American Red Cross
American Red Cross Advisory Council on First Aid, Aquatics, Safety and Preparedness

ACFASP Scientific Review

Hyperthermia

Question to be Addressed

What are the current diagnostic criteria used to differentiate between the different types of hyperthermia (i.e., heat cramps, heat exhaustion, and heat stroke [exertional and classic]) and what are the current recommendations for managing these conditions?

Introduction

Any individual(s) regardless of training or activity level (sedentary vs. athletic), including athletes, agriculture and construction labors, as well as many other professions (those working indoors and outdoors) are at risk for developing heat-related illness when exposed to high ambient temperatures and humidity. This occurs because they are unable to adequately dissipate increases in body core temperature physiologically. Special populations including children, elderly, and individuals with chronic medical conditions are significantly at risk for developing heat related illnesses (Lugo-Amador, Rothenhaus, & Moyer, 2004), particularly classic heat stroke (longer onset compared to exertional heat stroke). Risk of hyperthermia increases during intense exercise or under certain environmental conditions (Centers for Disease Control, 1996, 1999; Centers for Disease Control and, 2003; Centers for DiseaseControl, 2000; Dematte et al., 1998; Donaghue et al., 2003; Luber & Sanchez, 2006; Piver, Ando, Ye, & Portier, 1999; Poumadère, Mays, Le Mer, & Blong, 2005), particularly exertional heat stroke. Athletes (Binkley, Beckett, Casa, Kleiner, & Plummer, 2002; Brennan & O'Connor, 2005; D. Casa et al., 2000; Cooper, Ferrara, & SP, 2006; Epstein, Moran, Shapiro, Sohar, & Shemer, 1999; Francis, Feinstein, & Brasher, 1991; Murphy, 1984; W. Roberts, 2007; Stofan et al., 2003), military personal (Assia, Epstein, & Shapiro, 1985; Atar, Rozne, & Rosenfeld, 2003; Heled, Rav-Acha, Shani, Epstein, & Moran, 2004; Kark, Burr, Wenger, Gastaldo, & Gardner, 1996; Rav-Acha, Shuvy, Hagag, Gomori, & Biran, 2007; Shibolet, Lancester, & Danon, 1976) and certain professions (Faerevik & Reinertsen, 2003; McLellan & Selkirk, 2004) are also at a greater risk for developing heat related illnesses when the compensatory adjustments in the circulatory, thermoregulatory, and endocrine systems fail due to variety of inherent and external risk factors.

Exertional heat related illnesses traditionally are broken down into three categories: 1) heat cramps, 2) heat exhaustion, and 3) heat stroke. These illnesses are normally encountered while engaging in physical activity in warm and hot environments, however, exercise, or increased physical activity is not a prerequisite to the development of heat related illnesses. Of the heat illnesses listed, heat cramps are considered least severe and are considered the least severe and a possible prelude to more serious heat related illnesses such as heat exhaustion and heat stroke.

Heat exhaustion left untreated may manifest into heat stroke, a life-threatening condition (Rav-Acha, Hadad, Epstein, Heled, & Moran, 2004; Schnirring, 2004).

Non-exertional heat related illnesses may also lead to death. For example in 2003 over 14,000 deaths were directly attributed to the heat during the 2003 heat wave in France. Factors leading to death included dehydration, hyperthermia and heat stroke (Poumadère et al., 2005). Other factors leading to the overwhelming number of deaths occurred due a combination of factors such as preexisting medical conditions, certain medications, urban residences, isolation, poverty, and probably air pollution (Centers for Disease Control, 1999; Centers for Disease Control and, 2003; Centers for DiseaseControl, 2000; Piver et al., 1999; Poumadère et al., 2005; Weisskopf et al., 2002).

Because of the potential threat to life, remaining aware of and being able to recognize the most common signs and symptoms of heat related illnesses and acting accordingly in a timely fashion by any level of emergency medical responders (i.e., all levels of first aid providers) and the lay public can greatly reduce the risk of developing a heat related illness and has the potential to improve outcomes. Therefore, emergency medical responders should be aware of the most accurate signs and symptoms of a variety of heat related illness and most current evidence based management of said conditions. Emergency medical responders should also be able to initiate the most appropriate and feasible treatment available to reduce heat related morbidity and mortality.

Review Process and Literature Search Performed

Medline, CINAHL, SPORTDiscuss, and Academic Search Premiere databases were searched using the terms “heat-illness” (90 hits), “hyperthermia” (3676 hits – narrowed to “body temperature regulation” = 30 hits), “heat cramp” (12 hits), “heat exhaustion” (133 hits), and “heat stroke” 229 hits). The search was limited to full text, peer-reviewed, English only articles with available abstracts. No specific date guidelines were set. In situations where a review article referred a possible source, the abstract and/or article was located and reviewed at www.pubmed.com or at one of the above databases. To date approximately 150 abstracts, articles, textbooks have been read and reviewed. Ninety of roughly 150 sources reviewed were utilized in this paper.

Summary of Findings

Heat Cramp

Heat cramps, often considered the mildest form of heat related illnesses are defined as painful involuntary muscle spasms of skeletal muscle(s) often associated with fluid and electrolyte (i.e., sodium, potassium, calcium) loss. Heat cramps most commonly occur in the calves and

abdomen, however; whole body cramps are plausible (Bergeron, 2003; A. Dreyer & M. Kulesa, 2002; Griffin, 2006; Wexler, 2002). In one study, heat cramps were the most commonly reported exertional heat related illness in collegiate football players during the month of August at five universities in the southeastern region of the United States (Cooper et al., 2006) (LOE 2a).

Currently no exact cause of heat cramps exists in the literature. Possible contributing factors to the development of heat cramps include but are not limited to: 1) strenuous exercise, 2) dehydration (loss of fluid and electrolytes), 3) high ambient temperatures, 4) i.e., inadequate sodium intake, 5) poor hydration, and 6) lack of adequate sleep (LE Armstrong et al., 2007; Bergeron, 2003, 2007; Binkley et al., 2002; D. Casa et al., 2000; A. Dreyer & M. Kulesa, 2002). First aiders may be all to limit the development of heat cramps through proper prevention and identification of predisposing factors (LE Armstrong et al., 2007; Binkley et al., 2002) . Individuals at greatest risk for exertional heat cramps include but are not limited to individuals: 1) having a past history of cramps, 2) high body mass index, 3) poor physical conditioning, 4) overexert themselves in excessive heat, 5) failure to acclimate to the environment(Gardner & Kark, 2003) and 6) failure to develop and implement a proper hydration and diet plan (Bergeron, 2003, 2007; Binkley et al., 2002; D. Casa et al., 2000; Cleary, 2007).

Commonly reported signs and symptoms of heat related cramps reported in the literature are found in table 1.

Table 1. Commonly Reported Signs and Symptoms of Heat Cramps.

Signs	Symptoms
• Decreased water intake (thirst) • Loss of 2% body water (sweating) • Loss of electrolytes (e.g., sodium and chlorine) • Palpable muscle spasm • Tachycardia • Dehydration	• Fatigue • Intense pain • Thirst

(L Armstrong et al., 2007) (LOE 5); (Bergeron, 2003) (LOE 5); (Bergeron, 2007) (LOE 5); (Binkley et al., 2002) (LOE 5); (Coris, Ramirez, & Van Durme, 2004) (LOE 5); (A. R. Dreyer & M. G. Kulesa, 2002) (LOE 5); (Wexler, 2002) (LOE 5).

Common treatment recommendations for managing of heat cramps includes: 1) rest and removal from the activity, 2) fluid and electrolyte replacement, 3) cryotherapy, and 4) muscle stretching. Table 2 provides a summary of these recommendations.

Table 2. Recommended Treatment for Heat Cramps.

Treatment	Author	LOE
• Discontinue all activity and sit in a cool environment.	• (Coris et al., 2004; A. R. Dreyer & M. G. Kulesa, 2002; Griffin, 2006)	5
• Rehydrate using water (may add 1/8–1/4 teaspoon of table salt to 10-16 oz of fluids or bullion broth) or a commercial electrolyte solutions (Sports Drinks – 16 to 20 ounces). • May add 0.5 teaspoon (3 g) of salt to the sports drink.	• (L Armstrong et al., 2007; Binkley et al., 2002; Wexler, 2002) • (Bergeron, 2007)	5 5
• Prolonged stretch with the muscle groups at full length.	• (L Armstrong et al., 2007; A. R. Dreyer & M. G. Kulesa, 2002; Wexler, 2002)	5
• Apply an ice application, either crushed ice or ice massage.	• (Coris et al., 2004; A. R. Dreyer & M. G. Kulesa, 2002)	5
• Eat a well balanced meal to ensure adequate consumption of electrolyte. If inadequate access to meals or meals are not eaten regularly is a common occurrence, consider adding sodium chloride (salt) at meal time.	• (Bergeron, 2007)	5
• Do not return to strenuous activity for a few hours after the cramps subside, because further exertion may lead to heat exhaustion or heat stroke. • Seek medical attention for heat cramps if they do not subside in 1 hour or spread to other parts of the body.	• (A. R. Dreyer & M. G. Kulesa, 2002)	5

Heat Exhaustion

Heat exhaustion is a medical condition caused by an elevated body temperature as the body's thermoregulatory system begins to become over-taxed particularly during physical activity and/or high ambient temperatures. This heat illness is most often seen in individuals in poor physical condition (LE Armstrong et al., 2007; Binkley et al., 2002) (i.e., not acclimatized) who attempt to exercise in the heat, however; individuals exposed to hot, humidity, work environments such as aircrews (Faerevik & Reinertsen, 2003), firefighters (McLellan & Selkirk, 2004), athletes (Binkley et al., 2002; Cleary, 2007; Cooper et al., 2006; Francis et al., 1991), agricultural labors and construction workers (Mirabelli & Richardson, 2005) are at increased risk. Predisposing factors such as: 1) increase in ambient temperature, 2) increased humidity, 3) work-related activity (i.e, poor ventilation, humid environment), and 4) athletic performance cause an increase metabolic activity (Binkley et al., 2002; Faerevik & Reinertsen, 2003; Wexler, 2002). As a result of increased metabolic activity, the body generates higher levels of internal heat causing an increase in core body temperature. In fact, during exercise internal heat production in the muscle may increase more than 20-fold to that of an individual's body at rest and can raise core body temperature by 1°C (1.8°F) every 5 minutes if no heat is removed from the body (Nadel, Wenger, Roberts, Stolwijk, & Cafarelli, 1977). As core body temperature increases the need for cooling increases in an effort to maintain thermoregulatory homeostasis for normal body functions to continue.

To maintain a homeostatic body temperature the body initiates a cooling process which is critical to maintaining a relatively constant body core temperature (between 97-100 ºF). The body's parasympathetic system initiates sweating and the sympathetic system causes an increase in blood vessel diameter and increased blood flow to allow for the dissipation of the increased core temperature (R. Kenefick & M. Sawka, 2007; Wexler, 2002) through the sweating process. The increased blood flow to the skin is also associated with reduced cardiac filling, reduced right atrial pressure and reduced stroke volume, thereby requiring a higher heart rate (tachycardia) to maintain an individual's cardiac output (R. Kenefick & M. Sawka, 2007; Noakes, 2006). A reduction in cardiac output and increased vasodilatation however also make it difficult to maintain an adequate blood pressure (Carter, Cheuvront, Vemieuw, & Sawka, 2006) and is manifested as hypotension upon an emergency medical responder's initial evaluation. Un-coordination, mental confusion, and irritability have also been reported to as signs of heat exhaustion (Binkley et al., 2002; Casey, 2006; A. Dreyer & M. Kulesa, 2002; Lee-Chong & Stitt, 1995; Parson, 1993; Wexler, 2002) and may be the result of diminished cerebral blood flow due to the reduction in cardiac output and vasodilatation of the peripheral blood vessels (Carter et al., 2006). Decreased performance of the cardiac system also affects the body's ability to deliver sufficient amount of blood volume to the peripheral tissues in order to maximize cooling and allow for continued physical activity (Binkley et al., 2002).

Sweating accounts for about 80% of an individual's heat loss, however, while sweating is necessary to maintain a normal body temperature because it is one of the body's natural response for thermoregulation (evaporation) it also forces increased fluid and electrolyte lose (L. E. Armstrong, Hubbard, Szlyk, Sils, & Kraemer, 1988; D. Casa et al., 2000; R. Kenefick & M. Sawka, 2007). If fluids and electrolytes (e.g., sodium, potassium, chlorine) are not replaced an individual is considered at greater risk of dehydration (reduced blood volume), resulting in further decreased physical performance, impaired skin blood flow, delayed sweat response, decreased ability to sweat, increased core temperature, and altered cardiac (D. Casa et al., 2000; R. Kenefick & M. Sawka, 2007) and vital organ function (LE Armstrong et al., 2007; Binkley et al., 2002; Wexler, 2002).

Signs and symptoms of heat exhaustion include those commonly seen with heat cramps as well as those found in table 3. Hallmark signs of heat exhaustion commonly reported in the literature is an individual who presents with fatigue and cool, clammy skin (Binkley et al., 2002; Griffin, 2006; Luber & Sanchez, 2006). Skin which is cool and moist indicates that the body's mechanism for cooling is still functioning (Luber & Sanchez, 2006). Alternation in cardiac output leads to a rapid, weak pulse and rapid, shallow breathing (Luber & Sanchez, 2006). Individuals suffering from heat exhaustion are reported to still be sweating at this point in time. If heat exhaustion though is not recognized and dealt with immediately the likelihood of further complications such as heat stoke is increased (Binkley et al., 2002; Centers for Disease Control, 1996; A. Dreyer & M. Kulesa, 2002; Griffin, 2006; Wexler, 2002).

Distinguishing between heat exhaustion and heat stoke requires measurement of an individual's core body temperature. The literature suggests that a core body temperature is usually higher than 38-39 ° C (100.4 -102.2° F) but below the cutoff for heatstroke, which is 40° C (104° F) (Binkley et al., 2002; Casey, 2006; R. Kenefick & M. Sawka, 2007; Wexler, 2002). Kenefick and Swaka (2007) (LOE 5) suggest that a relationship exists between core temperature and incidence of exhaustion from heat strain for heat-acclimated persons exercising in uncompensable (most likely very hot skin) and compensable (most likely cool skin) heat stress. During compensable heat exhaustion the core body temperature will be greater than 39° C (102.2° F) and the individual will present with cool skin (R. Kenefick & M. Sawka, 2007).

During uncompensable heat related illness, heat exhaustion is often associated with a core temperature less than 39° C (102.2° F), and individuals present with very hot skin. Furthermore they suggest that heat exhaustion will almost always occur by a core temperature of 40° C (104° F) (an indicator of heat stroke). They further suggest that "… higher skin temperature (>35°C (95° F), as seen during uncompensable heat stress) will be associated with greater cardiovascular strain for a given core temperature and therefore result in earlier exhaustion.

Table 3. Commonly Reported Signs and Symptoms of Heat Exhaustion.

Signs	Symptoms
• Decreased water intake (thirst) • Loss of 2% body water (sweating) • Loss of electrolytes (sodium) • Muscle cramps • Tachycardia • Dehydration • Hypotension • Syncope • Decreased urination • Uncoordination • Confusion • Irritability • Normal or elevated body-core temperature (usually > 38° C (100.4° F) but below the cutoff for heatstroke, which is 40° C (104° F))	• Fatigue • Nausea/vomiting • Headache • Malaise/myalgias • Lightheadedness

(Binkley et al., 2002) (LOE 5); (Casey, 2006) (LOE 5); (A. R. Dreyer & M. G. Kulesa, 2002) (LOE 5); (Glazer, 2005) (LOE 5); (R. W. Kenefick & M. N. Sawka, 2007) (LOE 5); (Lee-Chong & Stitt, 1995) (LOE 7); (Lee-Chong & Stitt, 1995) (LOE 3a); (O'Connor et al., 2007) (LOE 5); (Parson, 1993) (LOE 7); (Wexler, 2002) (LOE 5).

Faervik and Reinertsen (2003) (LOE 1b) examined the effects of three different environmental conditions on aircrews wearing protective clothing at 0° C at 80% relative humidity (RH), 23°C at 63% RH and 40°C (104°F) at 19% RH for 3 hours. They found a rise in rectal (core) temperature, skin temperature, heart rate and body water loss in the 40°C ambient temperature condition compared to with the other two. They concluded that over long periods of time, wearing protective clothing in hot ambient temperatures increases the risk of increased core thermal temperatures and associated heat related conditions.

Heat Stroke

Heat exhaustion may quickly progress to a life-threatening condition commonly referred to as heat stroke (LE Armstrong et al., 2007). Normal metabolic heat production, coupled with changes in environmental conditions (e.g., increased heat index, high ambient temperatures) that overpowers the body's ability to dissipate heat, thereby increasing the body's core temperature causes life threatening heat stroke (L Armstrong, 1992; LE Armstrong et al., 2007; Binkley et al., 2002; Brendon et al., 2007; Centers for Disease Control, 1996; E Hadad, Rav-Acha, Heled, Epstein, & Moran, 2005; Heled et al., 2004; W. O. Roberts, 2007b). Failure of the body's ability to regulate elevated core temperatures increases the risk of multi-system organ failure (Binkley et al., 2002; Dematte et al., 1998) such as circulatory collapse, neurological dysfunction, renal failure, rhabdomyolysis, liver failure, cerebral congestion, pulmonary congestion, disseminated intravascular coagulation, immune system dysfunction (Carter, Cheuvront, & Sawka, 2007; Gardner & Kark, 2003; O'Connor et al., 2007; Rav-Acha et al., 2004; Rav-Acha et al., 2007; Shephard & Shek, 1999; Tan & Herzlich, 1995) and complications such as transient cardiac dysfunction, pulmonary edema, (Atar et al., 2003), seizures and cerebral edema (Weir, 2002) and in rare cases persistent neurological complications (Rav-Acha et al., 2007).

Two types of heat stroke are typically reported in the literature, classic heatstroke, and exertional heatstroke. Classic heatstroke is typically caused by caused by environmental changes (i.e., extreme weather events with climate changes and high ambient temperatures) (Greenough et al., 2001; Kare & Shneiderman, 2001) and often occurs during the summer months (Wexler, 2002). Classic heatstroke most often occurs in infants, children, the elderly (Bross, Nash, & Carlton, 1994; Hamdy, 2002; Hiss, Kahana, Kugel, & Epstein, 1994; Kare & Shneiderman, 2001; Lugo-Amador et al., 2004; Wexler, 2002), those with chronic medical illnesses (Lugo-Amador et al., 2004; Wexler, 2002) and individual who suffer from inefficient body heat-regulation mechanisms (Hart et al., 1982) those in poor socioeconomic settings with limited access to air conditioning (E Hadad et al., 2005) and on certain medications (Atar et al., 2003; Bross et al., 1994) such as antihistamines, amphetamines, anticonsulivants, anticholinergics, diuretics and blood pressure and heart medicines (Centers for Disease Control, 1999; Howell, 2005; Kare & Shneiderman, 2001). Typically classic heatstroke develops slowly, over a period of several days with patients presenting with minimally elevated core temperatures (Glazer, 2005; Lugo-Amador et al., 2004).

Heat waves (defined as temperatures > 90° for > 3 days) in Chicago, IL during 1996 and 2001 resulted in 588 heat related deaths (Donoghue et al., 2003) (LOE 3a) with age being a contributing a factor. From 1999–2003, 3,442 deaths in the United States resulting from exposure to extreme heat were reported where 65% (2,239) of these death were identified with an underlying causes as being exposure to excessive heat (Luber & Sanchez, 2006) (LOE 3a). The remaining 35% of the death listed hyperthermia as the contributing factor, with 1,810 (53%)

victim between the ages of 15–64 years, and 1,363 (40%) greater than 65 years of age (Luber & Sanchez, 2006).

During the summer of 1998, the Missouri Department of Health attributed 12 deaths to high temperatures, and the state's heat surveillance system recorded 470 heat-related illnesses: the average age among decedents was 65.6 years (range: 4–92 years; median 73.5 years); seven (58%) decedents were female (Centers for Disease Control, 1999) (LOE 3a). Contributing to heat-related illness were alcohol consumption (resulting in dehydration), previous heatstroke, physical activity (e.g., exertion during work or recreation), and the use of medications that interfere with the body's heat regulatory system, such as neuroleptics (antipsychotics or major tranquilizers) and medications with anticholinergic effects (e.g., tricyclic antidepressants, antihistamines, some antiparkinsonian agents, and some over-the-counter sleeping pills.

In France during the summer of 2003 another heat wave led to an excess of 14,947 deaths between August 4 and 18, 2003 (Poumadère et al., 2005) (LOE 3a). Poumadère et al. reported that the high mortality rate was directly attributed to heat, dehydration and hyperthermia or heat stroke. Factors such as age, heath status, medication, urban residence, and duration of the heat wave all were associated with the high mortality rate. For example, Poumadère et al. found the mortality for individuals over "75 years amounted to 1,200 deaths for nine consecutive days of heat over 35°C, with a progressive return to normal mortality as the maximum temperature sank to below 30°C."

Exertional heatstroke however is the opposite of classic heatstroke as classic heat stroke is generally found in the older population. Exertional heatstroke, which primarily affects younger, active individuals such as athletes (recreational and competitive), military recruits, and heavy labors (Assia et al., 1985; Brendon et al., 2007; Brennan & O'Connor, 2005; Cleary, 2007; Coris et al., 2004; Epstein, 2000; Epstein et al., 1999; Gardner & Kark, 2003; Glazer, 2005; Hiss et al., 1994; Kark et al., 1996; R. Kenefick & M. Sawka, 2007; Wexler, 2002) is a "state of extreme hyperthermia that occurs when excess heat that is generated by muscular exercise exceeds the body's ability to dissipate it at the same rate" (Epstein et al., 1999) (LOE 2c). A review of fatal cases of exertional heat stroke found that many of subjects exerted themselves beyond their physical limits (Rav-Acha et al., 2004) (LOE 2c). Exposure to factors such as high ambient temperatures, high relative humidity, increased workloads (i.e., increased production internal heat), dehydration, and poor thermoregulation homeostasis increases the risk for developing exertional heat stroke as the body's cooling system shuts down resulting in the body producing dangerous levels of internal core heat (LE Armstrong et al., 2007; Binkley et al., 2002) (LOE 5 and 5).

Exertional heatstroke is often characterized by a rapid onset, developing in hours (in some cases within 2 hours of exercise) (Epstein et al., 1999; Heled et al., 2004) (LOE 2c and 2c) rather than

days and is commonly associated with increased core body temperatures greater than> 40°C (104°F) (LE Armstrong et al., 2007; Binkley et al., 2002; Epstein, 2000; Glazer, 2005; E Hadad et al., 2005; Heled et al., 2004; O'Connor et al., 2007; W. Roberts, 2007) (LOE 5 and 2c for Heled, Rav-Acha, et al). It is believed that this increase in body core temperature is due to the reduction in central venous pressure which is caused by an increase in fluid loss (i.e., sweating and dehydration) and peripheral blood loss (As cited in Epstein, 2000). Severe volume depletion leads to peripheral vasoconstriction and a decrease in heat transfer to the body's peripheral system (A. Dreyer & M. Kulesa, 2002) (LOE 5). Further reduction in central venous pressure results in an extensive decrease in skin blood flow and blood volume. This further increases the body's core temperature as the ability to transport the increased internal heat produced by the exercise and as a loss of fluid does not allow for removal of the heat. Vasodilatation of the peripheral blood vessels also causes tachycardia (a rate of up to 180 beats per minute) and an elevated cardiac index (A. Dreyer & M. Kulesa, 2002) (LOE 5). The risk of morbidity increases the longer the victim's core body temperature remains elevated above 41°C (106°F) (Assia et al., 1985; Graham, Lichtenstein, Hinson, & Theil, 1986; Vicario, Okabajue, & Haltom, 1986) (LOE 2c).

Signs and symptoms of heat stroke include those commonly seen with heat exhaustion, as well as those presented in table 4. One of the hallmark signs of heat stroke often proposed in many emergency medical textbooks and literature is warm/hot, red, dry skin (Atar et al., 2003; Donaghue et al., 2003; E. Hadad, Rav-Acha, Heled, Epstein, & Moran, 2004; Kim, 1989; Vera & Cross, 1993) (LOE 2c, 3a, 5, 5, 7) particularly when dealing with exertional heatstroke (Newsham, Saunders, & Nordin, 2002) (LOE 1b), as peripheral vasoconstriction often is present (Glazer, 2005) (LOE 5), limiting the body's ability to sweat. However, Newsham, et al (2002) point out that many victims of exertional heatstroke present with sweat-soaked clothing and that "contrary to previous beliefs, victims of exertional heat stroke may present with perspiration, not dry skin" (as cited in Newsham) as they may continue to sweat (Glazer, 2005; Roberts, 1998) (LOE 5) as the sweat glands continue to remain active (Epstein, 2000). Shibolet, Lancaster and Danon (1976) (LOE 5) in their review of heat stoke found that dry skin only presented itself as a clinical sign in 26% of the cases reviewed. Therefore, potential emergency medical responders should be made aware that heat stroke should still be considered even in the presence of moist, warm skin.

The most critical clinical indicators of heat stroke though involves measurement of the victim's body core temperature (LE Armstrong et al., 2007; Binkley et al., 2002; D. Casa et al., 2007; D. J. Casa, Anderson, Armstrong, & Maresh, 2006; D. J. Casa, Armstrong, Ganio, & Yeargin, 2005; Moran & Mendal, 2002; Newsham et al., 2002) (LOE 5, 5, 1b, 5, 5, 5, 1a). The longer the rectal exceeds a critical threshold temperature (> 40° C or 104° F) the greater the likelihood of cell damage and greater the dictates the severity of injury (Bouchama & Knochel, 2002; E Hadad et al., 2005; Rav-Acha et al., 2004; Rav-Acha et al., 2007) (LOE 5, 5, 2c 3b). The American

Table 4. Commonly Reported Signs and Symptoms of Heat Stroke.

Signs	Symptoms

• Hypotension (< 90mm Hg)
• Inability to tolerate heat
• Hyperventilation
• Shock
• Moderate to Sever changes to the CNS
  o AMS
  o Disorientation
  o Coma
  o Convulsions
  o Irrational behavior
  o Attention deficit
• Core body temperature is usually higher than 40° C (104° F) as measure with a rectal thermometer
• Coma
• Seizures
• Diarrhea
	• Vertigo
	• Tingling in the fingers

Note: These signs and symptoms are in conjunction with those identified in table 3 under heat exhaustion. Not all individuals will present will all of the above signs and symptoms.

(LE Armstrong et al., 2007) (LOE 5); (Binkley et al., 2002) (LOE 5); (Bouchama & Knochel, 2002) (LOE 5); (Brennan & O'Connor, 2005) (LOE 5); (Cooper et al., 2006) (LOE 1b); (Costrini, Pitt, Gustafson, & Uddin, 1979) (LOE 2a); (Donaghue et al., 2003) (LOE 3a); (Eichner, 2002) (LOE 7); (Gardner & Kark, 2003) (LOE 6); (Glazer, 2005) (LOE 5); (Kare & Shneiderman, 2001) (LOE 5); (O'Connor et al., 2007) (LOE 5); (W. O. Roberts, 2007a) (LOE 5, 3b); (Shibolet et al., 1976) (LOE 5); (Coris et al., 2004) (LOE 5).

Academy of Sports Medicine (ACSM) (LE Armstrong et al., 2007) (LOE 5), National Athletic Trainers' Association (NATA) (Binkley et al., 2002) (LOE 5) and others (D. Casa et al., 2007; D. J. Casa et al., 2006; D. J. Casa et al., 2005; Moran & Mendal, 2002; W. O. Roberts, 2007a) (LOE 5, 5, 5, 1a, 3b) all recommend the use of a flexible rectal thermometer to assess core temperature for any victim suspected of a heat related illness as the gold standard in measuring core temperature. However, the feasibility and practicality of a flexible rectal thermometry for many emergency medical responders and all basic first aiders is an unrealistic option.

Some researchers have suggested that the use of tympanic temperature measurement may provide a reliable measurement of body core temperature (D. J. Casa et al., 2007; Newsham et al., 2002; Tayeb & Marzouki, 1989). Newsham, Saunders, and Nordin (2002) (LOE 1b) found that tympanic thermometry was a reliable indicator of changes in body core temperatures, demonstrating a difference of less 1° C between tympanic and rectal temperatures. Another study to examine the reliability of measuring body temperature at the tympanic membrane using a sheep model found no major superiority of tympanic over rectal thermometry to detect heat stroke cases (Tayeb & Marzouki, 1989) (LOE 4). However, organizations such as ACSM and NATA both suggest avoiding the use of ear (aural canal or tympanic membrane), oral, skin over the temporal artery, and axillary temperature measurements when diagnosing exertional heat stroke (LE Armstrong et al., 2007; Binkley et al., 2002) (LOE 5). A recent study by Casa et al. (2007) (LOE 1a) compared rectal temperatures with other methods used to measure body temperature such as oral, axillary, aural, temporal, and field forehead temperatures were found to be significantly different from the gold standard, rectal temperatures. Gastrointestinal temperature using a digital pill was the only measurement that accurately reflected the rectal temperature. The ACSM suggests that factors such as the temperature of air, skin, and any liquids that come in contact with the skin (as cited in Armstrong et al., 2007) results in a false negative and may not accurately depict the current state of the core temperature.

Evidenced Based Findings for Treatment

Heat Cramp – Treatment

Gentle stretching and proper hydration before exercise can help prevent heat cramps (A. Dreyer & M. Kulesa, 2002) (LOE 5). Because heat cramps are associated with nutritional deficits, the addition of extra salt in foods can be useful (A. Dreyer & M. Kulesa, 2002) (LOE 5) or the use of a sports drink can assist to replace the salt and minerals lost during sweating (Centers for Disease Control, 1996) (LOE 7). However, salt tablets are not indicated because gastric irritation can occur (A. Dreyer & M. Kulesa, 2002) (LOE 5) and should be prescribed by a physician. If salt tablets (1 g of NaCl per tablet) the tablets should be taken with plenty of fluid (eg, 3 crushed and dissolved tablets to 1 liter of water). When heat cramps due occur individuals should be cautioned to avoid strenuous activities for a few hours after the cramps subside because this can lead to heat exhaustion or heat stroke (A. Dreyer & M. Kulesa, 2002).

Bergeron (2007) (LOE 5) suggests that the recovery priority from a nutrient perspective for an individual's suffering exertional heat cramps is to replace and retain the salt (NaCl) and water lost during periods of sweating. During this rehydration period it is recommended that in order to better retain and sufficiently distribute the ingested water throughout the extracellular and intracellular fluid compartments, enough NaCl should be provided concomitantly with the appropriate amount of fluid. However, prevention of such an instance is recommended. This can be accomplished if an appropriate response is initiated at the first sign of muscle twitching.

Consuming 16 to 20 ounces (~0.5 L) of a sport drink (such as Gatorade®) with 0.5 teaspoon (3 g) of salt added and thoroughly mixed (consumed either all at once, if tolerable, or distributed over 10 minutes or so, with a little additional water) has been shown to be effective in relieving cramping or preventing muscle twitches from developing any further.

The treatment recommend by Griffin (2006) (LOE 5) includes rest, in a cool open place, preferably one with access to a breeze and stretching of the affected muscle. Increased fluid intake using sports drinks to help replace fluids and sodium

Coris, Ramirez, and Van Durme (2004) (LOE 5) in their review paper provides only cursory information on the treatment of heat cramps, recommending stretch, ice massage, and replacing fluid and sodium loss. Ice massage reduces muscle cramps by suppressing the stretch reflex via 2 mechanisms: 1) decrease pain by reducing the threshold of the afferent nerve endings, 2) decrease the sensitivity of muscle spindle activity (Starkey, 2004).

Auber (2004) (LOE 7) recommends that once an individual has been identified as suffer from heat cramps to have him stop what he/she's doing, move him/her to a shady or cool location if possible, and encourage him to drink cool fluids, preferably a sports drink containing electrolytes.

In Wexler's (2002) (LOE 5) review of heat-related illnesses muscle cramping was believed to be the result of a lack of fluid intake and fluid loss during sweating in conjunction with sodium depletion. Poorly conditioned individuals may lose up to 1-2 L of fluid and 65 mEq per L of sodium per hour while highly conditioned individuals may lose 3-4 L of fluid per hour. Treatment recommendations include stretching the affected muscle, maintaining good hydration, commercial electrolyte solutions (Sports Drinks), and increased dietary salt to help prevent cramps.

In Binkely, et al (2002) (LOE 5) the inclusion of sodium chloride in fluid-replacement beverages for individuals suffering electrolyte imbalance should be considered under the following conditions: 1) inadequate access to meals or meals not eaten; 2) physical activity exceeding 4 hours in duration; 3) or during the initial days of hot weather. Under these conditions, they recommend adding modest amounts of salt (0.3 to 0.7 g/L) to offset salt loss in sweat and minimize medical events associated with electrolyte imbalances (e.g, muscle cramps). Adding a modest amount of salt (0.3 to 0.7 g/L) to all hydration beverages would also be acceptable to stimulate an individual's thirst reflex and increase voluntary fluid intake (LOE 5).

Management of Heat Exhaustion and Heat Stroke by First Aid Providers

Casa et al (2007) (LOE 5) and Smith and Wallis (2005) (LOE 5) provides scientific evidence supporting the use cold water immersion for athletes experiencing exertional heatstroke. Casa et al (2007) states the number one criticism of cold water immersion is that patients will actually heat up (or at least not cool down) in cold water because of peripheral vasoconstriction (PVC)

and shivering. They further state that evidence from basic physiological studies looking at the effect of cold water immersion on cooling rates in hyperthermic individuals and treatment of actual exertional heat stroke victims clearly shows that cold water immersion has cooling rates superior to any other known modality as cited [(LE Armstrong, Crago, Adams, Roberts, & Maresh, 1996) (LOE 1b); (Clements et al., 2002) (LOE 1b); (Costrini et al., 1979) (LOE 2a); (Proulx, Ducharme, & Kenny, 2003) (LOE 2a)] in Casa et al (2007). However, a rescuer must consider which cooling modalities are feasible and optimal for each particular circumstance such as logistics of moving an individual into and out of a tub; maintenance of the airway, breathing, and circulation; and monitoring of temperature measurements and access to definitive care.

Casa et al (2005, 2007) (LOE 5) and Smith and Wallis (2005) (LOE 5) also suggests that the superior cooling rates documented in literature favors cold water immersion studies. However, Casa et al (2005, 2007) point out that other cooling methods such as a combination of spraying moderate-temperature water plus rapid air movement provided by a fan encourage evaporative and convective cooling with good results in military personnel (E Hadad, Moran, & Epstein, 2004) (LOE 3b). Others have placed cold, wet towels over the entire body and/or continuously doused with cold water and placed ice packs on peripheral arteries (and massaging the limbs with ice bags in some situations) Massaging the limbs with ice bags results in cooling rates that are 2/3 to 3/4 as fast as CWI, and because medical care begins immediately, survival rates are believed to be excellent (McDermott, Casa, Adams, & et al, 2007) (LOE 2a). Casa et al (2007) also recommends that medical staff personnel should also consider cold showers or dousing athletes with cold water from a hose when cold water immersion is not possible.

Casa et al (2007) (LOE 5) recommends the following emergency management individuals (i.e., athletes) suffering from exertional heat stroke:

1. Measure rectal temperature and use clinical judgment regarding central nervous system dysfunction and other signs and symptoms to quickly and accurately determine the patient's condition and whether EHS is occurring [1,3,8,9,14,28 as cited in Casa et al 2007].

2. Begin cooling individuals with exertional heat stroke as rapidly as possible. The concept of "cool first, transport second" is strongly recommended, assuming that properly trained medical professionals, such as athletic trainers or physicians, are present to complete the cooling on-site via cold water immersion (or a suitable alternative modality if cold water immersion is not possible) and then transport to a medical facility. This method eliminates delays in treatment caused by the time constraints of arrival of care, transport, and the possibility that cooling may not be immediate or aggressive at the hospital. This protocol should be discussed with supervisors, colleagues, and adjunct medical personnel before a case of EHS occurs, so that the involved parties are in agreement during the stress of the moment [7 as cited in Casa et al 2007]. Implementing these recommendations emphasizes in the strongest possible manner the importance of

immediate and aggressive cooling and the critical initial 30-60 min after exertional heat stroke onset.

3. Use a cooling modality that has cooling rates sufficient to lower core temperature to less than 40°C (104°F) within 30 min. And while no specific cooling modality was identified by Casa et al (2007) Haddad et al (2004) (LOE 5) reviewed various cooling techniques in terms of efficacy, availability, adverse effects, and mortality rates. They found that the prognosis of heat stroke in patients is directly related to the degree of hyperthermia and its duration and recommend that the most important feature in the treatment of heat stroke is rapid cooling as rapid cooling exploits the high conductance properties of water, which are 25 times that of air. According to their review, initiating cooling at time of collapse should be based on feasible field measures including ice or tepid water (1-16°C [33.8-6-.8°F]), which are readily available. Of the studies examined:

    1. Tap water immersion (12-16°C [53.6-60.8°F]) in heat stroke patients demonstrated an average cooling rate of .46-.34°C/min while ice-water immersion (1-5°C [33.8-41°F]) averaged .15-.23°C/min.

    2. In healthy subjects the average cooling rates for different applications were as follows from best to worst:

        1. Ice packs (whole body) @ .34°C/min (ice water @ .15-.30°C/min

        2. Tap water .04-.16°C/min; ice packs (local) .028-.087°C/min

        3. Evaporative @.027-.11°C/min.

    Once in the emergency department, management should be matched to the patient's age and medical background and include immersion in ice water (1-5 °C) or evaporative cooling. Casa et al (2007) suggest that the cooling rate of ice bags on the peripheral arteries (**about 0.03°C/min [0.05°F/min]),** is an example of an inferior cooling method because the area under the curve (*i.e.,* time vs. rectal temperature) is to large and increases the likelihood of tissue and organ injury. Therefore, an intervention providing a **cooling rate of less than 0.1°C/min (0.18°F/min**) when cooling begins immediately, and not less than **0.15°C/min (0.27°F/min**) if cooling is **delayed longer than 20-30 min after collapse is not recommend.**

4. Remove from the cooling modality when the rectal temperature reaches 39°C (102.2°F); then immediately transport to medical facility (or continue to monitor if an adequately staffed on-site medical facility is available). This would require the use of rectal thermometer.

5. Assure physician supervision after cooling is complete to monitor for sequelae, provide clearance for discharge from the hospital or medical tent, and guide (in conjunction with the athletic trainer) the return-to-participation process [8 as cited in Casa et al, 2007]

In Carter et al (2007) a case observation of on one individual with augmented hyperthermia revealed that an acute inflammatory response from an acute local infection response can augment the hyperthermia of exercise and possibly increase heat illness susceptibility. The case report also suggests that the 37 US Army heat stroke deaths were likely due to the medical status of the individual prior to the incident (i.e., medical records noted respiratory infections, influenza, streptococcus pneumonia, cardiovascular disease, hypertension, acute renal failure, and rhabdomyolysis), severity of the heat stroke involving multi-organ failure, and possibly delayed medical treatment (LOE 2b).

O'Connor et al (2007) describes and discusses the current guidelines used by United States military branches of the Army, Air Force, and Navy to return active duty soldiers, airman, sailors, and marines afflicted by a heat illness back to a full duty status. Each branch (Army and Air Force and Navy and Marine) of the military have their own guidelines for defining heat related illnesses, however, some units have their own specific guidelines. The U.S. Army's 82nd Airborne for examples places Severe Exertional Heat Illness (defined as heat stroke) or Rhabdomyolysis in what they refer to as group B. Patients categorized as group B are treated with rapid cooling (preferably with ice bath), followed by rapid evacuation (LOE 5).

In Robert's (2007) (LOE 5) review of exertional heat stroke in marathon runners an emphasis was placed on proper recognition and immediate on-site treatment. Recognition of exertional heat stroke during or following a marathon race can be fatal if not recognized and that immediate rapid cooling markedly improved outcomes. **He recommends an initial field treatment consisting of immediate and total-body cooling with ice-water tub immersion or through the use of rapidly rotating ice-water towels to the trunk, extremities and head, combined with ice packing in the neck, axillae and groin in order to produce an average cooling rate of 7°-8°C/hour (44.6-46.4°F/hour)** The more common field cooling techniques such as ice packs to the neck, axillae, and groin cool blood in the major vessels has a cooling rate less than half that of the immersion technique. Any combination of delayed recognition or cooling increases the potential for morbidity and mortality. For optimal outcomes, it is best to treat immediately with on-site whole body cooling if cardio espiratory status is 'stable' and then to transfer the runner for additional evaluation and care. Reports that the generally accepted endpoint for ice-water immersion cooling is 39°C (102.2°F), however, cooling to the 35-37°C (95-98.6°F) range may also have benefits such as reducing oxygen demands and cell metabolism, similar to the current recommendations for myocardial infarction.

Armstrong et al (2005) examined whether 3 levels of controlled caffeine consumption would affect fluid-electrolyte balance and renal function differently. Imbalances in fluid-electrolytes may increase an individual's risk of suffering from hyperthermia, including cramps, heat

exhaustion, and exertional heat stroke. The investigation of healthy, active males provided no evidence to support the notion that active individuals should refrain from consuming moderate daily levels of caffeine (i.e., 0 mg/d 226 mg/d; 452 mg/d). This investigation supports recent literature reviews cited in Armstrong et al that found no evidence of a detrimental caffeine-induced fluid-electrolyte imbalance (LOE 2a).

Smith and Wallis (2005) (LOE 5) reviewed different methods of reducing body core temperature in patients with exertional heatstroke. A literature search from 1966 to July 2003 using the databases Medline and Premedline, Embase, Evidence Based Medicine (EBM) reviews, SPORTDiscus, and cross referencing the bibliographies of relevant papers yielded 17 papers on cooling times or cooling rates in patients with heat illness (classic and exertional) or normal subjects who were subjected to heat stress. Table 5 is a summary table provided by Smith and Wallis of within subject crossover trials and case series including outcomes and key results.

Table 5. Summary Table Describing Case Series and Experimental Cooling Models by Smith and Wallis.

Author, Date	Patient Group and Interventions	Study Type	Outcomes	Key Findings
Wyndham et al,[20*] 1959	6 healthy volunteers exercised until T_{re} 40°C, cooled by immersion or evaporation	Within subject crossover trial	Cooling time to T_{re} 38.3°C, cooling rate (fall in T_c) over 60 min	Fastest mean cooling time 50 min, rate 0.07°C/min, with evaporative cooling
Weiner & Khogali,[21*] 1980	6 healthy volunteers exercised until T_{ty} 39.5°C, then cooled by immersion in 15°C water, cold air spray or warm air spray Spraying atomised water at 15°C over the patient combined with warm air (45–48°C) fanning. Air movement was ~0.5 m/sec	Within subject crossover trial	Reduction in T_{ty} of 2.0°C (to 37.5°C)	Cooling time 6.5 min (rate 0.31°C/min) with BCU, 18.4 min (rate 0.11°C/min) with immersion. **Immersion was superior.**
Kielblock et al,[22*] 1986	5 healthy volunteers exercised until T_{re} reached 2°C above baseline, then subjected to different methods of cooling (local ice-pack, total coverage, evaporative)	Within subject crossover trial	Reduction in T_{re} of 2.0°C (to baseline)	Mean cooling time 73.6 min (cold packs), 59.8 min (evaporative cooling); $p<0.01$. **Combined: 53.6 min or 0.04°C/min (no difference).** Local ice-pack application resulted in a significantly slower cooling rate (0.027 °C/min) than total coverage with ice packs (0.034 °C/min) or evaporative cooling (0.034 °C/min). The combination of local ice-packs with evaporative cooling yielded higher cooling rate than the latter (0.036 °C/min). Not neither rate meets the recommend rate of not less than .15°C/min.

Clapp et al,[23*] 2001	5 healthy volunteers exercised until T_{re} 38.8°C, then subjected to different methods of cooling (see results)	Within subject crossover trial	Cooling rate (fall in T_c) over 30 min	**Mean cooling rates 0.25°C/min (torso immersion)**, 0.16°C/min (hand and feet immersion) and 0.11°C/min (fan).
Mitchell et al,[24*] 2001	10 healthy volunteers exercised to preset limits, then cooled using 4 methods (see results)	Within subject crossover trial	Cooling rate (fall in T_c) during two 12 min cooling periods	Mean cooling rates 0.02–0.05°C/min, no difference between passive cooling and fan or spray assisted cooling.
Hart et al,[14*] 1982	28 **classical** heatstroke patients. All cooled by iced water immersion or ice massage if immersion not possible	Case series	Cooling time to T_{re}<102°F (38.89°C)	26 out of 28 cooled within 30 min, all within 45 min.
Vicario et al,[6*] 1986	39 **classic** heatstroke patients, cooled by various means depending on physician preference	Case series	Temperature reduction over first hour, mortality	69% cooled to T<38.9°C within 60 min. Mortality 21%, lower when cooled faster.
Graham et al,[15*] 1986	14 **classical** heatstroke patients, cooled by evaporative methods. Ice also used around torso	Case series	Cooling time to T_{re}<39.4°C, mortality	Cooling times ranged from 34 to 89 min, median 60 min. 1 patient died.
Al-Aska et al,[16*] 1987	25 **classical** heatstroke patients cooled with simplified cooling bed	Case series	Cooling time to T_{re}<39°C	Mean cooling time 40 min (rate 0.09°C/min) with simplified cooling bed.
Poulton & Walker,[17*] 1987	3 heatstroke patients (2 **exertional, 1 classical),** cooled by helicopter downdraft.	Case series	Cooling time to T<38.5°C	Mean cooling time 24 min, rate 0.10°C/min. combination of a large and powerful fan with a warm environment provide an efficient cooling method. However, believed to be very dangerous.
Costrini,[18*] 1990	27 patients with **exertional** heat illness, patients were immersed in ice water and their skin was concomitantly massaged vigorously until core temperature was decreased to 39°C or less	Case series	Cooling time to T_{re}<39°C, mortality	Mean cooling time 19.2 min or rate 0.15°C/min. Evidence for 100% human survival rate associated with cold/ice water immersion.
Horowitz,[19*] 1989	Heatstroke patient cooled by iced peritoneal lavage	Case report	Cooling rate. No end point given	Cooling rate 0.11°C/min during lavage cycle. Patient survived.

* Reference number in the Smith and Wallis article.

Haddad et al (2004) (LOE 5) reviewed various cooling techniques in terms of efficacy, availability, adverse effects, and mortality rates. They found the prognosis of heat stroke in patients is directly related to the degree of hyperthermia and its duration and recommend that the most important feature in the treatment of heat stroke is rapid cooling as rapid cooling exploits the high conductance properties of water, which are 25 times that of air. According to their review, initiating cooling at time of collapse should be based on feasible field measures including ice or tepid water (1-16°C), which are readily available. Of the studies examined tap water immersion (12-16°C) in heat stroke patients demonstrated an average cooling rate of .046-.34°C/min while ice-water immersion (1-5°C) averaged .15-.23°C/min. In healthy subjects the average cooling rates for different applications were as follows from best to worst: ice packs (whole body) @ .34 °C/min; ice water @ .15-.3°C/min; tap water .04-.16°C/min; ice packs (local) .028-.087°C/min; evaporative @.027-.11°C/min. Once in the emergency department, management should be matched to the patient's age and medical background and include immersion in ice water (1-5 °C) or evaporative cooling. Casa et al (2007) suggest that the cooling rate of ice bags on the peripheral arteries (about 0.03°C/min [0.05°F/min]), is an example of an inferior cooling method because the area under the curve (*i.e.,* time vs. rectal temperature) is to large and increases the likelihood of tissue and organ injury. Therefore, an intervention providing a cooling rate of less than 0.1°C/min (0.18°F/min) when cooling begins immediately, and not less than 0.15°C/min (0.27°F/min) if cooling is delayed longer than 20-30 min after collapse is not recommend.

Munro (2004) (LOE 5) in her review of heat related illness recommends that anyone believed to be suffering from heat stroke must be cooled as rapidly as possible in order to reduce body core temperature. If the core temperature is left untreated it will continue to rise until the victim becomes unconscious, and with increased risk of medical complications including respiratory distress, kidney failure, liver failure possibly death. This will be followed by admittance to hospital's intensive care unit where body temperature will be lowered to a safe level and abnormal fluid and electrolyte balance corrected.

In a public health practice article Weir (2002) examines the clinical management of heatstroke. The initial emphasis is placed on the ABCs of resuscitation followed by a rapid reduction in the core temperature to 40°C (104°F) as the two primary goals (LOE 6). Weir cites two possible methods of cooling the body, including evaporative cooling (use of a fans close to the completely undressed patient and sponging the skin or spraying it with tepid water) (Tintinalli, Kelen, & Stapczynski, 1999) (LOE 7) and immersion techniques (drenching the patient with sheets soaked in ice water or immersing the person in a tub of ice water, or both) (Gaffin, Gardner, & Flinn, 2000) (LOE 5).

In a study to determine the efficacy of ice-water immersion or cold-water immersion on rapidly cooling hyperthermic runners researchers found that cooling rates between the two therapies were nearly identical (Clements et al., 2002) (LOE 2a). No initial differences in cooling rates

were found when comparing ice-water immersion (5.15 +/- 0.20° C), cold-water immersion (14.03 +/- 0.28° C), and a mock immersion (no water, air temperature = 28.88 +/- 0.76 degrees C) at the start of immersion to 4 minutes, 4 to 8 minutes, and the start of immersion to 8 minutes. However, ice-water immersion and cold-water immersion cooling rates were similar (P >.05) to each other and greater (P <.05) than mock immersion at 8 to 12 minutes, the start of immersion to 10 minutes, and the start of immersion to every other time point thereafter. Rectal temperatures were similar (P >.05) between ice-water immersion and cold-water immersion at the completion of immersion and 15 minutes post-immersion, but ice-water immersion rectal temperatures were less (P <.05) than cold-water immersion at 6 and 10 minutes post-immersion.

A similar study (Proulx et al., 2003) (LOE 2a) examining the effects of water temperature on cooling efficiency on hyperthermia in healthy adults found the cooling rate was about two times greater during the 2° C water immersion compared with the 8, 14, and 20° C water immersions. Researchers found a significantly higher thermal gradient during a 2°C water immersion resulting in greater amounts of heat to dissipated at a faster rate during the 2°C water immersion compared with the other water immersion temperatures. It should be noted that shivering, which is thought to increase metabolic heat production, substantially reducing the rate the core temperature drops was seldom observed during the 2°C water immersion. Of the seven subjects, only one experienced shivering at 8 minutes, 2 at 13 minutes, and 1 at 14 minutes.

Clapp et al (2001) (LOE 2a) examined three core cooling techniques using subjects that exercised in the heated environmental chamber (WBGT = 39°C) until the onset of heat-strain (typically an additional 30-minutes). Subjects underwent each of three core cooling treatments in random order: 1) torso-only immersion in cool water (into a pool of cold water (10-12° C), 2) hands and feet only immersion in cool water only in cold water tubs (10-12° C), and 3) sit-in-shade directly on the treadmill with no radiant heating and with a 0.67 m/s (1.5 mph) breeze from a 91-cm diameter fan (Clapp, Bishop, Muir, & Walker, 2001). Significantly lower ($p<0.05$) rectal temperature readings Significantly lower (P <0.05) rectal temperature readings were seen in the torso-immersion trial compared to both the hand and feet immersion technique and the sit in shade technique. Torso immersion produced a significantly (P <0.05) greater rate of drop in rectal temperature reduction (0.25±0.10° C/min) than the hands and feet immersion technique (0.16±0.05° C/min) and the sit in the shade technique (0.1±0.04° C/min). At 10 minutes and 30 minutes the torso immersion had greater drops in rectal temperature (Table 6). Clapp et al. concluded that rectal temperatures can be reduced rapidly through the use of a cool water torso-immersion technique.

Table 6. Rectal Temperature Drops Initially, 10 and 30 minutes Post Immersion.

Group	Initially	10 minutes	30 minutes
Torso Immersion	0.25±0.10° C/min	0.63±0.41° C/min	1.2 ±0.46 °C/min
Hands and Feet Immersion	0.16±0.05° C/min	0.45±0.30° C/min	0.74 ±0.33°C/min
Shade	0.1±0.04° C/min	0.14±0.14° C/min	0.42±0.14 °C/min

In a review by Moran (2001) (LOE 5) stress indices (ie., Wet-Bulb Globe Temperature (WBGT), gender, age, fluid intake) should be used more readily by athletes, coaches and officials to prevent injury and improve safety conditions for competitors and participants in recreational activities to minimize the risk of heat related illnesses. Thus, the emphasis should be on prevention of heat related illnesses not treatment. He also suggests that appropriate fluid ingestion before and during activity can minimize dehydration and reduce the rate of increased core temperature (LOE 5). He recommends a fluid replacement policy of 0.5 to 1 quart/h for easy work, 0.75 to 1 quart/h for moderate work and 1 quart/h for hard work (As cited by Montain et al 1999) (LOE 2a). The range in the fluid volume is derived from the heat category and the time periods of the work-rest cycles. However, an individual should not exceed an hourly fluid intake of 1.5 quarts and a daily fluid intake of 12 quarts (As cited by Montain et al 1999) (LOE 2a).

Kare (2001) (LOE 5) reviewed the epidemiology, precipitating factors, clinical presentations, and various treatment modalities of hyperthermia and hypothermia in the older population. Initial management of a patient with heat stroke is consistent with Weir (2002) in that an initial evaluation must begin with assessment of the ABCs and removal for the poor environmental conditions. Patient should be administered high flow oxygen. Cardiac monitoring and pulse oximetry will also help provide the continuous status of the patient. Rapid cooling is also necessary and states that evaporative cooling to reduce the patient's core temperature is favored by physicians. Ice packs placed on the groin and axillae and cooling blankets may be used as adjuncts to help accelerate the cooling process. Cooling measures are then discontinued once a core temperature of 38.33°C to 38.89°C (101°F to 102°F) is reached in order to prevent overshoot hypothermia. In this age population Kare does not advocate the use of cold water immersion as a means of cooling the body. Kare states that this method has the disadvantages of limiting access to the patient, preventing defibrillation, and allows for the displacement of monitor leads (however first aiders will not be using monitors so this may be a mute point). However, in humid environment or when evaporative cooling is impossible cold water immersion may be indicated on a case by case basis.

Heat exhaustion in the elderly population has been described as being caused by water depletion (dehydration) and salt depletion (loss of electrolytes). In water depletion heat exhaustion, fluid replacement is inadequate, resulting in hypovolemia, while salt depletion heat exhaustion, water losses from sweating are replaced with free water. Treatment is aimed at adequate fluid resuscitation and electrolyte correction. These patients may be sent home once fluid deficits and electrolyte abnormalities are corrected (Kare & Shneiderman, 2001) (LOE 5).

In a review excerpt by Gaffin et al (2000) (LOE 5), authors reported the results of three studies examining cooling of heat stroke patients. They report one study where patients with classic heatstroke and those with exertional heatstroke cooled in ice-water cooling were cooled twice as rapid as compared to the evaporative spray. Furthermore evidence suggests that ice-water treatment in the clinic reduced rectal temperatures to less than 39°C (102.2 °F) in 10 to 40 minutes with no death or development of renal failure. However, when the evaporative spray

method was used for 18 patients with classic heatstroke, 26 to 300 minutes was required to reduce the core temperature to 38°C (100.4°F) with 2 patients dying, suggesting that cold-water or ice-water treatment cools patients with exertional heatstroke more rapidly, but the decision to induce cold water stress should be made on a case by case basis. And while cold-water immersion is available in most hospitals without requiring purchase of capital equipment, it may not be readily available in the field. Which is why a suspected victim of heat stroke is stripped down to shorts and T-shirts and if the rectal temperature is measured: if > 39.4°C (102.9°F), the patient is wet down, ice is packed around the groin/axillary areas, and the patient is immediately transported to the clinic on a stretcher.

An observational study describing the clinical features of patients admitted to an intensive care unit with near-fatal classic heat stroke found that lower the body core temperature to below 38-39° C (100.4°F-102.2°F) within 30 minutes of presentation improved survival rates (Dematte et al., 1998) (LOE 3a). This is consistent with another study which found that the mainstay of therapy heat stroke is rapid cooling, with a reduction of core temperature of at least 40°C (104°F) within 10 to 40 minutes (Costrini et al., 1979) (LOE 2a). **Thus, the goal of lowering the body's core temperature to less than 40°C (104°C) within 30 min should be the primary goal of managing any victim heat stroke, particularly exertional heat stroke** (LE Armstrong et al., 2007; D. J. Casa et al., 2006) (LOE 5). In treating exertional heat stroke, the adage "cool first, transport second" should guide immediate care if proper medical staff is on site (i.e., athletic trainer or team physician) (D. J. Casa et al., 2006). In addition to calling an ambulance when an extertional heat stroke victim has been identified, the medical staff should have an emergency plan in place to cool the athlete until the rectal temperature reaches 102.2°F (39°C; in the absence of any other emergent issues) before the athlete is transported to the nearest medical facility. Even if medical personnel are not present, cooling the victim is necessary until the ambulance arrives because rapid cooling is the key to surviving exertional heat stroke (D. J. Casa et al., 2006) (LOE 5).

Armstrong et al (1996) (LOE 1b) in a controlled study found in their examination of two cooling therapies for hyperthermic distance runners who had completed an 11.5-km summer foot race that ice water immersion cooled approximately twice as fast as air exposure. Twenty-one distance runners with a mean rectal temperature of 41.2 +/- 0.2° C were treated either by ice water immersion (1-3 ° C, n = 14) or by air exposure while wrapped in wet towels (24.4 ° C ambient, n = 7). **Cold water immersion** resulted in significantly different ($P < .005$) pre-therapy to post-therapy changes in rectal temperature (-3.0 +/- 0.3 vs. -1.4 +/- 0.3 ° C) and mean cooling rate (0.20 +/- 0.02 vs. 0.11 +/- 0.02 ° C/min) when compared to the wet towels.

Bross (1994) agrees the immediate goal of treatment for a victim of heatstroke is to achieve rapid cooling to a core temperature of 39°C (102°F). In the field he advocates the removal all of unnecessary clothing, spraying the body with lukewarm water, and augmenting airflow over the

patient during transport with a fan, administration of oxygen to patients in respiratory distress (LOE 5).

According to a review paper by Bross (1994) individuals suffering from heat exhaustion should also be removed from the warm environment and taken to a cool environment immediately. Excess clothing should be removed and the body should be sponged with tap water. He suggests that appropriate fluid ingestion be initiated if tolerated with cool water given at a rate of 1 L per hour over several hours. In severe cases consider immediate transportation. Cooling may be included as part of the treatment and is accomplished by spraying lukewarm water on the patient and then increasing convection by placing the patient in front of fans (LOE 5).

A case-control series of 14 patients with non-exertional heatstroke were evaluated in a general hospital during the summer of 1980. They were managed according to a prospectively devised protocol designed to effect heat dissipation primarily via convection and evaporation rather than by conduction. Patients were disrobed and then were subjected to spraying or sponging cool water (approximately 32°C [89.6°F]). The time from entry into the emergency room to the first recorded rectal temperature of less than 103°F (39.4 degrees C) ranged from 34 to 89 minutes (median, 60 minutes). One patient died and none had residual neurologic deficits (Graham et al., 1986) (LOE 2c).

In a prospective controlled study to compare the efficiency of instant ice packs over the large vessels of the neck, axillae and groin with total body covering and with evaporative cooling (Kielblock, Van Rensburg, & Franz, 1986). Results suggest that covering the whole body with instant cold packs plus induced evaporative cooling produced higher cooling rates in bringing about a 2° C reduction in rectal temperature than those achieved by the strategic placement of instant cold packs (0.034 °C/min and 0.034 °C/min respectively) (LOE 2a) in areas of high blood flow.

Overall Recommendations including any Standards, Guidelines, or Option:

Summary:

Heat Cramps

The current ARC first aid procedures for the identification and management of heat cramps appear to be consistent with the current literature. When discussing recognition, consider the possible inclusion of more signs and symptoms of heat cramps may be warranted including: decreased water intake fatigue, and thirst. When discussing care, consider adding the application of cryotherapy (Level III) and consuming a well balanced diet.

Heat Exhaustion

The current first aid procedures for the identification and management of heat exhaustion appear to be consistent with the current literature, however, a better distinction needs to be made between heat exhaustion and heat stroke within the text as well the mechanism for the development of heat exhaustion. For example, in *Responding to Emergencies* heat related, particularly heat exhaustion and heat stroke are referred to as "early' and "late" heat illness. T his is not common terminology used in the literature. Furthermore, more emphasis should be placed on early recognition and prevention. When discussing recognition, consider the possible inclusion of more signs and symptoms of heat exhaustion such as those seen with heat cramps, as well as rapid, weak pulse, shallow breathing, early AMS, hypotension. When discussing care, consider adding, rest, removal from the environment to a cooler environment, using a fan to

Heat Stroke

The current first aid procedures for the identification and management of heat stoke appears inadequately discussed in the current format. Given the prevalence of this condition and the potential for harm including, organ failure and death the current writing would appear inadequate. Consider providing greater detail of the etiology, including the affects of ambient temperature and humidity on the core temperature and defining the difference between exertional and classic heat stroke. When discussing recognition, consider the possible inclusion of more signs and symptoms of heat stroke (from above) may be warranted including that while warm/hot, red, dry skin may be apparent, it would be much more common for a victim of exertional heat stroke to be sweating at the time of collapse. The recommended care guidelines need to reinforce prompt and immediate referral to EMS. While waiting for the arrival of EMS it should be stressed that immediate cooling of the body should begin using a cooling modality that provides an effective cooling rate. Cold water immersion has been proven to have the best cooling and survival rates. Whenever feasible, immersion in a kiddie pool, tub, etc. is recommended. When this is not possible it is recommended to rotate cold/wet towels over the patient's body. As with heat exhaustion, emphasis should be placed on prevention which includes decreasing intensity of exercise during more extreme environmental conditions as well

as maintaining hydration status, avoiding the hottest times of the day, minimizing clothing and equipment, seeking shade, taking frequent breaks, and gradually increasing exposure time to the heat (heat acclimatization).

Recommendations and Strength (using table below):

Standards: N/A

Guidelines:

For a person with suspected exertional heat stroke (a core temperature at or above 40° C (104° F) and central nervous system (CNS) dysfunction occurring during exercise in heat) (Level II) immediately initiate EMS and begin rapid cooling of the victim (try to begin aggressive cooling as soon as possible, the amount of time the patient is hyperthermic is the key determinant of outcome) till core temperature returns to 38.8°C or 102°F (Level II). Rapid cooling methods include cold water immersion (~5°-15°C or ~40°-60°F Degrees) (Level II) ice water doused towels over the entire body (Level II).

For rescuers working without equipment to measure core temperature, a victim exhibiting signs and symptoms of heat stroke (i.e., heat exhaustion plus change in normal mental status that occurs during exercise in heat) immediately initiate EMS and begin rapid cooling (i.e,, cold water immersion, ice water dowsed) for 20 minutes or until baseline mental status is achieved. On-site cooling is best to optimize outcome.

Options:

The strength of all recommendations and conclusions is related to the scientific evidence upon which they are based. All recommendations therefore derive from critical review of the available medical literature including formal clinical trials and studies and the strength of their design, standard reference material, textbooks, and expert opinion. All recommendations are weighted based upon the source and strength of the scientific evidence and are classified into one of three groups - Standards, Guidelines, or Options.

Treatment Standards represent the strongest recommendations and have a high degree of clinical certainty. These recommendations result from strong evidence obtained from well designed, prospective, randomized controlled studies.

Treatment Guidelines provide a moderate degree of clinical certainty and are based on less robust evidence such as non-randomized cohort studies, case-control studies, or retrospective observational studies.

Treatment Options result from all other evidence, publications, expert opinion, etc. and are the least compelling in terms of scientific evidence.

Class	Description	Implication	Level of Evidence
I	Convincingly justifiable on scientific evidence alone.	Usually supports Standard	One or more Level 1 studies are present (with rare exceptions). Study results consistently positive and compelling
II	Reasonably justifiable by scientific evidence and strongly supported by expert opinion.	Usually supports Guideline but if volume of evidence is great enough and support from expert opinions is clear may support standard	Most evidence is supportive of guideline. Level 1 studies are absent, or inconsistent, or lack power. Generally higher levels of evidence. Results are consistently supportive of guideline.
III	Adequate scientific evidence is lacking but widely supported by available data and expert opinion. Based on	Usually supports Option.	Generally lower or intermediate levels of evidence. Generally, but not consistently results are supportive of opinion.
IV	No convincing scientific evidence available but supported by rational conjecture, expert opinion and/or non peer-reviewed publications	Usually does not support standard, guideline, or option. Statement may still me made which presents what data and opinion exists. In some cases and in conjunction with rational conjecture may support option.	Minimal evidence is available. Studies may be in progress. Results inconsistent, or contradictory.

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