\circ}\text{C}\cdot\text{min}$  of temperature elevation above  $40.5^{\circ}\text{C}$ , tend to have increased morbidity and mortality. Outcomes of 20 “light” and 16 “severe” cases of EHS during military training (150) showed that coma was relatively brief in light cases when hyperthermia was limited to  $<1$  h, despite evidence of multiple organ involvement that was confirmed with elevated serum muscle and liver enzymes (74,172). Severe EHS cases were moribund at the time of admission and died early with evident central nervous system damage (150). The primary difference between light and severe EHS cases appears to be the length of time between collapse and the initiation of cooling therapy (14,20,47,48).

Hyperthermia of heart muscle tissue directly suppresses cardiac function, but the dysfunction is reversible with body cooling, as demonstrated by echocardiography (133). Cardiac tissue hyperthermia reduces cardiac output, oxygen delivery to tissues, and the vascular transport of heat from deep tissues to the skin. Cardiac insufficiency or failure associated with hyperthermia accelerates the elevation of core temperature and increases tissue hypoxia, metabolic acidosis and organ dysfunction. The concurrent heating of the brain begins a cascade of cerebral and hypothalamic failure that also accelerates cell death by disrupting the regulation of blood pressure and blood flow. Interestingly, direct hyperthermia-induced brain dysfunction may lead to collapse that can be “lifesaving,” if stopping exercise allows the body to cool or the collapse triggers medical evaluation that leads to cooling therapy.

Exercise stimulates increased blood flow to working muscle. During a maximal effort, for example, approximately 80–85% of maximal cardiac output is distributed to active muscle tissue (139). As core temperature increases during exercise, the thermoregulatory response increases peripheral vasodilatation and blood flow to the cutaneous vascular beds to augment body cooling. The brain also regulates blood pressure during exercise by decreasing blood flow to splanchnic organs. This decreased intestinal blood flow limits vascular heat exchange in the gut and promotes bowel tissue hyperthermia and ischemia. Gut cell membrane breakdown allows lipopolysaccharide fragments from intestinal gram-negative bacteria to leak into the systemic circulation, increasing the risk of endotoxic shock. Dehydration can accentuate these effects on the GI tract and speed the process.

Rhabdomyolysis, the breakdown of muscle fibers, occurs in EHS as muscle tissue exceeds the critical temperature threshold of cell membranes (i.e., about  $40^{\circ}\text{C}$ ). Although eccentric and concentric muscle overuse is a common cause of rhabdomyolysis, muscle membrane permeability increases due to hyperthermia and occurs earlier in exercise when the muscle tissues are hyperthermic (71,74). As heat decomposes cell membranes, myoglobin is released and may cause renal tubular toxicity and obstruction if renal blood flow is inadequate. Intracellular potassium is also released into the extracellular space, increasing serum levels and potentially inducing cardiac arrhythmias. Heating renal tissue above its critical threshold can directly suppress renal function and induce acute renal failure that is worsened by sustained hypotension, crystallization of myoglobin, disseminated intravascular coagulation, and the metabolic acidosis associated with exercise (31,70,153).

**Incidence.** The incidence of EHS varies from event to event and increases with rising ambient temperature and relative humidity. Limited data exist regarding the incidence of EHS during athletic activities. While fatal outcomes are often reported in the press, there is limited reporting of non fatal EHS unless it involves high profile athletes. In most cases, fatal EHS is a rare event that strikes “at random” in sports like American football, especially during the initial four days of preseason conditioning, where the incidence of fatal EHS was about 1 in 350,000 participants from 1995 through 2002 (131). Fatal EHS in American football players often occurs when air temperature is  $26\text{--}30^{\circ}\text{C}$  ( $78\text{--}86^{\circ}\text{F}$ ) and relative humidity is 50–80% (87). EHS is observed more often during road racing and other activities that involve continuous, high-intensity exercise. The Twin Cities Marathon, which is run in cool conditions, averages  $<1$  EHS per 10,000 finishers (136); this incidence rises as the WBGT rises. In contrast, one popular 11.5-km road race, staged in hot and humid summer conditions (WBGT  $21\text{--}27^{\circ}\text{C}$ ), averages 10–20 EHS cases per 10,000 entrants (18,34). The same race course, run in cool conditions, had no cases of EHS (A Crago, M.D., personal communication). Such a high incidence burdens the medical care system and suggests that the summer event is not scheduled at the safest time for the runners.

**Recognition.** Immediate recognition of EHS cases is paramount to survival (68). The appearance of signs and symptoms depends on the degree and duration of hyperthermia (14,48,71,81,150). The symptoms and signs are often nonspecific and include disorientation, confusion, dizziness, irrational or unusual behavior, inappropriate comments, irritability, headache, inability to walk, loss of balance and muscle function resulting in collapse, profound fatigue, hyperventilation, vomiting, diarrhea, delirium, seizures, or coma. Thus, any change of personality or performance should trigger an assessment for EHS, especially in hot-humid conditions. In collision sports like American football, EHS has been initially

mistaken for concussion; among nonathletes, EHS also has been initially misdiagnosed as psychosis.

A body core temperature estimate is vital to establishing an EHS diagnosis, and rectal temperature should be measured in any athlete who collapses or exhibits signs or symptoms consistent with EHS. Ear (aural canal or tympanic membrane), oral, skin over the temporal artery, and axillary temperature measurements should not be used to diagnose EHS because they are spuriously lowered by the temperature of air, skin, and liquids that contact the skin (18,134,135). Oral temperature measurements also are affected by hyperventilation, swallowing, ingestion of cold liquids, and face fanning (33,151). At the time of collapse, systolic blood pressure <100 mm Hg, tachycardia, hyperventilation, and a shocklike appearance (i.e., sweaty, cool skin) are common.

**Evidence statement.** Ear (i.e., aural), oral, skin, temporal, and axillary temperature measurements should not be used to diagnose or distinguish EHS from exertional heat exhaustion. *Evidence category B.* Early symptoms of EHS include clumsiness, stumbling, headache, nausea, dizziness, apathy, confusion, and impairment of consciousness (71,85,149,161). *Evidence category B.*

**Treatment.** EHS is a life-threatening medical emergency that requires immediate whole body cooling for a satisfactory outcome (14,44,48,72,82,85,120,132,149). Cooling should be initiated and, if there are no other life-threatening complications, completed on-site prior to evacuation to the hospital emergency department. Athletes who rapidly become lucid during cooling usually have the best prognosis.

The most rapid whole body cooling rates (i.e., range 0.15–0.24°C·min<sup>-1</sup>) have been observed with cold water and ice water immersion therapy (13,43,47,63,78,83,111, 125,163), and both have the lowest morbidity and mortality rates (47). An aggressive combination of rapidly rotating ice water-soaked towels to the head, trunk and extremities and ice packs to the neck, axillae and groin, (e.g., as currently used at the Twin Cities, Chicago, and Marine Corps marathons) provides a reasonable rate of cooling (i.e., range 0.12–0.16°C·min<sup>-1</sup>). Ice packs to the neck, axilla, and groin will decrease body temperature in the range of 0.04–0.08°C·min<sup>-1</sup> (13). Warm air mist and fanning techniques provide slower whole body cooling rates and are most effective only when the relative humidity is low because this method depends heavily on evaporation for cooling efficacy. Although some patients exhibit a misleading “lucid interval” that often delays the diagnosis, observation and cooling therapy should continue until rectal temperature and mental acuity indicate that treatment is successful. Road race competitors, with rectal temperatures of >42°C and profound CNS dysfunction, who are identified and treated immediately in ice water baths, often leave the medical tent without hospitalization or discernable sequelae (18,34,134).

**Evidence statement.** Cold water immersion provides the fastest whole body cooling rate and the lowest

TABLE 1. Suggested equipment and supplies for treatment of heat related illness.

Stretchers
Cots
Wheelchairs
Bath towels
High temperature rectal thermometers (>43°C, >110°F)
Disposable latex-free gloves
Stethoscopes
Blood pressure cuffs
Intravenous (IV) tubing and cannulation needles
D5%NS and NS IV fluids in 1-L bags
3% saline IV fluid in 250-mL bags
Sharps and biohazard disposal containers
Alcohol wipes, tape, and gauze pads
Tables for medical supplies
Water supply for tubs or ice water buckets
Tub for immersion therapy
Fans for cooling
Oxygen tanks with regulators and masks
Ice, crushed or cubed
Plastic bags
Oral rehydration fluids
Cups for oral fluids
Glucose blood monitoring kits
Sodium analyzer and chemistry chips
Diazepam IV 5 mg or midazolam IV 1 mg vials
Defibrillator (automatic or manual)

<sup>a</sup> Revised from references (2) and (117).

morbidity and mortality for EHS. *Evidence category A.* When water immersion is unavailable, ice water towels/sheets combined with ice packs on the head, trunk, and extremities provide effective but slower whole body cooling. *Evidence category C.*

A medical record should be completed for each athlete who receives treatment (2,132). This provides a record of care and information that can be used to improve the medical plan for future EHS incidents. Table 1 lists the equipment and supplies needed to evaluate and treat exertional heat illnesses that may occur during an athletic event.

EHS casualties often present with cardiovascular collapse and shock. Immediate cooling can reverse these conditions but, if prolonged core temperature elevation and multiple organ failure exist, the victim will require extensive intervention beyond body cooling and fluid replacement. Clinical, hematological, serum chemistry, and diagnostic imaging assessments should be initiated during cooling when possible, but tests that delay body cooling should not be employed unless they are critical to survival (39). Clinical markers of disseminated intravascular coagulation, prolonged elevation of liver and muscle enzymes in the serum, multiple organ failures, and prolonged coma are associated with a grave prognosis.

Preserving intravascular volume with normal saline (NS) infusion improves renal blood flow to protect the kidney from rhabdomyolysis and improves tissue perfusion in all organs for heat exchange, oxygenation, and removal of waste products. Dantrolene, a direct muscle relaxant that alters muscle contractility and calcium channel flow in membranes, purportedly is effective for treating rhabdomyolysis and athletes who have a genetic predisposition to malignant hyperthermia (104), but additional investigations are needed to clarify its efficacy in

EHS. The sheer volume of dantrolene needed to reverse malignant hyperthermia precludes its routine use in the field, but empiric use may be considered in EHS athletes who do not respond to aggressive cooling techniques. Seizures triggered by heat-induced brain dysfunction can be controlled with intravenous benzodiazepines until the brain is cooled and the electrochemical instability is reversed. Treatment of multiple organ system failure associated with prolonged EHS is beyond the scope of this position paper; accepted protocols can be found in most medical texts and handbooks.

**Return to training or competition.** There are no evidence-based recommendations regarding the return of athletes to training after an episode of EHS. For the majority of patients who receive prompt cooling therapy, the prognosis for full recovery and rapid return to activity is good (47,48,123,147). Nine out of 10 prior heatstroke patients tested about two months after an EHS episode demonstrated normal thermoregulation, exercise-heat tolerance, and heat acclimatization with normal sweat gland function, whole body sodium and potassium balance, and blood constituents (14). One of these patients was found to be heat intolerant during laboratory testing at 2 and 7 months after EHS, but was heat tolerant at 1 yr. Physiological and psychological recovery from EHS may require longer than a year, especially in those who experience severe hepatic injury (28,140).

Five recommendations have been proposed for the return to training and competition (37).

1. Refrain from exercise for at least 7 d following release from medical care.
2. Follow up in about 1 wk for physical exam and repeat lab testing or diagnostic imaging of affected organs that may be indicated, based on the physician's evaluation.
3. When cleared for activity, begin exercise in a cool environment and gradually increase the duration, intensity, and heat exposure for 2 wk to acclimatize and demonstrate heat tolerance.
4. If return to activity is difficult, consider a laboratory exercise-heat tolerance test about one month post-incident (14,98,103,138).
5. Clear the athlete for full competition if heat tolerance exists after 2–4 wk of training.

**Evidence statement.** EHS casualties may return to practice and competition when they have reestablished heat tolerance. *Evidence category B.*

### Exertional Heat Exhaustion

Exhaustion defined as the inability to continue to exercise, occurs with heavy exertion in all temperatures and may or may not be associated with physical collapse. From a clinical perspective it is difficult to distinguish athletes who collapse with exhaustion in cool conditions

from those in hot conditions. Exertional heat exhaustion was first described between 1938 and 1944 in medical reports (6,30,169,170) involving laborers and military personnel in the deserts of North Africa (4) and Iraq (89). These reports differentiated heat syncope (i.e., orthostatic hypotension) from heat exhaustion involving significant fluid-electrolyte losses and cardiovascular insufficiency (67,93,170,167). Heat exhaustion is also postulated to be the result of central failure that protects the body against overexertion in stressful situations (99). This paradigm suggests that heat exhaustion is a brain-mediated “safety brake” against excess activity in any environment (114,115,158).

**Etiology.** Heat exhaustion related to dehydration is more common in hot conditions. Rectal temperature can be elevated in heat exhaustion, because circulatory insufficiency predisposes to elevated core body temperatures (16). Laboratory and field studies have shown that exercise in 34–39°C (93–102°F) at 40–50%  $\dot{V}O_{2max}$  does not induce heat exhaustion unless dehydration is present, and that identical exercise performed in a cool environment does not induce heat exhaustion (35,143).

Several lines of evidence suggest that heat exhaustion results from the central fatigue that induces widespread peripheral vascular dilation and associated collapse (11). A Saudi Arabian research group (146) measured echocardiography images of heat exhaustion patients who had participated in consecutive days of desert walking during a religious pilgrimage. These images showed that heat exhaustion involved tachycardia and high cardiac output with peripheral vasodilatation, characteristic of high output heart failure. The vasodilatation lowered peripheral vascular resistance resulting in hypotension and cardiovascular insufficiency. The blood volume pooled in the skin and extremities reduces intravascular heat transport from the core to the body surface and, in turn, heat loss from the skin surface. If the air humidity is high, evaporative cooling is impaired because the air is nearly saturated with moisture, signaling the body to increase cutaneous blood flow to support nonevaporative radiation and convection heat loss. This likely explains why both EHS and heat exhaustion occur more frequently on humid days.

**Predisposing factors.** There are several factors that predispose athletes to heat exhaustion, including the variables that affect exhaustion during exercise. Three studies of underground miners identified that the following factors were associated with an increasing number of heat exhaustion cases: a body mass index  $> 27 \text{ kg}\cdot\text{m}^{-2}$ ; work during the hottest months of the year; elevated urine specific gravity, hematocrit, hemoglobin, or serum osmolality suggesting inadequate fluid intake; an air temperature  $> 33^\circ\text{C}$  and an air velocity  $< 2.0 \text{ m}\cdot\text{s}^{-1}$  (50–52).

**Evidence statement.** Dehydration and high body mass index increase the risk of exertional heat exhaustion. *Evidence category B.* Ten to 14 days of exercise training in the heat will improve heat acclimatization and reduce the risk of exertional heat exhaustion. *Evidence category C.*

**Incidence.** Heat exhaustion is the most common heat-related disorder observed in active populations (5,75, 79,85,97), but the incidence has not been systematically tracked with respect to sport participation. The incidence among religious pilgrims, who walked in the desert at 35–50°C (95–122°F) and had variable fitness and age, was 4 per 10,000 individuals per day (5). Reserve soldiers participating in summer maneuvers at 49–54°C (120–130°F) were affected at a rate of 13 per 10,000 individuals per day (97). Presumably fit competitors running in a 14-km road race with mild air temperatures of 11–20°C (52–68°F) were affected at the rate of 14 per 10,000 individuals per day (127), demonstrating the effects of increased intensity in less stressful heat. During a 6-day youth soccer tournament with early morning WBGT > 28°C (82°F), 34 players out of 4000 (85 per 10,000 incidence) were treated for heat exhaustion with a large increase in cases on the second day of the tournament, demonstrating the effects of cumulative exposure (54). These groups demonstrate the interactions of exercise duration, exercise intensity, and environment on the incidence of exertional heat exhaustion.

**Recognition.** The signs and symptoms of heat exhaustion are neither specific nor sensitive. During the acute stage of heat exhaustion the blood pressure is low, the pulse and respiratory rates are elevated, and the patient appears sweaty, pale, and ashen. Other signs and symptoms include headache, weakness, dizziness, “heat sensations” on the head or neck, chills, “goose flesh”, nausea, vomiting, diarrhea, irritability, and decreased muscle coordination (71,72,76). Muscle cramps may or may not accompany heat exhaustion (70). In the field, rectal temperature measurement may discriminate between severe heat exhaustion (<40°C, 104°F) and EHS (>40°C) (36). If rectal temperature cannot be measured promptly, empiric heatstroke cooling therapy should be considered, especially if there are CNS symptoms. One study systematically examined the role of exercise in heat exhaustion by observing 14 healthy males (15) who ran 8.3–9.8 km·d<sup>-1</sup> on a treadmill at 63–72%  $\dot{V}O_{2max}$  for eight consecutive days in a 41°C (106°F), 39% RH environment and all the common signs and symptoms of heat exhaustion occurred in this study group (see section above titled, “Recognition”).

**Treatment.** An athlete with the clinical picture of exertional heat exhaustion should be moved to a shaded or air conditioned area, have excess clothing removed, placed in the supine position with legs elevated, and have the heart rate, blood pressure, respiratory rate, rectal temperature, and central nervous system status monitored closely. The vast majority of athletes will resolve their collapse with leg elevation, oral fluids, and rest. Heat exhaustion does not always involve elevated core temperature, but cooling therapy will often improve the medical status. An athlete with suspected heat exhaustion who does not improve with these simple measures should be transported to an emergency facility.

Oral fluids are preferred for rehydration in athletes who are conscious, able to swallow well, and not losing fluids via vomiting or diarrhea. As long as the blood pressure, pulse, and rectal temperature are normal and no ongoing fluid losses exist, intravenous fluids should not be required. Intravenous fluid administration facilitates rapid recovery from heat exhaustion (50–52,72) in those who are unable to ingest oral fluids or have more severe dehydration. The decision to utilize intravenous fluids in dehydrated casualties hinges on the patient’s orthostatic pulse, blood pressure change, other clinical signs of dehydration, and ability to ingest oral fluids. Progressive clouding of consciousness should trigger a detailed evaluation for hyperthermia, hypothermia, hyponatremia, hypoglycemia, and other medical problems (112,113). Muscle twitching or cramping that is not easily relieved by stretching may be associated with symptomatic hyponatremia. If dehydration is not clinically obvious in the collapsed athlete with suspected heat exhaustion, consider dilutional hyponatremia as a potential cause of the collapse before administering intravenous fluids (102).

The most commonly recommended IV fluids for rehydrating athletes are NS or 5% dextrose in NS. For empirical field treatment, the primary goal is intravascular volume expansion with saline, to protect organ function and improve blood pressure in athletes with signs of shock. The 5% dextrose solution provides glucose for cell energy. Current protocols suggest starting with NS unless the blood glucose is low. Intravenous fluids (1–4 L) have been used to speed recovery in miners (50–52) and are also used during the half time of soccer matches and American football games, although this practice is not evidence based nor recommended.

The vast majority of athletes with heat exhaustion recover on site and, when clinically stable, may be discharged in the company of a friend or relative with instructions for continued rest and rehydration. A simple check of urine volume and color (i.e., pale yellow or straw color) for the next 48 h will help gauge the recovery process. Prognosis is best when mental acuity was not altered and the athlete becomes alert quickly, following rest and fluids. An athlete with severe heat exhaustion should be instructed to follow-up with a physician (29,36,132).

**Return to training or competition.** An immediate return to exercise or labor following heat exhaustion is not prudent or advised. Athletes with milder forms of heat exhaustion can often return to training or work within 24–48 h, with instructions for gradually increasing the intensity and volume of activity. Neither rest nor body cooling allows heat exhaustion cases to recover to full exercise capacity on the same day (3). In a series of 106 cases of heat exhaustion in underground miners, 4 were sent to the hospital for treatment and 102 were treated on site and released to home. Of 77 miners who returned to work the next day, 30 had persistent mild symptoms of headache and fatigue, and were not allowed to return to normal work duties that day.

Of the asymptomatic miners, 46 of 47 returned to normal duties while 22 of the 30 symptomatic miners were restricted to an air conditioned environment. All of these workers were back to full duties by the third day and none required further medical treatment (52). Serious complications are very rare. Athletes who are rehydrated during games with intravenous fluids and allowed to return to play often are profuse sweaters suffering from dehydration, rather than true heat exhaustion casualties.

### Exercise-Associated Muscle Cramps (Exertional Heat Cramps)

**Etiology.** Exercise associated muscle cramps (EAMC), also called heat cramps, are painful spasms of skeletal muscles that are commonly observed following prolonged, strenuous exercise, often in the heat (95). EAMC is especially prevalent in tennis and American football players (26). EAMC is common in long distance races, where intensity or duration often exceeds that experienced during daily training.

Cramps that occur in the heat are thought by some to differ from EAMC (71) because the cramping is accentuated by large sodium and water losses and that cramps in the heat may present with different signs and symptoms (24,25,71,88). EAMC in the heat often appear unheralded and occur in the legs, arms or abdomen (25,71,92,94,95, 166), although runners, skaters, and skiers who exercise to fatigue in moderate to cool temperatures present in a similar clinical manner. Tennis players who experience recurrent heat cramps are reportedly able to feel a cramp coming on and can abort the cramps with rest and fluids (24). Few investigations have measured fluid-electrolyte balance in EAMC patients, but some have reported whole body sodium deficiencies (24,25,26,88,92,166). Some individuals have a peculiar susceptibility to EAMC that may be related to genetic or metabolic abnormalities in skeletal muscle or lipid metabolism (159).

**Predisposing factors.** Three factors are usually present in EAMC: exercise-induced muscle fatigue, body water loss, and large sweat  $\text{Na}^+$  loss (24,26,85). EAMC seems to be more frequent in long-duration, high-intensity events; indeed, the competitive schedule of certain athletic events may predispose to EAMC. In multiday tennis tournaments, competitors often play more than one match a day, with only an hour between matches. This format induces muscle fatigue, impedes both fluid and electrolyte replacement between matches, and often results in debilitating EAMC (24,25). A similar scenario occurs during the two-a-day practices or competitions and/or other multiday tournaments, both of which are associated with large sweat losses.

**Pathophysiology.** Sweat  $\text{Na}^+$  losses that are replaced with hypotonic fluid have been proposed as the primary cause of EAMC (24,25,32,53,71,92,95,96,166). Sugar cane cutters who experienced EAMC were found to have low

urinary  $\text{Na}^+$  levels (versus healthy laborers) and the authors concluded a whole body  $\text{Na}^+$  deficit existed (92,95). A young tennis player with a history of recurring EAMC successfully treated this disorder by increasing his dietary salt intake (24). In anecdotal reports, steel mill workers prevent EAMC when they increase their consumption of table salt (88,166). Significant quantities of intracellular calcium, magnesium, and potassium ( $\text{K}^+$ ) are not lost during activity, so painful cramps in hot environments are likely not related to changes in those levels (25).

The resting electrical potentials of nerve and muscle tissues are affected by the concentrations of  $\text{Na}^+$ ,  $\text{Cl}^-$  and  $\text{K}^+$  on both sides of the cell membrane. Intracellular dilution or water expansion is believed to play a role in the development of EAMC (88,95). EAMC apparently are less likely to occur when interstitial or extracellular edema is observed (88).

**Incidence.** The incidence of EAMC has not been reported in any large epidemiologic study of athletes. In a 12-yr summary of marathon medical encounters, there were 1.2 cases of EAMC per 1000 race entrants and cramping accounted for 6.1% of medical encounters (136).

**Recognition.** In EAMC, the affected muscle or muscle group is contracted tightly causing pain that is sometimes excruciating. The affected muscles often appear to be randomly involved, and as one bundle of muscle fibers relax, an adjacent bundle contracts, giving the impression that the spasms wander (88). Twitches first may appear in the quadriceps and subsequently in another muscle group (25). Most EAMC spasms last 1–3 min, but the total series may span 6–8 h (95). Intestinal cramps (i.e., due to gaseous bloating or diarrhea) and gastrointestinal infections have been mistaken for abdominal EAMC (71,95).

EAMC can be confused with tetany. However, the characteristic flexion at metacarpophalangeal joints and the extension at interphalangeal joints of the fingers, give the hand its typical tetany appearance. Tetany rarely occurs concurrent with heat cramps, but is commonly observed in hyperventilation syndrome, hypokalemia associated with diuretic use, and in wrestlers who lose weight via dehydration (95).

**Treatment.** EAMC responds well to rest, prolonged stretch with the muscle groups at full length, and oral  $\text{NaCl}$  ingestion in fluids or foods (i.e., 1/8–1/4 teaspoon of table salt added to 300–500 mL of fluids or sports drink, 1–2 salt tablets with 300–500 mL of fluid, bullion broth, or salty snacks). Intravenous NS fluids provide rapid relief from severe EAMC (88,95) in some cases. Calcium salts, sodium bicarbonate, quinine, and dextrose have not produced consistent benefits when treating EAMC (25,95). In refractory muscle cramping, intravenous benzodiazepines effectively relieve muscle cramps through central mechanisms. The use of these medications requires close monitoring and excludes athletes from return to activity. Cramping also occurs in dilutional hyponatremia, so protracted cramping without clinical signs of dehydration

should trigger the measurement of serum Na<sup>+</sup> before administering IV NS to treat the spasms.

**Return to training or competition.** Many athletes with EAMC are able to return to play during the same game with rest and fluid replacement, while some require at least a day to recover following treatment. If the muscle cramping is associated with heat exhaustion or symptomatic hyponatremia (71), the recommendations for the more severe problem should guide the return to play.

**Prevention.** EAMC that occur in hot conditions seem to be prevented by maintaining fluid and salt balance. Athletes with high sweat Na<sup>+</sup> levels and sweat rates, or who have a history of EAMC, may need to consume supplemental Na<sup>+</sup> during prolonged activities to maintain salt balance (25,71,137) and may need to increase daily dietary salt to 5–10 g·d<sup>-1</sup> when sweat losses are large (95,166). This is especially important during the heat acclimatization phase of training. Calculating sweat Na<sup>+</sup> losses and replacing that Na<sup>+</sup> during and after activity allowed two athletes with previously debilitating EAMC to compete successfully in hot conditions (24). There are anecdotal reports of EAMC resolution in American football and in soccer players who increase their oral salt intake before, during, and after activity.

## ATHLETE SAFETY AND REDUCTION OF HEAT RELATED ILLNESS

Events should be scheduled to avoid extremely hot and humid months, based on the historical local weather data. During summer months, all events, games, and practices should be scheduled during the cooler hours of the day

(e.g., early morning). Unseasonably hot days in spring and fall will increase the risk of exertional heat illnesses because competitors are often not sufficiently acclimatized.

Heat acclimatization is the best known protection against both EHS and heat exhaustion. Acclimatization requires gradually increasing the duration and intensity of exercise during the initial 10–14 d of heat exposure, although maximal protection may take up to 12 wk (17). In a study of mortality, the minimum temperatures at which fatal heat stroke occurred decreased at higher latitudes (i.e., northern Europe), and the minimum temperature for fatal cases increased as the summer months progressed at the same latitude (80). Thus, natural heat acclimatization that occurs from living in a given geographic area and the recommended exercise limits and modifications, must consider regional climatic differences. Fitness also confers some protection such that prolonged, near-maximal exertion should be avoided before acquired physical fitness and heat acclimatization are sufficient to support high-intensity, long-duration exercise training or competition (59,122,124,152). Event-specific physical training in the heat reduces the incidence of heat exhaustion (36) by enhancing cardiovascular function and fluid-electrolyte homeostasis.

All athletes should be monitored for signs and symptoms of heat strain, especially during the acclimatization period and when environmental conditions become more stressful, because early recognition decreases both the severity of the episode and the time lost from activity. Athletes, who are adequately rested, nourished, hydrated, and acclimatized to heat are at less risk for heat exhaustion (67). If an athlete experiences recurrent episodes of heat exhaustion, a careful review of fluid intake, diet, whole-body sodium balance,

TABLE 2. WBGT levels for modification or cancellation of workouts or athletic competition for healthy adults.<sup>a,f</sup>

WBGT <sup>b</sup>		Training and Noncontinuous Activity		
°F	°C	Continuous Activity and Competition	Nonacclimatized, Unfit, High-Risk Individuals <sup>c</sup>	Acclimatized, Fit, Low-Risk Individuals <sup>c,d</sup>
≤50.0	≤10.0	Generally safe; EHS can occur associated with individual factors	Normal activity	Normal activity
50.1–65.0	10.1–18.3	Generally safe; EHS can occur	Normal activity	Normal activity
65.1–72.0	18.4–22.2	Risk of EHS and other heat illness begins to rise; high-risk individuals should be monitored or not compete	Increase the rest:work ratio. Monitor fluid intake.	Normal activity
72.1–78.0	22.3–25.6	Risk for all competitors is increased	Increase the rest:work ratio and decrease total duration of activity.	Normal activity. Monitor fluid intake.
78.1–82.0	25.7–27.8	Risk for unfit, nonacclimatized individuals is high	Increase the rest:work ratio; decrease intensity and total duration of activity.	Normal activity. Monitor fluid intake.
82.1–86.0	27.9–30.0	Cancel level for EHS risk	Increase the rest:work ratio to 1:1, decrease intensity and total duration of activity. Limit intense exercise. Watch at-risk individuals carefully	Plan intense or prolonged exercise with discretion <sup>f</sup> ; watch at-risk individuals carefully
86.1–90.0	30.1–32.2		Cancel or stop practice and competition.	Limit intense exercise <sup>f</sup> and total daily exposure to heat and humidity; watch for early signs and symptoms
≥90.1	>32.3		Cancel exercise.	Cancel exercise uncompensable heat stress <sup>g</sup> exists for all athletes <sup>f</sup>

<sup>a</sup> revised from reference (38).

<sup>b</sup> wet bulb globe temperature.

<sup>c</sup> while wearing shorts, T-shirt, socks and sneakers.

<sup>d</sup> acclimatized to training in the heat at least 3 wk.

<sup>e</sup> internal heat production exceeds heat loss and core body temperature rises continuously, without a plateau.

<sup>f</sup> Differences of local climate and individual heat acclimatization status may allow activity at higher levels than outlined in the table, but athletes and coaches should consult with sports medicine staff and should be cautious when exceeding these limits.

TABLE 3. Modifying practice sessions for exercising children.

WBGT		Restrains on Activities
°F	°C	
<75.0	<24.0	All activities allowed, but be alert for prodromes of heat-related illness in prolonged events
75.0–78.6	24.0–25.9	Longer rest periods in the shade; enforce drinking every 15 min
79.0–84.0	26.0–29.0	Stop activity of unacclimatized persons and high-risk persons; limit activities of all others (disallow long-distance races, cut the duration of other activities)
>85.0	>29.0	Cancel all athletic activities

Notes:

1. Source: reference (7).
2. These guidelines do not account for clothing. Although the effects of the uniform clothing and protective equipment (i.e., American football) on sweating and body temperature in younger athletes are unknown, uniforms should be considered when determining playing/practice limitations based on the WBGT.
3. Eight to 10 practices are recommended for heat acclimatization (30–45 min each; one per day or one every other day).
4. Differences of local climate and individual heat acclimatization status may allow activity at higher levels than outlined in the table, but athletes and coaches should consult with sports medicine staff and should be cautious when exceeding these limits.

recovery interval, and heat acclimatization should be undertaken and corrected (36). Athletes should have a monitored fluid replacement plan (8,9) to stay within 2% of the baseline body weight (i.e., initial weight in multiday events or practices) (30).

**Activity modification in high-risk situations.** The events posing the greatest risk of EHS, heat exhaustion and EAMC involve high-intensity exercise in a hot-humid environment. Athletes may not have adequate experience to withdraw voluntarily, and often assume that the act of conducting a competition or practice implies safe conditions for the activity. The internal motivation of athletes to succeed under any circumstances plays a role in EHS risk and must be recognized by coaches and administrators when making the decision to conduct an event in high-risk conditions.

One athlete experiencing heat-related symptoms (i.e., the “weak link”) often indicates that other exertional heat casualties will soon follow (75,97). Medical providers, event directors, coaches and athletes should be prepared to postpone, reschedule, modify, or cancel activities when environmental conditions pose undue risk, based on predetermined safety guidelines established for that event. The pressures exerted by parents, peers, coaches, administrators, and competition may encourage ill, fatigued, or dehydrated athletes to participate when the environmental conditions are unsafe.

Independent of an athlete’s heat acclimatization or fitness, practice sessions and competitions should be modified with unlimited fluid access, longer and/or more rest breaks to facilitate heat dissipation, shorter playing times to decrease heat production, and/or delays when the moderate or higher environment risk categories exist (Table 2). The following factors should be considered

when modifying training or events: environmental conditions, heat acclimatization status of participants, fitness and age of participants, intensity and duration of exercise, time of day, clothing or uniform requirements, sleep deprivation, nutrition, availability of fluids, frequency of fluid intake, and playing surface heat reflection and radiation (i.e., grass, asphalt) (157). During high heat stress conditions, remove equipment and extra clothing to reduce heat storage. Allow a minimum of 3, and preferably 6, hours of recovery and rehydration time between practice sessions and games.

**Evidence Statement.** Practice and competition should be modified on the basis of air temperature, relative humidity, sun exposure, heat acclimatization status, age, and equipment requirements by decreasing the duration and intensity of exercise and by removing clothing. *Evidence category C.*

**Monitoring the environment.** Event organizers should monitor the weather conditions before and during practice and competition. Ideally, heat stress should be measured at the event site for the most accurate meteorological data. Factors that affect heat injury risk include ambient temperature, relative humidity, wind speed, and solar radiant heat; as a minimum standard, the

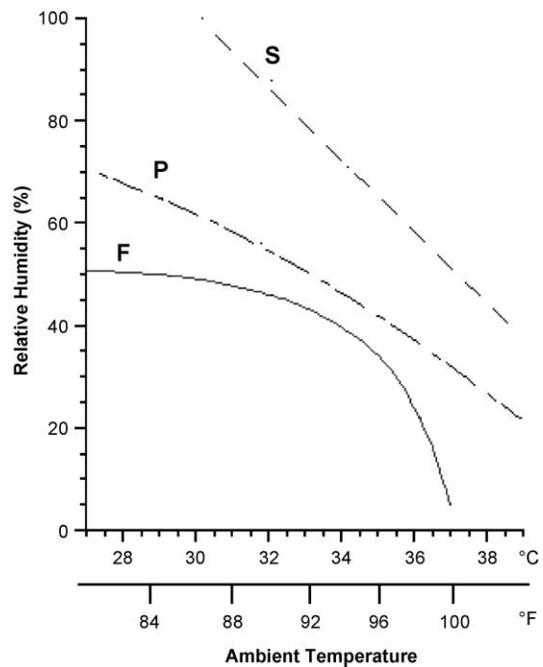


FIGURE 2—Environmental conditions that are critical for American football players wearing different clothing ensembles [S refers to a clothing ensemble of shorts, socks and sneakers; P (practice uniform) refers to helmet, undershirt, shoulder pads, jersey, shorts, socks and sneakers; F (full game uniform) refers to helmet, undershirt, shoulder pads, jersey, shorts, socks, sneakers, game pants, thigh pads and knee pads]. The zone above and to the right of each clothing ensemble (F, P, S) represents uncompensable heat stress with rising core temperature during exercise [redrawn with permission from reference (87); illustrates exercise at 35%  $\dot{V}O_{2max}$ ; uncompensable heat stress is defined in Table 2, footnote f]. The zone below and to the left of lines F, P and S represents compensable heat stress with heat balance possible.

dry bulb temperature and relative humidity should be considered in the decision to modify activity. The WBGT is used in athletic, military, and industrial settings (49,76,162,174) to gauge heat risk because it incorporates measurements of radiant heat ( $T_{bg}$ ) and air water content ( $T_{wb}$ ). The WBGT is calculated using the following formula (174):

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

where  $T_{wb}$  is the wet bulb temperature,  $T_{bg}$  is the black globe temperature, and  $T_{db}$  is the shaded dry bulb temperature (49).  $T_{wb}$  is measured with a dry bulb thermometer that is covered with a water-saturated cloth wick.  $T_{bg}$  is measured by inserting a dry bulb thermometer into a standard black metal globe. Both  $T_{wb}$  and  $T_{bg}$  are measured in direct sunlight. In this formula,  $T_{wb}$  accounts for 70% of the WBGT.

A portable monitor that measures the WBGT is useful to determine heat stress on site (49,77,162,174), but cost limits this use in many situations. Devices that measure temperature, relative humidity and wet bulb temperature can be purchased for less than \$75. These measurements can be mathematically converted to WBGT using shareware provided by a nonprofit source (177). When the WBGT is not available, on-site ambient temperature and relative humidity data can be applied to standardized algorithms or charts to estimate heat risk.

The risk of EHS and exertional heat exhaustion (while wearing shorts, socks, shoes, and a T-shirt) due to environmental stress can be stratified into three activity categories, as depicted in Table 2; these involve either continuous activity and competition, or training and noncontinuous activity. Large posters or signs should be displayed at the athletic venue or along the race course, to describe the risk of heat exhaustion and EHS. If the WBGT index is above 28°C (82°F), consideration should be given to canceling or rescheduling continuous competitive events until less stressful conditions prevail (117). Table 3, regarding children, presents a modified version of a previous publication (7). Although children have been considered “less heat tolerant” in the past, current data collected on boys does not necessarily support this belief (78,148). However, until more research is available, it is prudent to regard children as an “at risk” group. The decision to modify activity is often in the hands of coaches, who must be willing to make safety related changes for practices and games, based on environmental conditions.

**Uniforms.** Before athletes are acclimatized to heat, the effect of uniforms on body heat storage is significant, especially in American football. Helmets, protective pads, gloves and garments trap heat and reduce heat dissipation. Exercise-related metabolic heat production raises core body temperature without a plateau and readily induces an “uncompensable” heat stress situation. Athletes should

TABLE 4. Evidence-based statements, evaluated in terms of the strength of supporting scientific evidence. Criteria (column 2) are defined in the Summary section.

	Level of Evidence	References
Dehydration reduces endurance exercise performance, decreases time to exhaustion, increases heat storage.	A	11,12,16,41,57,141
Exertional heatstroke (EHS) is defined in the field by rectal temperature >40°C at collapse and by central nervous system changes.	B	37,39,56,71,150,156,175
The following conditions increase the risk of EHS or exertional heat exhaustion: obesity, low physical fitness level, lack of heat acclimatization, dehydration, a previous history of EHS, sleep deprivation, sweat gland dysfunction, sunburn, viral illness, diarrhea, or certain medications.	B	14,22,45,55,60,66,69,85,99,149,150,164,173
Physical training and cardiorespiratory fitness reduce the risk of EHS.	C	17,29,59,122,124,152
Cold water immersion provides the fastest whole body cooling rate and the lowest morbidity and mortality for EHS.	A	2,13,14,43,44,47,48,49,63,68,72,82,83,85,111,125,134,149,175
When water immersion is unavailable, ice water towels/sheets combined with ice packs on the head, trunk, and extremities provide effective but slower whole body cooling.	C	
Dehydration and high body mass index increase the risk of exertional heat exhaustion.	B	17,29,59,122,124,152
10–14 days of exercise training in the heat will improve heat acclimatization and reduce the risk of EHS.	B	14,17,85,175
10–14 days of exercise training in the heat will improve heat acclimatization and reduce the risk of exertional heat exhaustion.	B	14,17,85,175
EHS casualties may return to practice and competition when they have reestablished heat tolerance.	B	14,38,55,56,81,103,138
Ear (i.e., aural), oral, skin, temporal, and axillary temperature measurements should not be used to diagnose or distinguish EHS from exertional heat exhaustion.	B	18,36,39,134,135
Early symptoms of EHS include clumsiness, stumbling, headache, nausea, dizziness, apathy, confusion, and impairment of consciousness.	B	71,85,149,161
Practice and competition should be modified on the basis of air temperature, relative humidity, sun exposure, heat acclimatization status, age, and equipment requirements by decreasing the duration and intensity of exercise and by removing clothing.	C	38,49,108,157,174
Athletes should exercise with a partner in high-risk conditions, each being responsible for monitoring the other's well being.	C	49,128,157

remove as much protective equipment as possible to permit heat loss and to reduce the risks of hyperthermia, especially during acclimatization. Figure 2 demonstrates uncompensable heat stress in varying hot environments; it can guide coaches and athletes who select the practice uniform for a range of environmental conditions.

The National Collegiate Athletic Association (NCAA) regulates the introduction and use of protective padding for collegiate football players to aid heat acclimatization (107, 108). The current regulations allow players to wear helmets during the first 2 d of practice, helmets and shoulder pads during days 3 and 4, and the full uniform after the fifth day. These regulations also reduce the number and duration of workouts during the initial 5 d of summer training to one per day and limit “two a day” sessions to an every-other-day format for the remainder of the season. Although NCAA heat acclimatization strategies were designed for collegiate American football players, the model (107) is also recommended as a minimum standard for younger athletes. Specific recommendations should be uniquely designed for other age groups and sports to improve athlete safety.

#### Monitoring athletes across consecutive days.

Athletes practicing or competing during multiple-day and/or multiple-session same day events in hot and humid conditions should be monitored for signs and symptoms of heat illness and the cumulative effects of dehydration (3,12,41,57,141). Day-to-day body weight measurements (40) and urine color should be used to assess progressive dehydration and increased risk for heat illness. Replace fluid deficits before the next practice session (40). Wireless deep body temperature sensors transmit gastrointestinal temperature (145) and can be used to monitor high-risk athletes who have a history of EHS, although this is not a practical strategy for most athletes and will pose a potential risk for an athlete who might require an MRI.

**Education.** The education of athletes, coaches, administrators, medical providers (especially on site personnel and community emergency response teams) can

help with reduction, recognition, and treatment of heat related illness. Counsel athletes about the importance of being well-hydrated, well-fed, well-rested, and acclimatized to heat. Have athletes “buddy up” to monitor each other for signs of subtle changes of performance or behavior.

**Evidence statement.** Athletes should exercise with a partner in high-risk conditions, each being responsible for the other’s well being. *Evidence category C.*

## CONCLUSION

The challenges of hot environments and exercise are complex and difficult to fully comprehend because athletes are variably affected during high-intensity exercise in hot-humid environments. EHS, the most severe form of heat illness, cannot be studied in the laboratory because the risks of severe hyperthermia are ethically unacceptable for human research. Thus, our knowledge depends on the judicious field documentation of athletes who push beyond normal physiological limits. The survival of these athletes depends on prompt recognition and the most effective cooling therapy (i.e., ice water immersion or rapidly rotating ice water towels combined with ice packs) to limit tissue exposure due to destructive hyperthermia. The existing evidence is summarized in Table 4.

This Position Stand replaces, in part, the 1996 Position Stand “Heat and Cold Illnesses during Running,” *Med. Sci. Sport Exerc.* 28(12):i-x, 1996.

This pronouncement was reviewed for the American College of Sports Medicine by the Pronouncements Committee and by Anne L. Friedlander, Ph.D.; James P. Knochel, M.D.; Christopher T. Minson, Ph.D., FACSM; Denise L. Smith, Ph.D., FACSM; and Jeffrey J. Zachwieja, Ph.D., FACSM.

**Financial and Affiliation Disclosure:** Douglas J. Casa serves on the Board of Advisors for Education at the Gatorade Sports Science Institute, PepsiCo Inc. Dr. Casa has received research grants through the Gatorade Sports Science Institute, HQ, Inc., and CamelBak, Inc. Mindy Millard-Stafford serves as a scientific advisor for The Beverage Institute for Health and Wellness, The Coca-Cola Company. Jeffrey Zachwieja is employed as a principal scientist at the Gatorade Sports Science Institute, PepsiCo Inc.

## REFERENCES

1. AARSETH, H. P., I. EIDE, B. SKEIE, and E. THAULOW. Heatstroke in endurance exercise. *Acta Med. Scand.* 220:279–283, 1986.
2. ADNER, M. M., J. J. SCARLET, W. ROBINSON, and B. H. JONES. The Boston Marathon medical care team: ten years of experience. *Physician Sportsmed.* 16:99–106, 1988.
3. ADOLPH, E. F. In: *Physiology of Man in the Desert*, New York: Interscience, pp. 5–43, 326–341, 1947.
4. ADOLPH, E. F. Water metabolism. *Ann. Rev. Physiol.* 9:381–408, 1947.
5. AL-MARZOOGI, A., M. KHOGALI, and A. EL-ERGESUS. Organizational set-up: detection, screening, treatment, and follow-up of heat disorders. In: *Heatstroke and Temperature Regulation*, M. Khogali and J. R. S. Hales. Sydney, Australia: Academic Press, pp. 31–40, 1983.
6. ALLEN, S. D., and R. P. O’BRIEN. Tropical anhidrotic asthenia: preliminary report. *Med. J. Aust.* 2:335–337, 1944.
7. AMERICAN ACADEMY OF PEDIATRICS. Climatic heat stress and the exercising child and adolescent. *Pediatrics* 106(1):158–159, 2000.
8. AMERICAN COLLEGE OF SPORTS MEDICINE. Position Stand: the prevention of thermal injuries during distance running. *Med. Sci. Sports Exerc.* 19:529–533, 1987.
9. AMERICAN COLLEGE OF SPORTS MEDICINE. Position Stand: Heat and cold illnesses during running. *Med. Sci. Sports Exerc.* 28: i-x, 1996.
10. ARMSTRONG, L. E. Classification, nomenclature, and incidence of the exertional heat illnesses. In: *Exertional Heat Illnesses*. L. E. Armstrong. Champaign, IL: Human Kinetics, pp. 17–28, 2003.
11. ARMSTRONG, L. E., and J. M. ANDERSON. Heat exhaustion, exercise-associated collapse, and heat syncope. In: *Exertional Heat Illnesses*, L. E. Armstrong. Champaign, IL: Human Kinetics, pp. 57–90, 2003.
12. ARMSTRONG, L. E., D. L. COSTILL, and W. J. FINK. Influence of

- diuretic-induced dehydration on competitive running performance. *Med. Sci. Sports Exerc.* 17(4):456–461, 1985.
13. ARMSTRONG, L. E., A. E. CRAGO, R. ADAMS, W. O. ROBERTS, and C. M. MARESH. Whole-body cooling of hyperthermic runners: comparison of two field therapies. *Am. J. Emerg. Med.* 14:355–358, 1996.
  14. ARMSTRONG, L. E., J. P. DELUCA, and R. W. HUBBARD. Time course of recovery and heat acclimation ability of prior exertional heatstroke patients. *Med. Sci. Sports Exerc.* 22:36–48, 1990.
  15. ARMSTRONG, L. E., R. W. HUBBARD, W. J. KRAEMER, J. P. DE LUCA, and E. L. CHRISTENSEN. Signs and symptoms of heat exhaustion during strenuous exercise. *Ann. Sports Med.* 3: 182–189, 1988.
  16. ARMSTRONG, L. E., R. W. HUBBARD, P. C. SZLYK, I. V. SILS, and W. J. KRAEMER. Heat intolerance, heat exhaustion monitored: a case report. *Aviat. Space Environ. Med.* 59:262–266, 1988.
  17. ARMSTRONG, L. E., and C. M. MARESH. The induction and decay of heat acclimatization in trained athletes. *Sports Med. (New Zealand)* 12:302–312, 1991.
  18. ARMSTRONG, L. E., C. M. MARESH, A. E. CRAGO, R. ADAMS, and W. O. ROBERTS. Interpretation of aural temperatures during exercise, hypothermia, and cooling therapy. *Med. Exerc. Nutr. Health* 3:9–16, 1994.
  19. ARMSTRONG, L. E., C. M. MARESH, C. V. GABAREE, et al. Thermal and circulatory responses during exercise: effects of hypohydration, dehydration, and water intake. *J. Appl. Physiol.* 82: 2028–2035, 1997.
  20. ASSIA, E., Y. EPSTEIN, and Y. SHAPIRO. Fatal heatstroke after a short march at night: a case report. *Aviat. Space Environ. Med.* 56:441–442, 1985.
  21. BANGSBO, J. *The Physiology of Soccer-With Special Reference to Intense Intermittent Exercise*, Copenhagen: August Krogh Institute, University of Copenhagen, 1993.
  22. BAR-OR, O., H. M. LUNDERGREN, and E. R. BUSKIRK. Heat tolerance of exercising lean and obese women. *J. Appl. Physiol.* 26:403–409, 1969.
  23. BELL, D. G., I. JACOBS, T. M. McLELLAN, M. MIYAZAKI, and C. M. SABISTON. Thermal regulation in the heat during exercise after caffeine and ephedrine ingestion. *Aviat. Space Environ. Med.* 70(6):583–588, 1999.
  24. BERGERON, M. F. Heat cramps during tennis: a case report. *Int. J. Sport Nutr.* 6:62–68, 1996.
  25. BERGERON, M. F. Exertional heat cramps. In: *Exertional Heat Illnesses*, L. E. Armstrong. Champaign, IL: Human Kinetics, pp. 91–102, 2003.
  26. BERGERON, M. F. Heat cramps: fluid and electrolyte challenges during tennis in the heat. *J. Sci. Med. Sport* 6(1): 19–27, 2003.
  27. BERNHEIM, P. J., and J. N. COX. Coup de chaleur et intoxication amphetaminique chez un sportif. *Schweiz Medicine Wochenschrift* 90:322–331, 1960.
  28. BIANCHI, L., H. OHNACKER, K. BECK, and M. ZIMMERLI-NING. Liver damage in heatstroke and its regression. *Hum. Pathol.* 3:237–249, 1972.
  29. BINKLEY, H. M., J. BECKETT, D. J. CASA, D. M. KLEINER, and P. E. PLUMMER. Position statement: exertional heat illnesses. *J. Athl. Train.* 37:329–343, 2002.
  30. BLACK, D. A., R. A. McCANCE, and W. F. YOUNG. A study of dehydration by means of balance experiments. *J. Physiol. (London)* 102:406–414, 1944.
  31. BOSENBERG, A. T., J. G. BROCK-UTNE, M. T. WELLS, G. T. BLAKE, and S. L. GAFFIN. Strenuous exercise causes systemic endotoxemia. *J. Appl. Physiol.* 65:106–108, 1988.
  32. BRENDIA, C. Outwitting muscle cramps—is it possible? *Physician Sports Med.* 17:173–178, 1989.
  33. BRENGELMAN, G. L. Dilemma of body temperature measurement. In: *Man in Stressful Environments*, K. Shiraki and M. K. Yousef. Springfield, IL: C.C. Thomas Publishers, 1987, pp. 5–22.
  34. BRODEUR, V. B., S. R. DENNETT, and S. L. GRIFFIN. Hyperthermia, ice baths, and emergency care at the Falmouth Road Race. *J. Emerg. Nurs.* 15:304–312, 1989.
  35. BROWN, A. H. Dehydration exhaustion. In: *Physiology of Man in the Desert*, New York: Interscience, pp. 208–217, 1947.
  36. CASA, D. J., J. ALMQUIST, S. ANDERSON, et al. Inter-association task force on exertional heat illnesses consensus statement. *NATA News* 6:24–29, 2003.
  37. CASA, D. J., and L. E. ARMSTRONG. Exertional heatstroke: a medical emergency. In: *Exertional Heat Illnesses*, L. E. Armstrong. Champaign, IL: Human Kinetics, pp. 29–56, 2003.
  38. CASA, D. J., and E. R. EICHNER. Exertional heat illness and hydration. In: *Athletic Training and Sports Medicine*, C. Starkey and G. Johnson. Boston, MA: Jones and Bartlett Publishers, pp. 597–615, 2005.
  39. CASA, D. J., and W. O. ROBERTS. Considerations for the medical staff: Preventing, identifying, and treating exertional heat illnesses. In: *Exertional Heat Illnesses*, L. E. Armstrong. Champaign, IL: Human Kinetics, pp. 169–196, 2003.
  40. CHEUVRONT, S. N., R. C. CARTER, S. J. MONTAIN, and M. N. SAWKA. Daily body mass variability and stability in active men undergoing exercise-heat stress. *Int. J. Sports Nutr. Exerc. Metab.* 14:532–540, 2004.
  41. CHEUVRONT, S. N., R. C. CARTER, and M. N. SAWKA. Fluid balance and endurance exercise performance. *Cur. Sports Med. Reports* 2:202–208, 2003.
  42. CHEUVRONT, S. N., and E. M. HAYMES. Thermoregulation and marathon running: biological and environmental influences. *Sports Med. (New Zealand)* 31:743–762, 2001.
  43. CLEMENTS, J. M., D. J. CASA, J. C. KNIGHT, et al. Ice-water immersion and cold-water immersion provide similar cooling rates in runners with exercise-induced hyperthermia. *J. Athl. Train.* 37:146–150, 2002.
  44. CLOWES, G. H. A. JR., and T. F. O'DONNELL. Heatstroke. *N. Engl. J. Med.* 291:564–567, 1974.
  45. COSTILL, D. L., R. COTE, E. MILLER, T. MILLER, and S. WYNDER. Water and electrolyte replacement during days of work in the heat. *Aviat. Space Environ. Med.* 46:795–800, 1970.
  46. COSTILL, D. L., W. F. KAMMER, and A. FISHER. Fluid ingestion during distance running. *Arch. Environ. Health* 21:520–525, 1970.
  47. COSTRINI, A. Emergency treatment of exertional heatstroke and comparison of whole body cooling techniques. *Med. Sci. Sports Exerc.* 22:15–18, 1990.
  48. COSTRINI, A. M., H. A. PITT, A. B. GUSTAFSON, and D. E. UDDIN. Cardiovascular and metabolic manifestations of heatstroke and severe heat exhaustion. *Am. J. Med.* 66:296–302, 1979.
  49. DEPARTMENT OF THE ARMY AND AIR FORCE. In: *Heat Stress Control and Heat Casualty Management*, Washington, DC: Department of the Army, 2003, pp. 1–66. Technical bulletin No. TB MED 507.
  50. DONOGHUE, A. M., and G. P. BATES. The risk of heat exhaustion at a deep underground metalliferous mine in relation to body-mass index and predicted  $VO_{2max}$ . *Occup. Med.* 50:259–263, 2000.
  51. DONOGHUE, A. M., and G. P. BATES. The risk of heat exhaustion at a deep underground metalliferous mine in relation to surface temperatures. *Occup. Med.* 50:334–336, 2000.
  52. DONOGHUE, A. M., M. J. SINCLAIR, and G. P. BATES. Heat exhaustion in a deep underground metalliferous mine. *Occup. Environ. Med.* 57:165–174, 2000.
  53. EATON, J. M. Is this really a muscle cramp? *Postgrad. Med.* 86:227–232, 1989.
  54. ELIAS, S. R., W. O. ROBERTS, and D. C. THORSON. Team sports in hot weather: guidelines for modifying youth soccer. *Physician Sports Med.* 19:67–80, 1991.

55. EPSTEIN, Y. Heat intolerance: predisposing factor or residual injury? *Med. Sci. Sports Exerc.* 22:29–35, 1990.
56. EPSTEIN, Y., E. SOHAR, and Y. SHAPIRO. Exertional heatstroke: a preventable condition. *Isr. J. Med. Sci.* 31:454–462, 1995.
57. FALLOWFIELD, J. L., C. WILLIAMS, J. BOOTH, B. H. CHOO, and S. GROWNS. Effect of water ingestion on endurance capacity during prolonged running. *J. Sports Sci.* 14:497–502, 1996.
58. GALLOWAY, S. D., and R. J. MAUGHAN. Effects of ambient temperature on the capacity to perform prolonged cycle exercise in man. *Med. Sci. Sports Exerc.* 29:1240–1249, 1997.
59. GISOLFI, C. V., and R. J. COHEN. Relationships among training, heat acclimation and heat tolerance in men and women: the controversy revisited. *Med. Sci. Sports* 11:56–59, 1979.
60. GISOLFI, C. V., and J. R. COPPING. Thermal effects of prolonged treadmill exercise in the heat. *Med. Sci. Sports* 6:108–113, 1974.
61. GONZALEZ-ALONSO, J., C. TELLER, S. ANDERSEN, et al. Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *J Appl Physiol* 86(3):1032–1039, 1999.
62. GRABER, C. D., R. B. REINHOLD, and J. G. BREMAN. Fatal heatstroke. *J. A. M. A.* 216:1195–1196, 1971.
63. HADAD, E., M. RAY-ACHA, Y. HELED, Y. EPSTEIN, and D. S. MORAN. Heat stroke: a review of cooling methods. *Sports Med. (New Zealand)* 34:501–511, 2004.
64. HANSON, P. G., and S. W. ZIMMERMAN. Exertional heatstroke in novice runners. *J. A. M. A.* 242:154–158, 1979.
65. HART, L. E., B. P. EGIER, A. G. SHIMIZU, P. J. TANDAN, and J. R. SUTTON. Exertional heatstroke: The runner's nemesis. *Can. Med. Assoc. J.* 122:1144–1149, 1980.
66. HAYMES, E. M., R. J. McCORMICK, and E. R. BUSKIRK. Heat tolerance of exercising lean and obese prepubertal boys. *J. Appl. Physiol.* 39:457–461, 1975.
67. HEADQUARTERS, DEPARTMENTS OF THE ARMY AND AIR FORCE. *Heat stress control and heat casualty management*, Washington, D.C: Headquarters Department of the Army and Air Force, 2003. Publication TB MED 507.
68. HELED, Y., M. RAY-ACHA, Y. SHANI, Y. EPSTEIN, and D. S. MORAN. The “golden hour” for heatstroke treatment. *Mil. Med.* 169:184–186, 2004.
69. HOLTZHAUSEN, L. M., and T. D. NOAKES. Collapsed ultradistance athlete: proposed mechanisms and an approach to management. *Clin. J. Sport Med.* 7:292–301, 1997.
70. HUBBARD, R. W. An introduction: the role of exercise in the etiology of exertional heatstroke. *Med. Sci. Sports Exerc.* 22:2–5, 1990.
71. HUBBARD, R. W., and L. E. ARMSTRONG. The heat illness: biochemical, ultrastructural, and fluid-electrolyte considerations. In: *Human Performance Physiology and Environment Medicine at Terrestrial Extremes*, K. B. Pandolf, M. N. Sawka, and R. R. Gonzalez. Indianapolis: Benchmark Press, pp. 305–359, 1988.
72. HUBBARD, R. W., and L. E. ARMSTRONG. Hyperthermia: new thoughts on an old problem. *Physician Sportsmed.* 17:97–113, 1989.
73. HUBBARD, R. W., W. T. MATTHEW, R. E. L. CRISS, et al. Role of physical effort in the etiology of rat heatstroke injury and mortality. *J. Appl. Physiol.* 45:463–468, 1978.
74. HUBBARD, R. W., C. B. MATTHEW, M. J. DURKOT, and R. P. FRANCESCONI. Novel approaches to the pathophysiology of heatstroke: the energy-depletion model. *Ann. Emerg. Med.* 16:1066–1075, 1987.
75. HUBBARD, R. W., W. MATTHEW, D. WRIGHT. Survey and analysis of the heat casualty prevention experiment for Resphiblex 1–81, Operation Lancer Eagle, 43D, MAU. Natick, MA: U. S. Army Research Institute of Environmental Medicine, Technical Report T 5–82, 1982.
76. HUGHSON, R. L., H. J. GREEN, M. E. HOUSTON, J. A. THOMSON, D. R. MACLEAN, and J. R. SUTTON. Heat injuries in Canadian mass participation runs. *Can. Med. Assoc. J.* 122:1141–1144, 1980.
77. HUGHSON, R. L., L. A. STANDI, and J. M. MACKIE. Monitoring road racing in the heat. *Physician Sportsmed.* 11:94–105, 1983.
78. INBAR, O., N. MORRIS, Y. EPSTEIN, and G. GASS. Comparison of thermoregulatory responses to exercise in dry heat among prepubertal boys, young adults and older males. *Exp. Physiol.* 89:691–700, 2004.
79. KARK, J. A., P. BURR, C. B. WENGER, E. GASTALDO, and J. W. GARDNER. Exertional heat illness in marine corps recruit training. *Aviat. Space Environ. Med.* 67:354–360, 1996.
80. KEATINGE, W. R., G. C. DONALDSON, E. CORDIOLI, et al. Heat related mortality in warm and cold regions of Europe: observational study. *Brit. Med. J.* 321:670–673, 2000.
81. KEREN, G., Y. EPSTEIN, and A. MAGAZANIK. Temporary heat intolerance in a heatstroke patient. *Aviat. Space Environ. Med.* 52:116–117, 1981.
82. KHOGALI, M., and J. S. WEINER. Heatstroke: report on 18 cases. *Lancet* 2:276–278, 1980.
83. KIELBLOCK, A. J. Strategies for the prevention of heat disorders with particular reference to body cooling procedures. In: *Heat Stress*, J. R. S. Hales and D. A. B. Richards. Amsterdam: Elsevier, pp. 489–497, 1987.
84. KIELBLOCK, A. J., N. B. STRYDOM, F. J. BURGER, P. J. PRETORIUS, and M. MANJOO. Cardiovascular origins or heatstroke pathophysiology. An anesthetized rat model. *Aviat. Space Environ. Med.* 53:171–178, 1982.
85. KNOCHEL, J. P. Environmental heat illness: an eclectic review. *Arch. Intern. Med.* 133:841–864, 1974.
86. KNOCHEL, J. P., and G. REED. Disorders of heat regulation. In: *Clinical Disorders, Fluid and Electrolyte Metabolism*, M. H. Kleeman, C. R. Maxwell, and R. G. Narin. New York: McGraw-Hill, pp. 1197–1232, 1987.
87. KULKA, T. J., and W. L. KENNEY. Heat balance limits in football uniforms. *Physician Sportsmed.* 30:29–39, 2002.
88. LADELL, W. S. S. Heat cramps. *Lancet* 2:836–839, 1949.
89. LADELL, W. S. S., J. C. WATERLOW, and M. F. HUDSON. Desert climate: physiological and clinical observations. *Lancet* 2:527–531, 491–497, 1944.
90. LAMBERT, E. V., A. ST CLAIR GIBSON, and T. D. NOAKES. Complex systems model of fatigue: integrative homeostatic control of peripheral physiological systems during exercise in humans. *Br. J. Sports Med.* 39:52–62, 2005.
91. LAMBERT, G. P. Role of gastrointestinal permeability in exertional heatstroke. *ESSR* 32:185–190, 2004.
92. LEITHEAD, C. S., and E. R. GUNN. The aetiology of cane cutter's cramps in British Guiana. In: *Environmental Physiology and Psychology in Arid Conditions*, Leige, Belgium: United Nations Educational Scientific and Cultural Organization, pp. 13–17, 1964.
93. LEITHEAD, C. S., and A. R. LIND. Heat syncope. In: *Heat Stress and Heat Disorders*, Philadelphia: F. A. Davis Co, pp. 136–140, 1964.
94. LEITHEAD, C. S., and A. R. LIND. Water and salt depletion heat exhaustion. In: *Heat Stress and Heat Disorders*, Philadelphia: F. A. Davis Co, pp. 143–169, 1964.
95. LEITHEAD, C. S., and A. R. LIND. Heat cramps. In: *Heat Stress and Heat Disorders*, Philadelphia: F. A. Davis Co, pp. 170–177, 1964.
96. LEVIN, S. Investigating the cause of muscle cramps. *Physician Sportsmed.* 21:111–113, 1993.
97. MAGER, M., R. W. HUBBARD, M. D. KERSTEIN. Survey and analysis of the medical experience for CAX 8–80. Natick, MA: U. S. Army Research Institute of Environmental Medicine, Technical Report, 1980.

98. MARON, M. B., J. A. WAGNER, and S. M. HORVATH. Thermoregulatory responses during competitive marathon running. *J. Appl. Physiol.* 42:909–914, 1977.
99. MARTINEZ, M., L. DAVENPORT, J. SAUSSY, and J. MARTINEZ. Drug-associated heat stroke. *South. Med. J.* 95(8):799–802, 2002.
100. MAUGHAN, R. J., S. J. MERSON, N. P. BROAD, and S. M. SHIRREFFS. Fluid and electrolyte intake and loss in elite soccer players during training. *Int. J. Sport Nutr. Exerc. Metab.* 14:333–346, 2004.
101. MONTAIN, S. J., and E. F. COYLE. Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J. Appl. Physiol.* 73:1340–1350, 1992.
102. MONTAIN, S. J., M. N. SAWKA, and C. B. WENGER. Hyponatremia associated with exercise: risk factors and pathogenesis. *Exerc. Sport Sci. Rev.* 29:113–117, 2001.
103. MORAN, D. S., Y. HELED, L. STILL, A. LAOR, and Y. SHAPIRO. Assessment of heat tolerance for post exertional heat-stroke individuals. *Med. Sci. Mon.* 10(6):CR252–CR257, 2004.
104. MULDOON, S., P. DEUSTER, B. BRANDOM, and R. BUNGER. Is there a link between malignant hyperthermia and exertional heat illness? *Exerc. Sport Sci. Rev.* 32:174–179, 2004.
105. NADEL, E. R., C. B. WENGER, M. F. ROBERTS, J. A. J. STOLWIJK, and E. CAFARELLI. Physiological defenses against hyperthermia of exercise. *Ann. N. Y. Acad. Sci.* 301:98–109, 1977.
106. NATIONAL CENTER FOR HEALTH STATISTICS. *International Classification of Diseases, 9th revision*, Washington, D. C.: Department of Health and Human Services, 2001. Publication Number PHS-01-1260.
107. NATIONAL COLLEGIATE ATHLETIC ASSOCIATION. Five-day acclimatization period, Divisions I-A and I-AA. In: NCAA Division I Manual. Indianapolis, IN, 2003, Rule 17.11.2.3.
108. NATIONAL COLLEGIATE ATHLETIC ASSOCIATION. Game plan set for new preseason football practice policies. NCAA News June 9, 2003.
109. NICHOLSON, R. N., and K. W. SOMERVILLE. Heatstroke in a “run for fun”. *Brit. Med. J.* 1:1525–1526, 1976.
110. NIELSEN, B., and L. NYBO. Cerebral changes during exercise in the heat. *Sports Med. (New Zealand)* 33:1–11, 2003.
111. NOAKES, T. D. Body cooling as a method for reducing hyperthermia. *S. Afr. Med. J.* 70:373–374, 1986.
112. NOAKES, T. D. Fluid and electrolyte disturbances in heat illness. *Int. J. Sports Med.* 19:S146–S149, 1998.
113. NOAKES, T. D. Hyperthermia, hypothermia and problems of hydration. In: *Endurance in Sport, Vol. II*, 2nd edition, R. J. Shephard and P. O. Astrand. Oxford, England: Blackwell Science, pp. 591–613, 2000.
114. NOAKES, T. D., A. ST CLAIR GIBSON, and E. V. LAMBERT. From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans. *Br J Sports Med* 38:511–514, 2004.
115. NOAKES, T. D., and A. ST CLAIR-GIBSON. Logical limitations to the “catastrophe” models of fatigue during exercise in humans. *Br. J. Sports Med.* 38(1):648–649, 2004.
116. NOAKES, T. D., A. ST CLAIR GIBSON, and E. V. LAMBERT. From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans: summary and conclusions. *Br. J. Sports Med.* 39: 120–124, 2005.
117. NOBLE, H. B., and D. BACHMAN. Medical aspects of distance race planning. *Physician Sportsmed.* 7:78–84, 1979.
118. NYBO, L., and N. H. SECHER. Cerebral perturbations provoked by prolonged exercise. *Progr. Neurobiol.* 72(4):223–261, 2004.
119. NYBO, L., N. H. SECHER, and B. NIELSEN. Inadequate heat release from the human brain during prolonged exercise with hyperthermia. *J. Phys.* 545(2):697–704, 2002.
120. O'DONNELL, T. J. JR. Acute heatstroke, epidemiologic, biochemical, renal and coagulation studies. *J. A. M. A.* 234:824–828, 1975.
121. OH, R. C., and J. S. HENNING. Exertional heatstroke in an infantry soldier taking ephedra-containing dietary supplements. *Mil. Med.* 168(6):429–430, 2003.
122. PANDOLF, K. B., R. L. BURSE, and R. F. GOLDMAN. Role of physical fitness in heat acclimatization, decay and reinduction. *Ergonomics* 20:399–408, 1977.
123. PAYEN, J., L. BOURDON, H. REUTENAUER, et al. Exertional heatstroke and muscle metabolism: an in vivo <sup>31</sup>P-MRS study. *Med. Sci. Sports Exerc.* 24:420–425, 1992.
124. PIWONKA, R. W., S. ROBINSON, V. L. GAY, and R. S. MANALIS. Preacclimatization of men to heat by training. *J. Appl. Physiol.* 20:379–384, 1965.
125. PROULX, C. I., M. B. DUCHARME, and G. P. KENNY. Effect of water temperature on cooling efficiency during hyperthermia in humans. *J. Appl. Physiol.* 94:1317–1323, 2003.
126. PUGH, L. G. C. E., J. L. CORBETT, and R. H. JOHNSON. Rectal temperatures, weight losses and sweat rates in marathon running. *J. Appl. Physiol.* 23:347–352, 1967.
127. RICHARDS, R., and D. RICHARDS. Exertion-induced heat exhaustion and other medical aspects of the City-to-Surf fun runs, 1978–1984. *Med. J. Australia* 141:799–805, 1984.
128. RICHARDS, R., D. RICHARDS, P. J. SCHOFIELD, V. ROSS, and J. R. SUTTON. Reducing the hazards in Sydney's The Sun City-to-Surf Runs, 1971 to 1979. *Med. J. Aust.* 2:453–457, 1979.
129. RICHARDS, D., R. RICHARDS, P. J. SCHOFIELD, V. ROSS, and J. R. SUTTON. Management of heat exhaustion in Sydney's The Sun City-to-Surf fun runners. *Med. J. Aust.* 2:457–461, 1979.
130. RICHARDS, R., D. RICHARDS, P. J. SCHOFIELD, V. ROSS, and J. R. SUTTON. Organization of The Sun City-to Surf fun run, Sydney, 1979. *Med. J. Aust.* 2:470–474, 1979.
131. ROBERTS, W. O. Common threads in a random tapestry: Another viewpoint on exertional heatstroke. *Physician and Sportsmedicine* 33(10):42–49, 2005.
132. ROBERTS, W. O. Exercise-associated collapse in endurance events: a classification system. *Physician Sportsmed.* 17:49–55, 1989.
133. ROBERTS, W. O. Exertional heat stroke during a cool weather marathon: A case study. *Med Sci Sports Exerc* 38(7):1197–1202, 2006.
134. ROBERTS, W. O. Managing heatstroke: on-site cooling. *Physician Sportsmed.* 20:17–28, 1992.
135. ROBERTS, W. O. Assessing core temperature in collapsed athletes. *Physician Sportsmed.* 22:49–55, 1994.
136. ROBERTS, W. O. A 12-yr profile of medical injury and illness for the Twin Cities Marathon. *Med. Sci. Sports Exerc.* 32:1549–1555, 2000.
137. ROBINSON, S., J. R. NICHOLAS, J. H. SMITH, W. J. DALY, and M. PEARCY. Time relation of renal and sweat gland adjustments to salt deficiency in men. *J. Appl. Physiol.* 8:159–165, 1955.
138. ROBINSON, S., S. L. WILEY, L. G. BOUDURANT, and S. MAMLIN JR. Temperature regulation of men following heatstroke. *Isr. J. Med. Sci.* 12:786–795, 1976.
139. ROWELL, L. B. Cutaneous and skeletal muscle circulations. In: *Human Circulation: Regulation During Physical Stress.*, New York: Oxford University Press, pp. 96–116, 1986.
140. ROYBURT, M., Y. EPSTEIN, Z. SOLOMON, and J. SHEMER. Long term psychological and physiological effects of heatstroke. *Physiol. Behav.* 54:265–267, 1993.
141. SAWKA, M. N., S. J. MONTAIN, and W. A. LATZKA. Hydration effects on thermoregulation and performance in the heat. *Comp. Biochem. Physiol.* 128:679–690, 2001.
142. SAWKA, M. N., and K. B. PANDOLF. Effects of body water loss on physiological function and exercise performance. In: *Perspectives in Exercise Science and Sports Medicine. Vol. 3. Fluid Homeostasis During Exercise*, C. V. Gisolfi and D. R. Lamb. Carmel, IN: Benchmark Press, pp. 1–38, 1990.

143. SAWKA, M. N., A. J. YOUNG, R. P. FRANCESCOI, S. R. MUZA, and K. B. PANDOLF. Thermoregulatory and blood responses during exercise at graded hypohydration levels. *J. Appl. Physiol.* 59:1394–1401, 1985.
144. SAWKA, M. N., A. J. YOUNG, W. A. LATZKA, P. D. NEUFER, M. D. QUIGLEY, and K. B. PANDOLF. Human tolerance to heat strain during exercise: influence of hydration. *J. Appl. Physiol.* 73(1):368–375, 1992.
145. SCHNIRRING, L. Core temperature measurement goes high tech. Pill sensor enables wireless monitoring. *Physician Sportmed.* 32:10, 2004.
146. SHAHID, M. S., L. HATLE, H. MANSOUR, and L. MIMISH. Echocardiographic and doppler study of patients with heatstroke and heat exhaustion. *Int. J. Card. Imaging* 15:279–285, 1999.
147. SHAPIRO, Y., A. MAGAZANIK, R. UDASSIN, G. BEN-BARUCH, E. SHVARTZ, and Y. SHOENFELD. Heat tolerance in former heatstroke patients. *Ann. Intern. Med.* 90:913–916, 1979.
148. SHIBASAKI, M., I. YOSHIMITSU, N. KNODO, and A. IWATA. Thermoregulatory responses of prepubertal boys and young men during moderate exercise. *Eur. J. Appl. Physiol.* 75:212–218, 1997.
149. SHIBOLET, S., R. COLL, T. GILAT, and E. SOHAR. Heatstroke: its clinical picture and mechanism in 36:525–547, 1967.
150. SHIBOLET, S., M. C. LANCASTER, and Y. DANON. Heatstroke: a review. *Aviat. Space Environ. Med.* 47:280–301, 1976.
151. SHINOZAKI, T., R. DEANE, and F. M. PERKINS. Infrared tympanic thermometer: evaluation of a new clinical thermometer. *Crit. Care Med.* 16:148–150, 1988.
152. SHVARTZ, E., Y. SHAPIRO, A. MAGAZANIK, et al. Heat acclimation, physical fitness, and responses to exercise in temperate and hot environments. *J. Appl. Physiol.* 43:678–683, 1977.
153. SINERT, R., L. KOHL, T. RAINONE, and T. SCALEA. Exercise-induced rhabdomyolysis. *Ann. Emerg. Med.* 23:1301–1306, 1994.
154. SMALLEY, B., R. M. JANKE, and D. C. COLE. Exertional heat illness in air force basic military trainees. *Mil. Med.* 168: 298–303, 2003.
155. SMITH, H. R., G. S. DHATT, W. M. A. MELIA, and J. G. DICKINSON. Cystic fibrosis presenting as hyponatremic heat exhaustion. *Brit. Med. J.* 310:579–580, 1995.
156. SOHAR, E., D. MICHAELI, U. WAKS, and S. SHIBOLET. Heatstroke caused by dehydration and physical effort. *Arch. Intern. Med.* 122:159–161, 1968.
157. SPORTS MEDICINE AUSTRALIA. Revised hot weather guidelines. Web address <http://www.smasa.asn.au/>, web site active January 11, 2006.
158. ST CLAIR-GIBSON, A., and T. D. NOAKES. Evidence for complex system integration and dynamic neural regulation of skeletal muscle recruitment during exercise in humans. *Br. J. Sports Med.* 38(6):797–806, 2004.
159. STRAUSS, R. H. Skeletal muscle abnormalities associated with sports activities. In: *Sports Medicine*, Philadelphia: W. B. Saunders, pp. 159–164, 1984.
160. SUTTON, J. R. 43°C in fun runners! *Med. J. Australia* 2:463–467, 1979.
161. SUTTON, J. R. Heat illness. In: *Sports Medicine*, R. H. Strauss. Philadelphia: W. B. Saunders, pp. 307–322, 1984.
162. SUTTON, J. R. Thermal problems in the masters athlete. In: *Sports Medicine for the Mature Athlete*, J. R. Sutton and R. M. Brock. Indianapolis: Benchmark Press, pp. 125–132, 1986.
163. SUTTON, J. R. Clinical implications of fluid imbalance. In: *Perspectives in Exercise Science and Sports Medicine. Vol. 3. Fluid Homeostasis During Exercise*, C. V. Gisolfi and D. R. Lamb. Carmel, IN: Benchmark Press, pp. 425–455, 1990.
164. SUTTON, J. R., and O. BAR-OR. Thermal illness in fun running. *American Heart Journal* 100:778–781, 1980.
165. SUTTON, J. R., M. J. COLEMAN, A. P. MILLAR, L. LAZARUS, and P. RUSSO. The medical problems of mass participation in athletic competition. The “City-to-Surf” race. *Med. J. Aust.* 2:127–133, 1972.
166. TALBOT, J. H. Heat cramps. In: *Medicine*, Baltimore, MD: Williams and Wilkins, pp. 323–376, 1935.
167. U. S. DEPARTMENT OF HEALTH AND HUMAN SERVICES. Working in Hot Environments. DHHS Publication No. 86–112. Washington, D.C.: Public Health service, Centers for Disease Control, National Institute for Occupational Safety and Health, 1986.
168. WALLACE, R. F., D. KRIEBEL, L. PUNNETT, et al. The effects of continuous hot weather training on risk of exertional heat illness. *Med. Sci. Sports Exerc.* 37(1):84–90, 2005.
169. WEINER, J. S. An experimental study of heat collapse. *J. Ind. Hyg.* 20:389–393, 1938.
170. WEINER, J. S., and G. O. HORNE. A classification of heat illness. *Brit. Med. J.* 28(1):1533–1535, 1958.
171. WEIR, J. P., T. W. BECK, J. T. CRAMER, and T. J. HOUSH. Is fatigue all in your head? A critical review of the central governor model. *Br J Sports Med* 40(7):573–586, 2006.
172. WYNDHAM, C. H., M. C. KEW, R. KOK, I. BERSOHN, and N. B. STRYDOM. Serum enzyme changes in unacclimatized and acclimatized men under severe heat stress. *J. Appl. Physiol.* 37: 695–698, 1974.
173. WYNDHAM, C. H., and N. B. STRYDOM. The danger of inadequate water intake during marathon running. *S. Afr. Med. J.* 43:893–896, 1969.
174. YAGLOU, C. P., and D. MINARD. Control of heat casualties at military training centers. *Arch. Ind. Health* 16:302–305, 1957.
175. YARBROUGH, B. E., and R. W. HUBBARD. Heat-related illnesses. In: *Management of Wilderness and Environmental Emergencies*, 2nd edition, P. S. Auerbach and E. C. Geehr. St. Louis: C. V. Mosby, pp. 119–143, 1989.
176. YEARGIN, S. W., D. J. CASA, L. E. ARMSTRONG, G. WATSON, D. A. JUDELSON, E. PSATHAS, and S. SPARROW. Heat acclimatization and hydration status of American football players during initial summer workouts. *J. Strength Cond. Res.* 20(3):463–470, 2006.
177. ZUNIS FOUNDATION. Heat Stress Adviser: A Program For Palm-Type PDA's. World wide web address: [http://www.zunis.org/sports\\_p.htm](http://www.zunis.org/sports_p.htm) web site active January 11, 2006.