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WILDERNESS MEDICAL SOCIETY PRACTICE GUIDELINES

Wilderness Medical Society Practice Guidelines for the Prevention and Treatment of Heat-Related Illness: 2014 Update

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The Wilderness Medical Society (WMS) convened an expert panel to develop a set of evidence-based guidelines for the recognition, prevention, and treatment of heat illness. We present a review of the classifications, pathophysiology, and evidence-based guidelines for planning and preventive measures as well as best practice recommendations for both field and hospital-based therapeutic management of heat illness. These recommendations are graded on the basis of the quality of supporting evidence, and balance between the benefits and risks or burdens for each modality. This is an updated version of the original WMS Practice Guidelines for the Prevention and Treatment of Heat-Related Illness published in *Wilderness & Environmental Medicine* 2013;24(4):351–361.

Key words: heat stroke, heat illness, prevention, recognition, treatment

Introduction

Heat-related illnesses are a common occurrence worldwide. The European heat wave of 2003 resulted in at least 70,000 fatalities,[1] and in the last decade the United States averaged more than 600 deaths annually associated with excessive heat exposure.[2] Currently heat-related illness is the leading cause of morbidity and mortality among US high school athletes.[3] Exertional heat stroke (EHS) mortality approaches 10%,[4] and when presenting with hypotension increases to 33%.[5] Outcome is directly attributed to both the magnitude and duration of hyperthermia,[6,7] making early recognition and treatment a priority. The Wilderness Medical Society (WMS) convened an expert panel to develop a set of practice guidelines for the recognition, prevention, and treatment of heat-related illness. We present a review of the classifications, pathophysiology, and evidence-based guidelines for planning and preventive measures as well as best practice recommendations for both field and hospital-based therapeutic management of heat-related illness. Although the spectrum of heat-related illness is discussed, this practice group's focus was on the exploration of EHS, which is synonymous with the term *heat stroke* in this paper unless otherwise specified.

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Methods

A panel was selected at the 2011 WMS Annual Meeting in Snowmass, CO. Specialists in emergency medicine, primary care, and critical care were chosen on the basis of their clinical or research experience. Relevant articles were identified through the PUBMED database using the following key words: hyperthermia, heat stroke, heat illness, heat syncope, and heat exhaustion. This was supplemented by a hand search of articles from references in the initial PUBMED search. Studies in these categories including randomized controlled trials, observational studies, and case series were reviewed. Abstract-only studies were not included. Conclusions from review articles were cited in an effort to provide background information, but were not considered in the formulation of recommendation grades. The panel used a consensus

approach to develop recommendations for the recognition and management of heat-related illness, with level of evidence assigned according to methodology stipulated by the American College of Chest Physicians (ACCP) for grading of evidence and recommendations (see the ACCP online Supplementary Table 1). These recommendations are graded on the basis of the quality of supporting evidence, and balance between the benefits and risks or burdens for each modality or intervention.[8]

Definition of Heat-Related Illnesses

Heat-related illness can be manifested as a spectrum of disease from minor to severe, such as heat cramps, heat syncope, heat exhaustion, and life-threatening heat stroke. Exertional hyperthermia occurs when heat generated from muscular activity accumulates faster than can be dissipated via increased skin blood flow and sweating. Body heat loss is controlled by peripheral centers in the skin and organs and the central nervous system via the hypothalamus, with a greater cooling response to temperature elevation via central sensors.[9] A temperature gradient exists between the body core and skin, which promotes heat dissipation when the core is higher than the surface. When the core temperature increases during exercise and the skin temperature also rises as a result of the environment or internal heat production, heat dissipation is reduced. Similarly, when the body's metabolic heat production outpaces heat transfer, the core temperature rises and heat illness can occur.[10] In some cases, injury may result from the pathophysiologic end points of normal processes used to maintain homeostasis rather than elevated core temperature. For example, the dehydration that is common in hot environments can result in maximal utilization of endogenous vasopressin to reclaim free water. Although the goal is to maintain euvolemia, prolonged reclamation of free water out of proportion to sodium with the additional consumption of free water may result in dilutional hyponatremia. Extended dependence on aldosterone to maintain euvolemia can cause potassium wasting that in turn may predispose to muscular heat cramps.[11]

Heat edema is a benign self-limiting condition. Interstitial fluid accumulates in dependent extremities as a result of hydrostatic pressure, vascular leak, and cutaneous vasodilation. Heat syncope refers to a multifactorial syndrome involving transient loss of consciousness in the context of heat exposure with a relatively rapid return to normal function and baseline. Contributing factors may include peripheral vasodilation, orthostatic pooling of blood, prolonged standing, advanced age, and dehydration, as well as coexisting medical conditions such as ischemic heart disease that reduces cardiac output. Although syncope can occur in both milder as well as more severe forms of heat illness, the term *heat syncope* generally refers to a more benign clinical condition that should resolve with rest and possibly rehydration at comfortable ambient temperatures.[12,13]

Heat exhaustion results from exposure to high ambient heat or strenuous exertion. This mild to moderate illness may progress to heat stroke if the exhausted hyperthermic individual is not recognized or left untreated in a hot environment. Heat stroke is defined as a core temperature above 40°C (104°F) with encephalopathy. Heat stroke is generally divided into 2 categories, classic heat stroke resulting from passive exposure to high environmental temperatures and exertional heat stroke resulting from pathologic hyperthermia during strenuous exercise (Table 1).[14]

Pathophysiology of Heat Stroke

Heat stroke may occur when internal core temperatures rise above a critical level, leading to a cascade of cellular and systemic responses. The responses include thermoregulatory

Table 1. Characteristics of heat related illness

Condition	*Definition*
Hyperthermia	A rise in body temperature above the hypothalamic set point when heat-dissipating mechanisms are impaired (by clothing or insulation, drugs, or disease) or overwhelmed by external (environmental) or internal (metabolic) heat production
Heat edema	Dependent extremity swelling owing to interstitial fluid pooling
Heat cramps	Exercise-associated painful involuntary muscle contractions during or immediately after exercise
Heat syncope	Transient loss of consciousness with spontaneous return to normal mentation
Heat exhaustion	Mild-to-moderate heat-related illness owing to exposure to high environmental heat or strenuous physical exercise; signs and symptoms include intense thirst, weakness, discomfort, anxiety, and dizziness, syncope; core temperature may be normal or slightly elevated >37°C (98.6°F) but <40°C (104°F)
Heat stroke	Severe heat-related illness characterized by a core temperature >40°C (104°F) and central nervous system abnormalities such as altered mental status (encephalopathy), seizure, or coma resulting from passive exposure to environmental heat (classic heat stroke) or strenuous exercise (exertional heat stroke)

dysfunction, an acute phase response, and a heat shock protein response. A rise in blood temperature of less than 1°C triggers hypothalamic thermoregulation to increase blood flow to the skin by up to 8 L/min via sympathetic cutaneous vasodilation. As blood is shunted to the periphery to facilitate evaporative heat loss through sweating, renal and splanchnic perfusion is reduced.[15] The acute phase response to heat stress involves an inflammatory reaction of interleukins, cytokines, and proteins that progresses in a similar sequence to that seen in sepsis. It is theorized that an exaggerated acute phase and inflammatory response mark the progression from heat stress to heat stroke, possibly incited by the hypoperfused gastrointestinal tract.[14] Increased mucosal permeability from inflammatory mediators allows endotoxins from the gut to enter the systemic circulation. This combination of endotoxemia and cascade of inflammatory cytokines leads to alterations in the microcirculation, further endothelial and tissue injury, and impaired thermoregulation, thus precipitating heat stroke and hypotension. An overlapping hypothesis presupposes that at a similar critical threshold temperature, the expression of protective heat shock proteins is altered, decreasing their ability to prevent thermal denaturation of structural proteins and enzymes that start to fail at a cellular level, with ensuing end-organ dysfunction.[14,16] At critical levels of hyperthermia, heat causes direct tissue injury and death via apoptosis or necrosis, with the severity of injury dictated by both the level and duration of thermal stress.[17,18] This complex constellation of overlapping events leads to thermoregulatory failure, heat stroke, and circulatory shock.

Prevention and Planning

The proverb that "an ounce of prevention is worth a pound of cure" is especially apt to the potentially fatal nature of heat illness. Deliberate strategies for prevention should be included when planning for activities with a credible risk. Structured risk assessments can be built and validated for population-level use,[19,20] or the practitioner considering a particular scenario should consider the risk incurred by the individual participant's physiology, the environment, and the planned activity.

INDIVIDUAL FACTORS

Any condition that limits heat loss through the skin may lead to heat retention, including hypohidrosis, extensive scars, and diminished cardiopulmonary reserve of the elderly. Small studies have linked acute sunburn with impaired sweating, a risk factor for heat accumulation. Effects persisted for 7 days, considerably longer than the associated pain and erythema, but were of unclear clinical significance.[21,22]

Table 2. Medications and drugs that may contribute to heat-related illness

Alcohol
Alpha adrenergics
Amphetamines
Anticholinergics
Antihistamines
Antipsychotics
Benzodiazepines
Beta-blockers
Calcium-channel blockers
Clopidogrel
Cocaine
Diuretics
Laxatives
Neuroleptics
Phenothiazines
Thyroid agonists
Tricyclic antidepressants

Certain drugs can predispose individuals to heat injury by 2 primary pathways, increased heat production caused by drug actions and compromised function of thermoregulatory centers (Table 2).[23,24]

Moderate caffeine intake appears to have no detrimental effect.[16] Studies on military recruits show an increased risk of heat injury among obese or overweight individuals,[25,26] but other data suggest there is no excess exertional heat accumulation in heavier subjects when matched for aerobic fitness levels.[27]

Heat acclimatization, as induced by 1 to 2 hours of heat-exposed exertion per day for 10 to 14 days, results in reproducible adaptations that increase the body's ability to tolerate and divest heat.[28–30] These adaptations may persist for up to a month.[31,32] Evidence suggests that a bout of heat stroke may acutely reset these thermoregulatory adaptations and cause elevated risk for subsequent heat injury for months after the initial event,[33] although case reports indicate that heat tolerance can be recovered fully.[34,35] Individuals with high levels of cardiopulmonary fitness tolerate more activity in heat-strained conditions and acclimatize to heat more rapidly because they have increased sweat volumes and higher subjective tolerance for activity when hyperthermic. The most readily modifiable physiologic risk factor is hydration status. Although endurance athletes may comfortably tolerate weight losses of 3% to 4% during events,[36,37] fluid losses that result in a 2% to 3% decrease in body weight correlate with greater core temperatures at a given workload in the heat.[29,38,39]

Hyperhydration before activity has not been shown to have a significant effect on heat tolerance, nor has active

body cooling before activity.[40] One investigation on the effect of sex on thermal recovery was confounded by body mass index differences, such that no conclusion can be reached as a risk factor.[41] The luteal phase of the menstrual cycle that is associated with increased core temperatures does not appear to induce heat intolerance in females taking oral contraceptive pills.[42] The physiology of pediatric and elderly populations differs enough from that of healthy adults to warrant special considerations that are outside this panel's scope, but are discussed at length elsewhere.[43–45]

Recommendation grade: Screen for significant preexisting medical conditions: 1B.

Minimize use of medications that would limit the thermoregulatory response: 1C.

Recognize that a larger body habitus is associated with greater risk: 1C.

Promote regular aerobic activity before exposure: 1C.

Allow for acclimatization with 1 to 2 hours per day of heat-exposed exertion for at least 8 days: 1C.

Ensure euhydration before activity: 1B.

Ensure ongoing rehydration with a "drink to thirst" approach sufficient to prevent $>2\%$ loss of body weight: 1B.

Consider previous history of heat injury as a reversible risk factor for recurrence: 1C.

ENVIRONMENTAL CONSIDERATIONS

The body and the environment exchange heat through several mechanisms: conduction (passively transferring heat from the body to the surrounding environment along a temperature gradient by direct contact), evaporation (heat transfer from the body to sweat resulting in transition of water from the liquid to vapor phase), thermal radiation (infrared rays given off by any mass as a function of the temperature of that mass), or convection (transfer of heat from the body to air or water moving across the skin surface). As the environmental temperature increases, the body will eventually incur a net heat gain through convective and radiative processes, leaving evaporative thermoregulation as the only cooling mechanism. The vaporization of 1.7 mL of sweat consumes 1 kcal of heat[46]; however, evaporative cooling is less effective in highly humid environments as it lowers the water vapor pressure difference between the sweat on the skin and the water in the surrounding air. High wind optimizes this gradient. The wet-bulb globe temperature index (WBGT) is a composite index of temperature, humidity, and solar radiation that expresses the total thermal strain that an individual experiences. A series of WBGT values can be designated as cautionary warnings and triggers to activate guidelines for rehydration, active cooling, and limitations (or even cancellation) of physical activity.[32] An alternative to the WBGT that is more readily available is the heat index, which is a measure of the contribution that high temperature and high humidity (expressed as either relative humidity or dew point temperature) make in reducing the body's ability to cool itself. Although the WBGT is a metric likely not readily available to individual medical practitioners, its current use by military,[47] occupational,[48] and clinical groups[10,43] makes it the standard when discussing environmental thermal strain and choosing activity levels for ambient conditions. Guidelines for correlation of heat index and risk of heat injury and limitations of physical activity are readily available.[49]

Recommendation grade: WBGT should be used when assessing heat risk: 1A.

ACTIVITY CONSIDERATIONS

The metabolic thermal output of an activity is the product of its intensity and duration. The contribution of heat to the body is tempered in some circumstances by an activity that can enhance heat transfer with the environment (eg, water convection on a swimmer or wind past a cyclist). Occupational,[48,50] military,[47] and medical[10] guidelines recommend breaks in proportion to metabolic demand and ambient conditions, but there are few studies examining how to optimize the dosing of breaks.

Recommendation grade: Consider which mechanisms of heat accumulation or dissipation are dominant during an activity, and consider heat loss as a key feature of breaks: 1C.

CLOTHING AND EQUIPMENT

Clothing or other equipment worn during an activity may limit or enhance the body's thermoregulatory efficiency. Of particular importance is equipment that occludes regions of skin, resulting in compromise of evaporative, convective, radiative, or conductive heat transfer. For example, the American football uniform prevents full heat exchange across much of the torso and head and can therefore contribute to heat accumulation,[51] similar to military helmets and body armor.[52] Preventing heat exchange may be protective, as in the case of firefighting gear that prevents incumbent radiative and conductive heat from entering the body from a superheated environment. Sports medicine guidelines as well as military occupational guidelines have set examples of systematic reductions in clothing and equipment based on WBGT thresholds.[10,53]

Recommendation grade: Clothing and equipment for a given activity should be evaluated or modified to optimize evaporative, convective, conductive, and radiative heat exchange or isolation: 1C.

Field Treatment

Optimal field management of heat-related illness may be challenging because of limitations of resources or extreme settings. The ideal treatment, as emphasized in the previous section, is prevention by avoiding high-exertion activities in exposed or hot areas. The method and aggressiveness of cooling in the field depend on the type of heat-related illness encountered (Table 3). Regardless of the underlying cause, rapid reversal of the hyperthermia is critical as the extent of morbidity is directly related to both to the degree and duration.[25,54–57] All treatment in the field is first directed to stabilization of the patient's airway, breathing, and circulation before proceeding to more-specific cooling therapy. If no life-threatening complications exist, the implementation of on-site cooling before evacuation is preferred.[10] In transitioning patient care to emergency medical service providers, it is important to communicate any cooling techniques begun in the field, and to continue cooling the patient by the best available means en route to the destination.

MINOR HEAT-ILLNESS TREATMENT

There is scant evidence supporting treatments of minor and moderate heat-related illness. Most treatments are anecdotal but effective, and generalizable from the evidence-based treatment for more severe forms of heat-related illness (Table 3). Heat cramps, which are historically described as generalized[11] may differ from the focal exercise-associated muscle cramps seen in endurance athletes. Although exercise-associated muscle cramps occur with neuromuscular fatigue and are relieved with passive stretching,[58] heat cramps are relieved with oral salt solutions or electrolyte replacement that may be isotonic or hypertonic.[11] Heat edema is reversed by extremity elevation or wearing of compression stockings. Diuretics are ineffective and may worsen volume depletion.[59] Heat syncope by definition is self-limiting. After consideration of other medical causes of syncope or resultant trauma from the fall, treatment consists of ensuring replacement of vascular volume with isotonic oral fluids, and rest in a cool environment.[13] Individuals at risk for heat syncope should move often and flex their larger leg muscles to prevent peripheral pooling of blood caused by cutaneous vasodilation. Heat exhaustion in mild cases generally resolves with moving to a cool environment, ceasing physical activity, and oral rehydration of isotonic fluids. More severe exhaustion typically has more pronounced volume depletion and may require intravenous replacement of fluids as well as evaporative and convective cooling.

Temperature measurement

When possible, obtaining an accurate core body temperature is a critical diagnostic step in differentiating heat stroke from less severe heat injuries. Rectal temperature

Table 3. Heat-related illness treatments

Severity of heat-related illness	*Diagnosis*	*Treatment*
Mild	Heat cramps	Oral isotonic or hypertonic fluid replacement
	Heat edema	Extremity elevation
		Compression stockings
Moderate	Heat syncope	Remove from heat source
		Passive cooling
		Oral isotonic or hypertonic fluid hydration
	Heat exhaustion	Remove from heat source
		Evaporative and convective cooling
		Oral or intravenous isotonic or hypertonic fluid hydration
Severe	Heat stroke	Remove from heat source
		Supportive care of airway, breathing, and circulation
		Cold-water immersion
		Evaporative and convective cooling
		Intravenous hydration[a]
		Evacuation[b]

[a] Intravenous hydration with isotonic (0.9% NS) or hypertonic (D5NS) fluids, with 3% NS preferred if concern for exercise-associated hyponatremia as cause of encephalopathy.

[b] Initiate emergency medical services if unable to rapidly cool patient, prolonged encephalopathy, or concern of multiorgan dysfunction.

is widely considered the gold standard,[10,57] because it is the most reliable and practical measurement of core temperature and is more accurate than temporal, axillary, oral, or aural thermometry in the field setting.[12,60] Esophageal and ingestible thermistors have been validated but are impractical in the wilderness setting. Rectal temperature measurement is relatively invasive and carries implicit difficulty in maintaining patient privacy and hygienic conditions, so initial assessment and aggressive cooling should be implemented on the basis of clinical suspicion, regardless of the degree of hyperthermia or mode of measurement. When available, rectal temperature should be considered the most accurate measurement of core hyperthermia. In a hyperthermic individual with an altered sensorium, the initiation of empiric cooling for heat stroke should not be delayed by a measurement value that may be below the diagnostic threshold of 40°C.
Recommendation grade: 1B

Passive cooling

Simple measures can be easily taken to reduce the patient's exposure to heat transfer. Moving the victim into the shade can externally decrease the ambient temperature; however, this is most effective when temperatures are less than 20°C (68°F).[55] Placing the victim on an insulating barrier such as a sleeping pad or sleeping bag can decrease conduction of heat from the ground. Loosening or removing any tight-fitting clothing to optimize air circulation aids in convective heat exchange.[61]
Recommendation grade: 1C

Hydration

Rehydration is an important factor in reducing hyperthermia.[61,62] Hypohydration decreases sweat rates, increases core temperature,[62,63] and predisposes to a worsening of the severity of the heat illness. The National Athletic Trainers' Association lists hydration as an important factor in reducing hyperthermia. Both oral and intravenous hydration have been shown to be equally effective in replenishing water deficiencies related to heat stress,[62,64] but in a heat-stroke victim with altered mental status and risk of seizure, the intravenous route minimizes aspiration risk and subsequent airway compromise.
Recommendation grade: 1C

Symptomatic exercise-associated hyponatremia may present similarly to heat exhaustion,[65] and presence of altered mental status in the absence of hyperthermia may necessitate resuscitating with 3% normal saline (hypertonic saline).[66] Little data exist on the intravenous fluid type and amount relating specifically to heat-related illness. As those with EHS may be volume depleted from insensible fluid losses, the best choices of replenishment are 1 to 2 L of isotonic (normal saline, 0.9% NS) or hypertonic (NS with 5% dextrose) fluids. Care should be taken to not overhydrate patients (especially those with coronary comorbidity) as this may lead to pulmonary edema.[67] Any effort to provide hydration in suspected heat stroke should not delay rapid whole-body cooling.[68] If intravenous hydration is provided, field monitoring of blood pressure, heart rate, lightening of urine color, and increase in urine output can help guide patient response and fluid status.
Recommendation grade: 1B

Cold-water immersion therapy

Cold-water immersion therapy is the optimal field treatment to achieve rapid temperature reduction below critical levels in heat stroke. Ice water cooling has been shown to be twice as rapid to reduce core temperature as spraying water over the body to enhance evaporative cooling (0.20°C/min vs 0.11°C/min),[54] with faster cooling the colder the water.[69] Immersion takes advantage of water's high thermal conductivity, which is 24× greater than air,[70] and the high thermal gradient between ice water and skin,[71] which translates into a great capacity for heat transfer. The theoretical concern that cold-water immersion causes peripheral vasoconstriction and shivering, which slows cooling or may even increase the core temperature, is a prevalent misconception—possibly stemming from a misinterpretation of the "Currie response" that can increase the core temperature in normothermic individuals by 0.1° to 0.2°C. Although shivering has been observed in immersions lasting longer than 10 minutes in healthy volunteers,[72,73] such shivering may be less problematic in actual heat-stroke patients.[74] In addition, the hindrance of cooling EHS by heat-generating shivering has been physiologically refuted.[69,75] The availability of a water bath or body of water that is required for this cooling modality may limit its usefulness in many settings. Cold-water immersion is best achieved by removing all clothes and equipment and submersing the patient's trunk and extremities in a cold-water bath or other convenient body of water such as a stream, pond, river, or lake—using a natural body of water may be the only option in field treatment. Special care should be taken to protect against currents, to ensure the head does not go underwater, and to protect the airway—the victim should never be left alone because of the risk of aspiration and drowning. In lieu of a cold-water source, repeated dousing of the victim with cold water or snow, if available, is encouraged. Indeed,

multiple military studies on immersion cooling of comparatively young and healthy EHS victims boast a 0% fatality rate,[76] providing strong support that rapid treatment with this cooling modality has the best outcomes.
Recommendation grade: 1A

Evaporative cooling

If immersion is unavailable, evaporative cooling measures should be initiated. Start by loosening or removing clothing, spraying or dousing the victim with water to maximize water–vapor skin interface,[77] and facilitating convection with air movement by fanning. This is the preferred method of treatment for heat exhaustion in most conditions. Studies using cold or warm cooling mist water achieve rates from 0.04°C/min to 0.08°C/min.[75] No studies on traditional evaporative cooling have been done with exertional heat-stroke victims. Other evaporative methods such as using the downdraft from a helicopter from a small case series have been shown to be slightly more effective (0.10°C/min), albeit more technically complicated.[78]
Recommendation grade: 1C

Chemical cold packs/ice packs

There is a tradition of advocating for the use of ice packs or chemical cold packs strategically applied to the skin covering the neck, axillae, and groin to cool blood flow passing in the major vessels.[79] Limited studies show minimal benefit in heat reduction when ice packs or chemical cold packs are used alone in this traditional cooling method. Ice packs have been shown to be more efficacious when covering the entire body.[80,81]
Recommendation grade: 1C

Ice-towel application

Wrapping the EHS victim in wet towels may be used as an alternative conductive cooling method when immersion is not easily available. One study showed clinically significant cooling rates, but methodological flaws in the data precluded definitive conclusions.[54]
Recommendation grade: 2B

Antipyretics

As clinicians, we generally treat elevated temperatures with antipyretics. This class of medications, which include acetaminophen, ibuprofen, and aspirin, work by inhibiting the formation of prostaglandins and lowering the thermoregulatory set point;[82] although this may be elevated in infectious causes of hyperthermia, this is not the case in exercise-induced hyperthermia. Antipyretic drugs are ineffectual and should be avoided.[83,84]
Recommendation grade: 2B

Hospital Treatment

Patients with heat stroke should be transported to a medical facility capable of critical care management of patients with multiple organ failure. The primary goals of treatment for heat stroke are lowering core body temperature as rapidly as possible and supporting organ system function,[14] as patients may develop multiple organ failure with shock, acute respiratory failure, acute kidney injury, disseminated intravascular coagulopathy, and intestinal ischemia. Depending on the patient's clinical status, supportive treatment may include administering supplemental oxygen, performing intubation and mechanical ventilation, establishing adequate intravascular access, restoring intravascular volume with intravenous isotonic crystalloid solution, placing a bladder catheter to monitor urine output, and initiating vasopressors to support blood pressure (after adequate volume resuscitation).

The evidence on different cooling methods has involved a heterogeneous range of subjects with exercise-induced hyperthermia, EHS, or classic heat stroke. Of the studies comparing different cooling methods, those involving randomized trials generally have been performed on healthy volunteers with exercise-induced hyperthermia and have enrolled relatively few subjects. The remaining studies on treating heat-stroke patients have for the most part been case-series reports or nonrandomized comparisons of treatment methods—with considerable variations in the baseline characteristics of subjects from one study to the next. The majority of experimental studies have shown cold-water immersion as the most efficacious cooling method. However, clinical practice has historically promoted 2 methods of cooling: 1) conductive cooling via cold-water immersion of the patient, and 2) evaporative and convective cooling via the application of sprayed water and forced air currents over the body.

CONDUCTIVE COOLING

The historical record has suggested that cold-water immersion is safe and effective for young, athletic patients with exertional heat stroke. A cooling protocol used for more than 15 years involving an ice-water slurry has been applied effectively with no fatalities or adverse effects in hundreds of military personnel.[85–87] Agitation, intolerance, or combativeness may occur in encephalopathic heat-stroke patients, and benefits of immersive cooling should be balanced with the theoretical concerns

of impaired access to an immersed patient who may require advanced cardiac monitoring or resuscitation, especially among older patients.[74,88,89]
Recommendation grade: 1A.

EVAPORATIVE AND CONVECTIVE COOLING

Evaporative cooling in elderly patients may offer several theoretical advantages, such as greater patient comfort and less agitation, as well as easier access to patients who may need advanced monitoring or resuscitative procedures. In general, studies on evaporative and convective cooling have involved classic heat-stroke patients and experimental volunteers with exercise-induced hyperthermia, but not patients with actual EHS. The larger studies using a specially constructed device, termed a body-cooling unit (BCU), have produced cooling rates ranging from 0.04°C/min to 0.11°C/min, with an average cooling of time of 68 to 78 minutes and 10% mortality.[88,90] No direct comparisons between the BCU and cold-water immersion are available, but extrapolation of cooling rates suggests evaporative and convective cooling is an order of magnitude less efficacious. As classic heat-stroke patients are more likely to be older and obese, with medical conditions such as diabetes, high blood pressure, and heart disease, the evidence suggests that the evaporative plus convective cooling technique by wetting and fanning the skin has an acceptable hospital-based role in the treatment of classic heat stroke, with a less effective role in EHS.
Recommendation grade: 1C.

TARGET COOLING TEMPERATURES

The target cooling temperatures of EHS and exercise-induced hyperthermia to less than 39°C by ice-water immersion has been well tolerated, with no fatalities, adverse outcomes, or core temperature "afterdrop" resulting in hypothermia.[85–87,91] Practitioners should also be cautious of falsely elevated rectal temperature measurements in the recovery phase as a result of the insulating effect of body mass.[92]
Recommendation grade: 1B.

COMBINED OR ADJUNCTIVE COOLING TREATMENTS

If intravenous fluids are available, it is beneficial to use cold fluids (4°C) whenever possible. These can decrease core temperature at a 2-fold rate compared with room temperature fluids but provide insufficient cooling as a primary treatment for heat stroke.[68,93] *Recommendation grade*: 1C. More invasive techniques of body cavity lavage with cold isotonic fluid have been reported, but have not been adequately studied.[94,95] Intravascular cooling devices may provide adjunctive treatment for heat stroke but require further study.[96]
Recommendation grade: 2C.

PHARMACOLOGIC TREATMENT

No pharmacologic agent has been shown to be helpful as a treatment for heat stroke. Dantrolene has been used for treatment of malignant hyperthermia and neuroleptic malignant syndrome. It acts by impairing calcium release from the sarcoplasmic reticulum, thereby reducing the muscular rigidity and hypertonicity typical of these conditions. A well-designed randomized clinical trial of dantrolene vs placebo in classic heat stroke showed no difference in cooling rates or outcome, concluding that this pharmacologic treatment should not be used in heat-stroke patients.[97]
Recommendation grade: 1B

Conclusions

This article provides evidence-based guidelines for the prevention, recognition, and treatment of heat-related illness. Much of the available data are case series or extrapolation of results stemming from exercise-associated hyperthermia, which are an accepted research model as randomized controlled trials for treatments of EHS are ethically challenging to justify. These guidelines apply the strength of the evidence to 2 distinct populations of heat-stroke victims, and although the patient with EHS is more likely to be found in the wilderness environment, the medical provider should be aware of all therapeutic modalities and their inherent risks and benefits. We recommend that patients with heat stroke should be cooled by conductive means by whole-body ice-water or cold-water immersion (preferential method in EHS), or evaporative and convective cooling by a combination of cool water spray with continual airflow over the body (acceptable method in classic heat stroke). Evaporative and convective cooling may be augmented with the addition of ice packs over the entire body to promote conductive cooling. Future areas of research should include direct comparisons of available cooling modalities in controlled models, as well as further evaluation of endovascular catheters and hospital-based systems for optimum cooling of critical patients.

Supplementary tables

Supplementary ACCP Table 1 and Evidence Table 2 are available online at doi:10.1016/j.wem.2014.07.017.

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