Exertional Heat Stroke: New Concepts Regarding Cause and Care

Douglas J. Casa, PhD, ATC, FACSM, FNATA1,2; Lawrence E. Armstrong, PhD, FACSM1,2; Glen P. Kenny, PhD2,3; Francis G. O’Connor, MD, MPH, FACSM2,4; and Robert A. Huggins, MEd, ATC1,2

Abstract
When athletes, warfighters, and laborers perform intense exercise in the heat, the risk of exertional heat stroke (EHS) is ever present. The recent data regarding the fatalities due to EHS within the confines of organized American sport are not promising: during the past 35 years, the highest number of deaths in a 5-year period occurred from 2005 to 2009. This reminds us that, regardless of the advancements of knowledge in the area of EHS prevention, recognition, and treatment, knowledge has not been translated into practice. This article addresses important issues related to EHS cause and care. We focus on the predisposing factors, errors in care, physiology of cold water immersion, and return-to-play or duty considerations.

Introduction
Throughout the sports and military medicine literature, much evidence indicates that the common signs and symptoms of exertional heat stroke (EHS) include a core temperature usually >40°C (104°F) coupled with central nervous system alteration (3,13,17,49). Furthermore, much evidence indicates that death related to EHS is preventable through immediate recognition using a rectal thermometer and rapid treatment via cold water immersion (CWI). Even with this knowledge, the prevalence of EHS in competitive sport, military, and industrial population continues to increase (3,9,13,17,49). Within the last 5 years, the incidence of EHS deaths in American sports remains high when compared to the last 35 years, with the incidence from 2005 to 2009 being the highest ever recorded (49). This indicates that recent findings are not being applied in clinical practice and that a renewed emphasis is needed across the sports medicine community. It is the purpose of this article to review the predisposing factors, errors in care, physiology of CWI, and return-to-play or duty considerations as they relate to EHS.

Predisposing Factors
The discipline of labor, physical training, or sporting events may thrust military personnel and athletes into tasks that exceed exercise capacity or heat acclimatization status. These individuals have an increased risk in the presence of environmental, organizational, and individual factors that predispose humans to EHS (Table 1). Rav-Acha et al. (40) made some important observations of 130 EHS cases, of which 6 were fatal. Predisposing factors included low level of physical fitness, sleep deprivation, high ambient temperature, intense solar radiation, exercise intensity not matched with physical fitness, ineffective or absent medical triage, and disregard for organizational safety regulations. Clearly, the primary predisposing factor of EHS is complex and may vary from case to case, but hyperthermia is always the common denominator.

New concepts currently being investigated in the realm of EHS, found prominently in recent literature, are worthy of further research:

1. Advancements in the use of gene chip technology have enabled the identification of approximately 700 genes that are activated or suppressed by exercise heat stress in patients who had prior EHS (47). It is not known whether there is an upward or downward regulation in response to exercise heat stress, but perhaps if warfighters or athletes undergo genome screening, the predisposing factors for EHS may be identified by comparing normal individuals to patients who had prior EHS.

2. Various medications negatively affect heat dissipation and exercise performance by reducing sweat production (e.g., antihistamines, anticholinergicgs), altering
Table 1.

Predisposing factors for exertional heatstroke, as published by multiple authors.

<table>
<thead>
<tr>
<th>Predisposing Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever, illness, gastroenteritis, diarrhea, vomiting (3, 7, 10, 25, 40, 46)</td>
</tr>
<tr>
<td>Anhidrosis, sunburn, dehydration (3, 7, 10, 25, 40, 46)</td>
</tr>
<tr>
<td>Medications, drug abuse (7, 10, 28, 40, 46)</td>
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<tr>
<td>Sleep loss (7, 10, 25, 30, 40, 46)</td>
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<tr>
<td>Advanced age (7, 10, 25, 40, 46)</td>
</tr>
<tr>
<td>Excessive alcohol use (7, 10, 25, 45)</td>
</tr>
<tr>
<td>Lack of heat acclimatization (3, 7, 40, 46)</td>
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<tr>
<td>Sedentary lifestyle (7, 10, 25, 46)</td>
</tr>
<tr>
<td>Overweight/obesity (10, 25, 39, 42)</td>
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<tr>
<td>Cardiovascular dysfunction (7, 10, 25, 46)</td>
</tr>
<tr>
<td>Hypokalemia (3, 10, 46)</td>
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</table>

skin blood flow (e.g., calcium channel blockers, female reproductive hormones, capsaiain), reducing cardiac contractility (e.g., β-adrenergic or calcium channel blockers), or increasing heat production and/or elevating the hypothalamic set point (e.g., amphetamines, salicylates in large doses) (41,52). Similarly, nutritional supplements may contain compounds that negatively influence thermoregulation and physiological function depending on the dose, clothing worn by the individual, exercise intensity or duration, and environmental conditions. The consumption of herbal products (e.g., bitter orange) and prescription medications (e.g., ephedrine, ecstasy) concurrent with exercise in the heat deserves further study.

3. An investigation involving patients who had prior EHS identified sleep deprivation as a common predisposing factor. Of 10 warfighters, 7 experienced sleep deprivation before their EHS episode (40). The aforementioned review (40) also reported a high incidence of sleep deprivation among fatalities. Although sleep deprivation is recognized to decrease skin blood flow and sweat rate during exercise at a given body temperature (48), little else is known about how it might interact with EHS. Another possible suggestion is that sleep deprivation may cause changes in cortisol level or decrease in growth hormone, which may play a role in temperature regulation (30).

4. The interactions between clothing and body morphology appear to be more complex than previously recognized. In a series of controlled, randomized experiments (4), experienced American football players (body mass = 117.4 kg, body fat = 30.1%) performed 70 min of exercise in a hot (33°C (91.4°F)) environment while wearing (a) shorts with no shirt, (b) a full American football uniform, or (c) a partial uniform without helmet and shoulder pads. Interestingly, a strong positive correlation ($R^2 = 0.71, P = 0.005$) existed between lean body mass and the increase in rectal temperature when subjects wore the full uniform but not the partial-clothing ensemble. Further, a strong negative correlation existed between total fat mass (kg) and treadmill exercise time to the point of volitional exhaustion when subjects wore shorts ($R^2 = 0.90, P = 0.00005$) and the partial uniform ($R^2 = 0.69, P = 0.005$). These findings are consistent with observations of U.S. Army warfighters (8), for whom the risk of heat illness and the use of outpatient facilities were significantly higher among male recruits with excess body fat.

5. Dehydration has consistently been shown to exacerbate increases in deep body temperature primarily due to the reduced blood volume coupled with the concomitant demand for blood in both the exercising muscles and skin. Reduced blood perfusion of the skin results in decreased evaporation, which is the body’s most effective heat loss mechanism. Without the balance between heat loss and heat gain, the deep body temperature continues to rise. Increases in deep body temperature due to dehydration have been noted especially during exercise in the heat in laboratory settings. In addition, despite some recent argument that this relationship may not exist in field settings, a recent study has shown the same influence of dehydration in a field setting (20). It seems the influence of free control of pace may have eliminated the effect in previous field setting studies (i.e., a more dehydrated person could decrease intensity or a better hydrated person could increase intensity), but when intensity is controlled (as is the case with many coach- and supervisor-induced activities), the role of hydration in body temperature is ever present and consistent with laboratory findings: approximately a 0.2°C (0.36°F) increase for every 1% deficit in body weight (11,34,44).

**Why They Die: Errors in Care**

The reality of EHS medical care within the confines of organized sport and military training scenarios is that no one should die if proper, prompt, and aggressive care begins within 10 min of collapse (9,13,17,19,49). Unfortunately, many athletes, warfighters, and laborers who sustain EHS have fatal outcomes. A review of the causes of death provides a window into the common errors in care. With an understanding of these primal concepts, we can work toward a medical delivery system for EHS that eliminates such common errors.

**Inaccurate Temperature Assessment or Misdiagnosis**

The diagnosis of EHS usually follows the presentation of two hallmark criteria: extreme hyperthermia (usually >40.0°C to 40.6°C (104°F to 105°F)) at the time of collapse, and CNS dysfunction, as characterized by altered behavior (not necessarily a complete loss of consciousness, especially
Initially) (3,9,13,17). Given that lucid intervals of varying degrees may be part of an acute EHS episode, it is imperative to accurately determine deep body temperature as soon as possible after the EHS presents (13,17). To properly assess temperature in a person who has been performing intense exercise in the heat, the temperature measurement must be taken from within the body, and it must not be affected by external influences such as sweat, fluids, wind, clothing, etc. The literature has clearly shown that axillary, tympanic (aural), temporal, oral, and skin measurements are not valid or reliable predictors of deep body temperature (9,13,16,17,26,42,49). In many cases, these temperatures may provide a false sense of security (because they nearly always under predict temperature) regarding the level of hyperthermia and therefore the severity of the condition (16,26). Medical staff should rely on rectal temperature for a clear picture of the degree of acute hyperthermia (3,9,13,17,49). Although other modes of temperature assessment (i.e., esophageal, pulmonary artery) are effective in tracking rapid changes in core temperature, they are not practical in a field assessment scenario for EHS (15). Rectal temperature assessment has been part of the successful EHS care at the Falmouth Road Race, Peachtree Road Race, Quantico Naval Base, Parris Island Marine Base, and Marine Corps Marathon, among many other sites (B. Cooper, personal communication; Casa DJ et al., unpublished manuscript/observations, 2012) (1,23,37). In addition, the clinical presentation of EHS can be very similar to that of other medical conditions (e.g., exertional sickling, hypocalcemia, heat exhaustion [HE], asthma, head injury), but a prompt and accurate rectal temperature assessment can immediately rule in EHS (3,14).

No Care or Treatment Delayed

In many circumstances, especially when appropriate medical personnel (i.e., athletic trainers, team physicians) are not present, EHS victims are simply allowed to lie on the side of the playing field, locker room, or gymnasium. Those supervising assume that the condition is not as serious as it really is and that the athlete will recover on his or her own with rest. Failure to recognize the presence of EHS has been a common problem in high school sports. This scenario demonstrates two important needs: having qualified medical personnel on-site and not placing coaches in charge of medical care (3,14).

Inefficient Cooling Modality

Even if EHS is promptly recognized at the time of the incident, an athlete can still succumb if extreme hyperthermia is not rapidly reduced. The most important determinant of EHS outcome is the amount of time the patient is above some hypothetical critical threshold for cell damage (18,27). The exact initial temperature (probably in the range of 40.6°C (105°F) to 43°C (109.4°F)) is not the most important item, but reducing temperature to less than 40.0°C (104°F) in less than 30 min is critical. Experiences from various military installations (Quantico, Parris Island, Fort Benning) and medical tents (Falmouth Road Race, Peachtree Road Race, Marine Corps Marathon, Boston Marathon) have shown that if temperature is reduced to less than 40.0°C (104°F) within 30 min after collapse, the fatality rate is close to (or actually) zero (B. Cooper, personal communication; Casa DJ et al., unpublished manuscript/observations, 2012) (1,3,19,23,37). We have located no record of any EHS victim who died when this standard was met (19). The “golden hour” common to medicine has become the golden half-hour in the realm of EHS.

Thus, two very important concepts are introduced: 1) the cooling modality needs to be immediately accessible and 2) the cooling modality must cool rapidly enough to meet the time criterion. The gold standard for rapid cooling is CWI. This includes immersion in very cold (1.7°C to 14.0°C (35°F to 57°F)) or ice water over as much of the body as possible (except the head), constant and aggressive stirring of the water to keep bringing cold water to the skin’s surface, and an ice or wet towel wrapped around the top of the head (19). Cooling of this type (discussed in more detail below) has yielded significantly high cooling rates, lowering temperatures of even 43.3°C (110°F) to less than 40.0°C (104°F) in <20 min (19). Given that bringing the athlete to the tank and doing the initial assessment (checking airway, breathing, and circulation, placing rectal probe) takes only minutes, the critical 30-min timeline will be met. A readily portable alternative to CWI for scenarios of remote care (or when a tub is not accessible or prepared) is the rotation of ice or wet towels (3). Approximately 12 towels can be kept in a tub of ice water, and 6 towels can be placed on the patient who has EHS. After a few minutes, the six towels from the tub can be placed on the patient while the others are recooled. Two items should still be remembered when using this method: 1) if possible, the ice- or wet-towel system should be used until an immersion tub is ready or until transport to an immersion tub, and 2) emergency medical technicians should continue aggressive cooling during transport (instead of simply placing ice bags on peripheral arteries).

Immediate Transport (and/or Waiting for Transport)

One of the more controversial aspects of EHS care is the concept of “cool first, transport second” (13,16,19). This motto is based on the premise that the most important component of EHS care is the rapid cooling that should take place as soon as possible after the incident occurs (3,18). Waiting for an ambulance to arrive and transport the patient to the hospital can result in a delay of at least 45 min (even in optimal conditions: 5 min to determine the condition, 10 min for an ambulance to arrive, 10 min on-site, 10 min to the hospital, 10 min at the hospital before aggressive cooling begins) during which aggressive cooling could have taken place. Instead, we encourage taking 20 min to use CWI for rapid cooling of the patient on-site and then immediately transporting to the hospital (13,19). Transport occurs when temperature is (38.9°C to 40.0°C (102°F to 104°F)). This recommendation does not discount the potential for outstanding care at the hospital but rather prioritizes appropriate care in the first 30 min after collapse, which is likely more critical to survival.

Rapid Return to Play or Duty

When an individual succumbs to heat illness, no matter the severity, a few items should be addressed before returning to normal daily work or the exercise training regimen. First, the cause of the condition should be determined. If it is...
an item (e.g., predisposing factor, situational characteristic) that can be altered (e.g., heat acclimatization, appropriate intensity of exercise based on fitness, illness, sleep status, hydration status), this should be done before return (12,31). Second, sufficient time needs to pass to allow for full recovery from the incident itself, which varies based on the severity of the condition (described in more detail below) (12,31). Third, when the athlete returns to activity, his or her teammates or fellow warfighters will likely be better conditioned and acclimatized and more skill ready than when the original incident occurred, which may force the patient who has EHS to work harder to keep up with his or her compatriots (12). This is an important factor to consider in the rehabilitation process. Fourth, medical care may require further confirmation to determine whether the athlete or warfighter may return to activity or duty. One possible tool may be heat tolerance testing to examine the athlete or warfighter’s ability to mitigate the rise of internal body temperature and cardiovascular function (35).

**Physiology of Cooling Modalities for Treatment of EHS**

Under a hyperthermic state, effective heat dissipation is facilitated by elevated levels of skin perfusion and sweating; however, whole-body heat loss can be severely compromised in individuals experiencing EHS. Under these conditions, cooling treatments that enhance heat dissipation by increasing conductive heat transfer and/or evaporative heat loss are critical for the survival of the EHS victim. Current empirical evidence clearly demonstrates that CWI is a superior cooling modality, with ice water immersion (IWI) providing some of the highest core temperature cooling rates — an important element in surviving EHS (3,9,19,23,38).

To date, there remains some disagreement regarding the optimal water temperature that should be used. This is probably the result of large variations in cooling rates and water temperatures in previous studies. Differences in subjects’ physical characteristics (29) and immersion conditions (i.e., water circulation, full- vs partial-body immersion) (5,21) may, in part, explain variations in cooling rates. Despite these variations, only CWI or IWI has provided consistently superior core cooling rates (0.12 to 0.35°C/min⁻¹) (3,9,19,23,32,38) (Table 2) compared with other cooling modalities such as ice-wet towels (0.11°C/min⁻¹), ice packs over major arteries (0.028°C/min⁻¹), or fanning (0.02°C/min⁻¹) (32). For a more comprehensive discussion of the different methods of cooling, the reader is referred to a review by McDermott et al. (32).

The study by Proulx et al. (39) is the only study comparing cooling rates for a large range of water temperatures (i.e., 2°C to 20°C (35.6°F to 68.0°F)) in subjects rendered hyperthermic by prolonged exercise in the heat (end-exercise rectal temperature of 40°C). After exercise, the subjects were immersed in a circulated water bath controlled at 2°C, 8°C, 14°C, or 20°C until rectal temperature returned to 37.5°C.

**Table 2.**

Mean cooling rates using water immersion.

<table>
<thead>
<tr>
<th>Type of Immersion</th>
<th>Author</th>
<th>Water Temperature</th>
<th>Cooling Rate (°C/min⁻¹)</th>
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<tbody>
<tr>
<td>IWI</td>
<td>Lemire et al. (29)</td>
<td>2°C (35.6°F)</td>
<td>males$^a$ 0.12</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2°C (35.6°F), females$^a$ 0.22</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Proulx et al. (38)</td>
<td>2°C (35.6°F)</td>
<td>0.35</td>
</tr>
<tr>
<td></td>
<td>Costrini$^{a,b}$ (23)</td>
<td>1°C to 3°C (33.8°F to 37.4°F)</td>
<td>0.15</td>
</tr>
<tr>
<td></td>
<td>Armstrong et al. (5)</td>
<td>1°C to 3°C (33.8°F to 37.4°F)</td>
<td>0.20</td>
</tr>
<tr>
<td></td>
<td>Clements et al. (22)</td>
<td>5°C (41.0°F)</td>
<td>0.16</td>
</tr>
<tr>
<td>CWI</td>
<td>Lemire et al. (29)</td>
<td>8°C (46.4°F), low body fat (13%)</td>
<td>0.23</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8°C (46.4°F), high body fat (22%)</td>
<td>0.20</td>
</tr>
<tr>
<td></td>
<td>Proulx et al. (38)</td>
<td>8°C (46.4°F)</td>
<td>0.19</td>
</tr>
<tr>
<td></td>
<td>Clapp et al. (21)</td>
<td>10°C to 12°C (50.0°F to 53.6°F), torso only</td>
<td>0.25</td>
</tr>
<tr>
<td></td>
<td></td>
<td>10°C to 12°C (50.0°F to 53.6°F), limbs only</td>
<td>0.16</td>
</tr>
<tr>
<td></td>
<td>Kielblock (28)</td>
<td>12°C (53.6°F)</td>
<td>0.26</td>
</tr>
<tr>
<td></td>
<td>Taylor et al. (50)</td>
<td>14°C (57.2°F)</td>
<td>0.18</td>
</tr>
<tr>
<td></td>
<td>Proulx et al. (38)</td>
<td>14°C (57.2°F)</td>
<td>0.15</td>
</tr>
<tr>
<td></td>
<td>Clements et al. (22)</td>
<td>14°C (57.2°F)</td>
<td>0.16</td>
</tr>
<tr>
<td></td>
<td>Wyndham et al. (53)</td>
<td>14.4°C (57.9°C)</td>
<td>0.04</td>
</tr>
<tr>
<td>Temperate-water immersion</td>
<td>Proulx et al. (38)</td>
<td>20°C (68.0°F)</td>
<td>0.19</td>
</tr>
<tr>
<td></td>
<td>Taylor et al. (50)</td>
<td>26°C (78.8°F)</td>
<td>0.10</td>
</tr>
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</table>

$^a$Water immersion combined with skin massage.
$^b$Data obtained during field treatment.
CWI = cold water immersion; IWI = ice water immersion.
Rectal temperature cooling rate during immersion in water of 8°C (46.4°F), 14°C (57.2°F), and 20°C (68.0°F) averaged 0.15 to 0.19°C·min⁻¹. These values were consistent with the findings of Clements et al. (22), who reported similar rates of cooling (0.16°C·min⁻¹) during a 12-min CWI (14°C (57.2°F)) immersion. Noteworthy, Clements et al. (22) showed that 5°C (41.0°F) IWI also yielded similar rates of cooling (i.e., 0.16°C·min⁻¹). In contrast, however, Proulx et al. (38) showed that 2°C (35.6°F) IWI produced a rectal temperature cooling rate approximately twice that of warmer ≥8°C (46.4°F) water temperatures (i.e., 0.35°C·min⁻¹ in 2°C (35.6°F) IWI vs 0.19, 0.15, 0.19°C·min⁻¹ in 8°C, 14°C, and 20°C water, respectively).

The mean rectal temperature cooling rate obtained during 2°C (35.6°F) IWI (0.35°C·min⁻¹, 0.63°F·min⁻¹) by Proulx et al. (38) exceeded rates reported by Armstrong et al. (3) (0.20°C·min⁻¹) and Costrini et al. (24) (0.15°C·min⁻¹) (also measured during IWI at 1°C to 3°C (33.8°F to 37.4°F)) by 1.8- and 2.3-fold, respectively. This can be explained by some important differences between the studies. First, the latter two studies were performed in the field and involved distance runners (5) and military personnel (23) experiencing EHS, with rectal temperatures exceeding 41°C (105.8°F). Further, in the case of Armstrong et al. (5), the patients only had their torsos and upper thighs in the water, thereby limiting the potential for heat loss. Clapp et al. (21) showed that immersion of the torso only (lying supine in a small wading pool with legs and arms out) in 10°C to 12°C (50.0°F to 53.6°F) water resulted in a rectal temperature cooling rate of 0.25°C·min⁻¹ compared with only 0.16°C·min⁻¹ for leg and arm immersion only (seated upright with arm and leg only in a container filled with water). Water circulation is also an important consideration. Convection can influence the rate at which an individual cools, by dispersing the boundary layer of water adjacent to the skin and thereby maintaining the thermal gradient essential for heat dissipation. A slower cooling rate would therefore be expected in still water. For example, Wyndham et al. (53) reported a cooling rate of only 0.04°C·min⁻¹ in individuals immersed in a noncirculated water bath of 14°C (57.2°F), whereas Proulx et al. (38) measured a cooling rate of 0.15°C·min⁻¹ for the same immersion temperature with circulated water.

The misconception that CWI may cause peripheral vasoconstriction and intense shivering, thereby impeding cooling efficiency and affecting patient comfort, has some experts advocating the use of temperate-water (26°C (78.8°F)) immersion as providing a result equivalent to that of IWI or CWI (18,50). Recent evidence by Proulx et al. (38) clearly suggests otherwise. They reported that the temperature gradient between the skin surface and the water at the end of a 2°C (35.6°F) IWI was 1.8, 2.5, and 2.6 times greater than during the 8°C (46.4°F), 14°C (57.2°F), and 20°C (68.0°F) water immersions, respectively. We interpret these findings to mean that the large temperature gradient between the skin and ice water eliminates the need for elevated skin perfusion (5). The short duration of exposure during IWI also minimizes the effect of increased heat production via shivering thermogenesis. Shivering was seldom observed when hyperthermic individuals were immersed in 2°C (35.6°F) ice water (29,38). Finally, although subject comfort should not be the basis for determining the effectiveness of a treatment strategy in a medical emergency, Proulx et al. (38) reported that 2°C (35.6°F) IWI was no more painful or uncomfortable for the subjects than the warmer water immersion temperatures (i.e., ≥8°C (46.4°F)). Moreover, no cold-shock response (i.e., potentially lethal responses including hyperventilation, cardiac arrhythmias, elevated blood pressure, reduced cerebral blood flow, etc., that can occur with sudden immersion in cold water) (51) was observed.

Recently, Taylor et al. (50) argued that rapid and effective heat loss in the absence of an elevated skin-wa ter gradient can still be achieved using temperate-water immersion (26°C (78.8°F)) while avoiding the “cold-shock” responses (43) associated with IWI (18). Their study compared core cooling rates in an individual rendered hyperthermic by exercise in the heat while wearing a water-perfused suit circulated with 40°C water during subsequent exposure to cool ambient air conditions (20°C to 22°C (68°F to 71.6°F)), CWI (14°C (57.2°F)), and temperate-water immersion (26°C (78.8°F)). They suggested that, relative to IWI, immersion in temperate water (26°C (78.8°F)) reduces skin vasoconstriction (i.e., greater level of skin perfusion relative to IWI) sufficiently to maintain heat extraction and lower core temperature to safe levels at a similar rate (i.e., 2.16 and 2.99 min to bring esophageal temperature to 37.5°C (99.5°F) for 14°C and 26°C water immersion, respectively) (50). Based on actual skin temperature measurements (cold = 21.2°C, temperate = 26.8°C) and the assumption that skin perfusion achieves minimal and maximal levels at 10°C and 42°C, the authors surmised that circulatory convective heat transfer to the skin was 1.15-fold greater during the 26°C water immersion as compared to IWI. As such, they concluded that the similarity in the rate of esophageal temperature decay measured during 14°C (0.88°C·min⁻¹) and 26°C (0.71°C·min⁻¹) water immersion was due to the less powerful peripheral vasoconstrictor responses, resulting in physiologically and clinically insignificant differences in the rate of heat extraction in hyperthermic individuals between trials. However, they failed to consider that, while esophageal temperature dropped to 37.5°C (99.5°F) with a shorter immersion time, significant residual body heat storage (~50% of heat accumulated during exercise) likely remained in organs, with a corresponding elevated rectal temperature of ~39°C to 39.5°C (102.2°F to 103.1°F) (38). Rectal temperature cooling rate was only 0.10°C·min⁻¹ during 26°C (78.8°F) water immersion, at least three times slower than the 0.35°C·min⁻¹ measured by Proulx et al. (38) during 2°C (35.6°F) IWI. Moreover, CWI can minimize variations in cooling rate caused by differences in physical characteristics compared with warmer water temperatures (29).

The most important element in the treatment of EHS is rapid cooling. Current empirical evidence clearly demonstrates that CWI is a superior cooling modality, with IWI providing some of the highest core temperature cooling rates — an important element in surviving EHS. By adhering to the established cooling limits for CWI (i.e., exit rectal temperature of 38.6°C (101.5°F)) during immersion in water <10°C (50.0°F) (39), safe and effective treatment for hyperthermic individuals can be provided.
Return to Play/Duty after EHS

Return-to-play (RTP) decision making can be a complex and demanding process and is arguably the most challenging component of athlete injury management (2). Although the final decision is most commonly left to the providing physician, the assessments frequently require the input of and execution by the athletic trainer, physical therapist, coach, and family members, as well as the athlete. An American College of Sports Medicine (ACSM) guideline on RTP identified several key considerations to assist in safely returning athletes to activity (2):

- Status of anatomical and functional healing;
- Status of recovery from acute illness and associated sequelae;
- Status of chronic injury or illness;
- Whether the athlete poses an undue risk to the safety of other participants;
- Restoration of sport-specific skills;
- Psychosocial readiness;
- Ability to perform safely with equipment modification, bracing, and orthoses;
- Compliance with applicable federal, state, local, school, and governing body regulations.

The RTP decision making process ultimately requires a fundamental understanding of both the pathophysiology of the underlying disorder and the recovery of affected tissues, organs, and/or body systems. EHS RTP is especially challenging because of our incomplete understanding of the pathophysiological processes involved in the development of and recovery from this disorder (15,31). Current research indicates that most individuals recover completely from EHS within a few weeks; indeed, this occurs in most patients when treated promptly and cooled aggressively (i.e., IWI) (6,15,31). However, while most athletes and warfighters with EHS will recover and successfully return to play/duty, some EHS victims experience long-term complications that may include multisystem organ (liver, kidney, and muscle) and neurologic damage as well as reduced exercise capacity and heat intolerance (15,33,43,45).

Despite the frequency of EHS, and the associated morbidity and mortality, current civilian and military RTP guidelines for a particular athlete are largely based on anecdotal observation and caution (1,31,37). At this time, guidelines and recommendations for returning athletes/warfighters to play/duty are based on limited evidence. Most guidelines are common-sense recommendations that require an asymptomatic state and normal laboratory findings, targeting end organs such as the liver and kidney, coupled with a cautious reintroduction of activity and gradual heat acclimatization. Current suggestions vary widely from 7 to 15 months before EHS victims return to full activity (3). This lack of consistency and clinical agreement can negatively affect athletes and warfighters and force medical providers to guess what the best solution for each individual may be, directly influencing military force readiness. Whereas current guidance states that patients who have EHS may return to practice and competition when they have reestablished heat tolerance, clear clinical definitions of heat tolerance and intolerance are distinctly lacking. In addition, the role of heat tolerance testing and the potential effect of EHS on the thermoregulatory system are areas of scientific question and controversy (25).

This lack of clear evidence-based guidance has limited sports medicine professionals in making prudent RTP decisions when confronted with a patient with EHS. While clinicians can use basic hematologic parameters and blood chemistries to assess a return to normal of renal, hepatic, and coagulation functions, it is more challenging to consider recovery of potential exercise-heat tolerance deficits, neuropsychological impairments, or the altered fitness/acclimatization status (6,33,43). RTP after EHS should, as in any other injury, involve a carefully planned and progressively increased physical challenge that is closely supervised by an athletic trainer and physician, as previously identified in the ACSM conference statement (2,36). Clinicians should additionally remember that, as athletes rejoin colleagues on the athletic field, their training level, as well as their heat tolerance, may well be behind that of their peers; accordingly, caution is warranted. Although definitive high-level evidence-based guidelines regarding RTP do not presently exist, the current recommendations are summarized below.

Current Civilian Recommendations

The following summary of RTP guidelines set forth by the ACSM provides a rational process for guiding athletes who are returning to training and competition after EHS (3).

1. Refrain from exercise for at least 7 d after release from medical care;
2. Follow up about 1 wk after incident for a physical examination and laboratory testing or diagnostic imaging of the affected organs, based on the clinical course of the heat stroke (HS) incident;
3. When cleared for return to activity, begin exercise in a cool environment and gradually increase the duration, intensity, and heat exposure during 2 wk to demonstrate heat tolerance and to initiate acclimatization;
4. If return to vigorous activity is not accomplished during 4 wk, a laboratory exercise-heat tolerance test should be considered;
5. If heat tolerant, the athlete is cleared for full competition between 2 and 4 wk after the return to full training.

Current Army Recommendations

The military services do not share consensus recommendations on returning soldiers to duty after sustaining exertional heat illness (37). Accordingly, an ACSM roundtable of military and civilian experts was convened at the Uniformed Services University of the Health Sciences (Bethesda, MD) in 2008 to address inconsistencies (36). Key findings included the need for clearly defined terms, identification of necessary research, and emphasis on early intervention, which directly influences a warfighter’s capability to return to duty. Consequently, participants agreed that exertional heat illness cases require individualized risk stratification based on treatment and observed complications.
This conference assisted the Army Medical Department in moving forward with new consensus definitions for HE, heat injury (HI), and HS. The group specifically defined HS as a syndrome of hyperthermia (core temperature at time of event usually $\geq 40^\circ C (104^\circ F)$), collapse or debilitation, and encephalopathy (delirium, stupor, or coma) occurring during or immediately after exertion or significant heat exposure. HS can be complicated by organ and/or tissue injury, systemic inflammatory activation, and disseminated intravascular coagulation.

Participants agreed that, after an episode of HS, all affected warfighters should be placed on a temporary medical profile (i.e., removed from duty) for 2 wk. For the purpose of further profile and medical evaluation board (MEB) determination, the warfighter should be reassessed weekly for the presence or absence of complications and contributing risk factor(s). The warfighter will then be classified into one of the following three categories:

1. HS without sequelae: all clinical signs and symptoms resolved by 2 wk after the event;
2. HS with sequelae: evidence of cognitive or behavioral dysfunction, renal impairment, hepatic dysfunction, rhabdomyolysis, or other related condition that does not completely resolve within 2 wk of the event;
3. Complex HS: recurrent, or occurring in the presence of a nonmodifiable risk factor, either known (e.g., chronic skin condition, such as eczema or burn skin graft) or suspected (e.g., sickle cell trait, malignant hyperthermia susceptibility).

Using this guidance, the physician can then make a prudent recommendation regarding who may return to duty and who may require an MEB assessment with subsequent medical discharge from military service. This policy guidance is illustrated in Table 3 and further described at [http://champ.usuhs.mil/chclinicaltools.html](http://champ.usuhs.mil/chclinicaltools.html).

### Table 3.
Profile progression recommendations for the soldier with HS, or HE, HI, pending MEB.

<table>
<thead>
<tr>
<th>Profile Code*</th>
<th>Restrictions</th>
<th>HS without Sequelae</th>
<th>HS with Sequelae</th>
<th>Complex HS or HE/HI Pending MEB</th>
</tr>
</thead>
<tbody>
<tr>
<td>T-4 (P)</td>
<td>Complete duty restrictions</td>
<td>2 wk</td>
<td>2 wk minimum; advance when clinically resolved</td>
<td>2 wk minimum; advance when clinically resolved</td>
</tr>
<tr>
<td>T-3 (P)</td>
<td>Physical training and running/walking/swimming/bicycling at own pace and distance not to exceed 60 min $^{-1}$</td>
<td>1 mo minimum</td>
<td>2 mo minimum</td>
<td>Pending MEB</td>
</tr>
<tr>
<td></td>
<td>No maximal effort; no APFT; no wearing of IBA; no MOPP gear; no marching with rucksack</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>No AO</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T-3 (P)</td>
<td>Gradual acclimatization (TB Med 507).</td>
<td>1 mo minimum</td>
<td>2 mo minimum$^b$</td>
<td>N/A</td>
</tr>
<tr>
<td></td>
<td>No maximal effort; no APFT; no MOPP level IV gear; IBA limited to static range participation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>May march at own pace/distance with rucksack weighing no more than 30 lb</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Nontactical AO permitted</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T-2 (P)</td>
<td>Continue gradual acclimatization</td>
<td>N/A</td>
<td>Pending completion of 30-d heat exposure requirement, if not accomplished during prior observation period$^b$</td>
<td>N/A</td>
</tr>
<tr>
<td></td>
<td>May participate in unit physical training; CBRN training with MOPP gear for up to 30 min; IBA on static and dynamic ranges for up to 45 min; no recorded APFT; march with rucksack at own pace/distance with no more than 30 lb, up to 2 h</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Nontactical AO permitted</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

$^a$Warfighters manifesting no heat illness symptoms or work intolerance after completion of profile restrictions can advance and return to duty without an MEB determination. Any evidence/manifestation of heat illness symptoms during the period of the profile requires an MEB referral.

$^b$HS with sequelae return to full duty requires a minimum period of heat exposure during environmental stress (heat category 2 during the majority of included days).

T = temporary profile, P = physical category P (PULHES).

HS = heat stroke; HE = heat exposure; HI = heat injury; MEB = medical evaluation board; AO = airborne operations, APFT = Army physical fitness test, IBA = individual body armor, MOPP = mission-oriented protective posture, CBRN = chemical, biologic, radiologic, nuclear.
Conclusions

Proper recognition and understanding of the predisposing factors, errors in care, physiology of CWI, and return-to-play/duty considerations are critical to the appropriate prevention and care of EHS. A wide range of predisposing factors have been identified such as physical fitness, sleep deprivation, exercise intensity not matched with physical fitness, ineffective or absent medical triage, and disregard for organizational safety regulations; however, the primary predisposing factor of EHS is complex. EHS deaths are easily avoidable when accurate temperature assessment, prompt aggressive treatment using an efficient cooling modality (i.e., CWI or IWI) prior to transport, and medically supervised return to play/duty occurs.

The authors declare no conflict of interest and do not have any financial disclosures.

References


