Management of Heat-Related Illness and Injury in the Intensive Care Unit: A Concise Definitive Review

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OBJECTIVES: The increasing frequency of extreme heat events has led to a growing number of heat-related injuries and illnesses in ICUs. The objective of this review was to summarize and critically appraise evidence for the management of heat-related illnesses and injuries for critical care multiprofessionals.

DATA SOURCES: Ovid Medline, Embase, Cochrane Clinical Trials Register, Cumulative Index to Nursing and Allied Health Literature, and ClinicalTrials.gov databases were searched from inception through August 2023 for studies reporting on heat-related injury and illness in the setting of the ICU.

STUDY SELECTION: English-language systematic reviews, narrative reviews, meta-analyses, randomized clinical trials, and observational studies were prioritized for review. Bibliographies from retrieved articles were scanned for articles that may have been missed.

DATA EXTRACTION: Data regarding study methodology, patient population, management strategy, and clinical outcomes were qualitatively assessed.

DATA SYNTHESIS: Several risk factors and prognostic indicators for patients diagnosed with heat-related illness and injury have been identified and reported in the literature. Effective management of these patients has included various cooling methods and fluid replenishment. Drug therapy is not effective. Multiple organ dysfunction, neurologic injury, and disseminated intravascular coagulation are common complications of heat stroke and must be managed accordingly. Burn injury from contact with hot surfaces or pavement can occur, requiring careful evaluation and possible excision and grafting in severe cases.

CONCLUSIONS: The prevalence of heat-related illness and injury is increasing, and rapid initiation of appropriate therapies is necessary to optimize outcomes. Additional research is needed to identify effective methods and strategies to achieve rapid cooling, the role of immunomodulators and anticoagulant medications, the use of biomarkers to identify organ failure, and the role of artificial intelligence and precision medicine.

KEYWORDS: cooling; heat stroke; heat-related illness; heat-related injury; intensive care unit; organ failure

Heat waves and high temperatures are predicted to increase in frequency, and exposure to hot weather is considered one of the deadliest natural hazards (1, 2). During a heat wave, heat-related illness occurs in epidemic form, and ICU admissions increase significantly (3, 4). In the United States, between 2000 and 2010, there were over 30,000 hospitalizations for heat diseases, with a predominance of cases among the elderly, athletes, and outdoor workers (5, 6). Furthermore, mortality has been shown to increase by 3% with each 1°C increase in daily mean temperature and duration of exposure (7).

Data from a U.S. emergency medical services (EMS) tracking program report an increase in heat-related EMS activations from 10.5 to 14.9 per 100,000 people

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KEY POINTS

Question: What is the supporting evidence for the management of heat-related illness and injury?

Findings: High-quality data for many aspects of care are limited. Cooling therapies should be rapidly implemented using methods that achieve an optimal rate of temperature control. Multisystem organ failure is common, and appropriate management strategies should be applied.

Meaning: There are several practical issues that must be considered when implementing an evidence-based cooling strategy. Rapid cooling and treatment of organ failure are necessary to optimize outcomes. Research gaps exist regarding numerous aspects of care for heat-related illnesses and injuries.

between 2018 and 2022 (8). In the summer of 2022, there were an estimated 61,672 heat-related deaths in Europe, and a recent report noted the boreal summer of 2023, as the hottest ever recorded globally (9, 10). In July 2023, Phoenix, Arizona, recorded 31 consecutive days with temperatures above 110°F with 1350 reported emergency department (ED) visits for heat-related illness (11).

Extreme temperatures can also lead to heat-related injury from hot pavement burns often requiring lengthy hospital stays, and the need for operative intervention (12). Asphalt temperature can reach 166°F, porous rock 170°F, and sand 143°F; all of which can lead to full thickness (third-degree) burn injury in minutes (13).

In response to the increasing prevalence of extreme temperatures and the resultant need for critical care, the Society of Critical Care Medicine created a task force to evaluate current evidence on the management of heat-related illness and injury for critical care multiprofessionals. This Concise Definitive Review provides a detailed overview of these findings.

MATERIALS AND METHODS

A literature search was performed using PubMed, Ovid Medline, Embase, Cochrane Clinical Trials Register, Cumulative Index to Nursing and Allied Health Literature, and ClinicalTrials.gov databases from inception through August 2023. English-language systematic reviews, narrative reviews, randomized clinical trials, observational studies, and case reports pertaining to heat-related illness and injury were extracted. Bibliographies were reviewed for any articles that were missed by the primary literature search.

Pathophysiology

The pathophysiology of heat-related illness is centered around the body's ability to regulate heat loss in the setting of excessive heat gain. If compensatory mechanisms (e.g., thermoregulation, increased cutaneous blood flow) become overwhelmed, there is direct heat-related damage to the cell cytoskeleton, DNA, proteins, and organelles culminating in both necrotic and apoptotic cell death (14). Additionally, an inflammatory response occurs along with coagulation disorders, alterations in blood flow, cardiac collapse, and multisystem organ failure (15). (**Fig. 1**)

Classification and Definition of Heat-Related Illness and Injury

Heat-related illness can be classified along a continuum based on severity ranging from mild (e.g., heat cramps, rash) to life-threatening (16–21). (**Table 1**) Heat stroke, the most severe form of heat-related illness is defined as a core temperature above 40°C with CNS abnormalities (18). Patients with heat stroke should receive multidisciplinary ICU care and will be the focus of this article. Patients with less severe forms of heat-related illness generally do not require ICU admission unless they fail to respond to cooling therapies, symptoms worsen, or end-organ damage is recognized.

Heat stroke is subclassified as either classic or exertional. Classic, also referred to as nonexertional, heat stroke (CHS) results from exposure to high environmental temperatures and poor heat dissipation. It is not due to exertion. Classic heat stroke is typically encountered in the elderly, patients with preexisting medical conditions (e.g., diabetes, cardiovascular disease), decreased thirst sensation and certain medication therapies (e.g., anticholinergics, antipsychotics, beta-blockers, diuretics, etc.). Exertional heat stroke (EHS) occurs usually in healthy patients when excessive production of body heat exceeds physiologic heat loss during physical exercise, outdoor activity or in certain occupations (e.g., construction workers, landscapers, firefighters, military personnel) (15). EHS patients are frequently younger and healthier, and experience a rapid rise in temperature over a short duration of time.

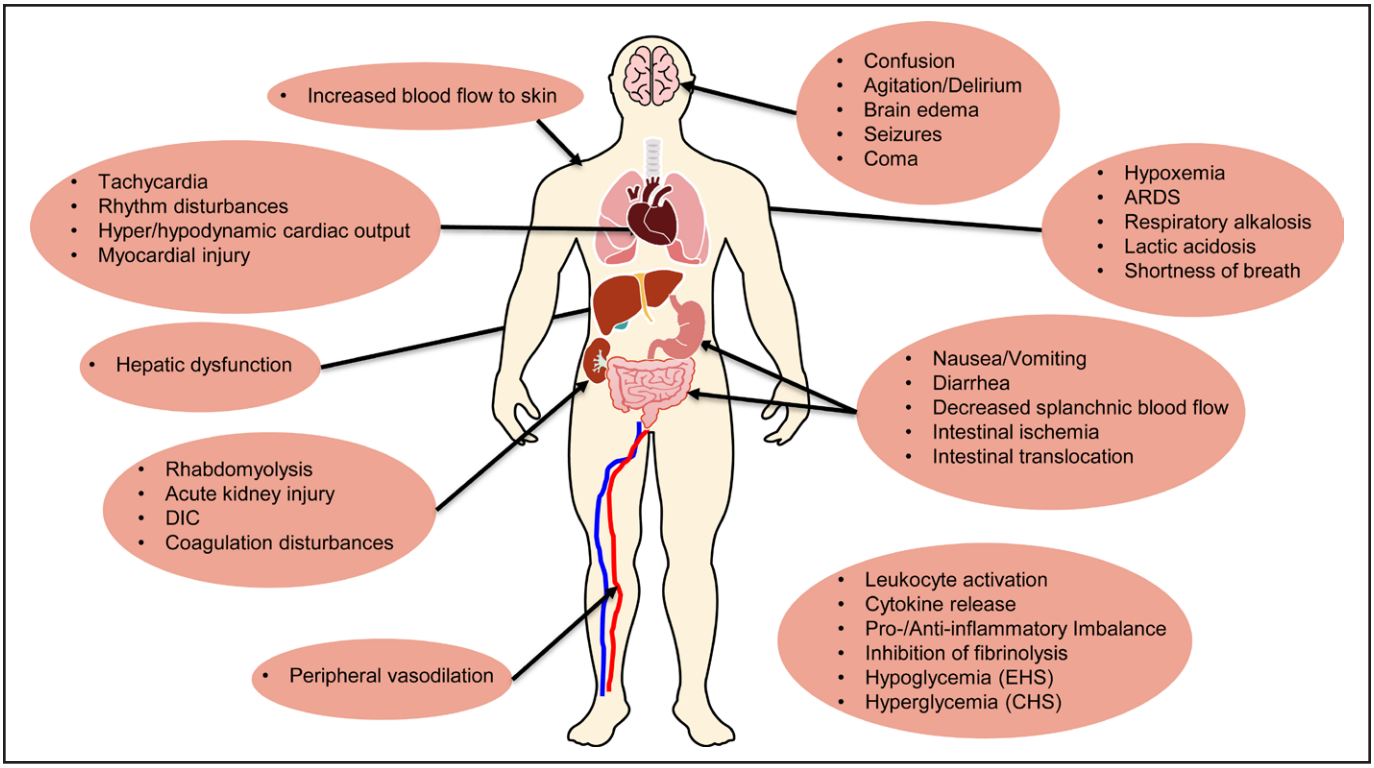

Figure 1. Signs, symptoms and physiologic abnormalities encountered in heatstroke patients. ARDS = acute respiratory distress syndrome, CHS = classic heat stroke, DIC = disseminated intravascular coagulation, EHS = exertional heat stroke.

TABLE 1.
Definitions of Conditions Associated With Heat-Related Illness

Clinical Condition	Definition	Signs and Symptoms
Heat stroke	Extreme core body temperature (> 40°C) and CNS dysfunction	Altered mental status, seizures, coma, multisystem organ failure
Heat exhaustion	Profound fatigue and/or diminished physical activity in the heat due to salt or water depletion and inability to sustain cardiac output	Fatigue, nausea, vomiting, headache, dizziness, anxiety, flushing, profuse sweating, normal mental status
Heat syncope	Dizziness or fainting in a hot environment due to volume depletion and postural blood pooling in lower extremities	Generalized weakness, syncope, postural syncope with rapid recovery once supine
Heat cramp	Painful muscle spasms during exercise in the heat usually due to fluid and electrolyte depletion	Painful muscle contractions, affected muscles are stiff and tender to palpation
Heat edema	Extremity swelling due to peripheral vasodilation and interstitial fluid pooling	Swollen extremities (usually lower limbs) from sitting or standing for a long time in a hot environment
Heat intolerance	Inability to tolerate extreme heat	Excessive sweating, feeling overheated, dizziness, lightheadedness
Heat rash	Results from blockage of sweat glands from excessive sweating	Presence of rash, inflammation, infection

EHS does not necessarily coincide with hot weather, so a high index of suspicion is needed when patients present.

Risk Factors

There are numerous contributing factors for heat-related illness which are related to duration and extent of heat exposure, inability to dissipate heat, decreased thirst sensation, individual susceptibility, societal, and predisposing factors (22) (**Table 2**). Additionally, children left unattended in parked vehicles represent a preventable cause of heat-related deaths (23). From 1998 to 2023, 968 pediatric vehicular heatstroke deaths have been reported in the United States (24). Individuals at risk for heat-related injury include children unaware of the potential danger of walking barefoot or playing on a hot surface, diabetics with peripheral neuropathy, and those who are rendered unconscious for medical reasons, such as stroke, seizures, trauma, and intoxication (12, 25, 26).

Clinical Presentation and Diagnosis

The formal diagnosis of CHS is clinically based on mental status changes (coma, delirium, convulsions, disorientation, seizures), a body temperature greater than 40°C, and the presence of hot and dry skin, although some patients do not have skin aberrations (21). With EHS, high temperatures (i.e., > 40°C) and CNS dysfunction are present (e.g., ataxia, dizziness, weakness, irritability, nausea, vomiting, seizures), and profuse sweating and wet skin are typically observed (27, 28). The occurrence of organ dysfunction is high in both categories of heat stroke (3). Clinical features of system dysfunction are depicted in Figure 1 (3, 14, 29–33).

CLINICAL MANAGEMENT

A detailed description of therapies for heat-related illness and injury is provided in the text below. These therapies represent a continuum of care across a

TABLE 2.
Risk Factors for Heat-Related Illness and Injury

Individual Susceptibility or Societal Factors	Chronic Disease or Predisposing Treatment
• Absence of air conditioning • At-risk occupations (e.g., outdoor workers, firefighters, military) • Children unintentionally left in automobiles • Duration of exposure • Excessive exercise • Inappropriate clothing • Living alone/social isolation • Maximum temperature • Mechanical falls • Older age • Obesity • Prepubertal children • Undomiciled • Walking on hot pavement	• Alcohol consumption • Burns • Dehydration • Infection • Medications ◦ Anticholinergics ◦ Antihistamines ◦ Antipsychotics ◦ Benzodiazepines ◦ Beta-blockers ◦ Calcium-channel blockers ◦ Diuretics ◦ Laxatives ◦ Lithium ◦ Serotonin-reuptake inhibitors ◦ Thyroid medications ◦ Tricyclic antidepressants • Preexisting comorbidities ◦ Cognitive impairment ◦ Diabetes mellitus ◦ Heart disease ◦ Neurologic disorders ◦ Psychologic disorders ◦ Respiratory disease ◦ Sweat gland dysfunction/skin disorders • Substance abuse disorders (e.g., cocaine, heroin, amphetamines)

variety of settings (e.g., field, ED, ICU) recognizing that the provision of critical care has no geographic boundaries. There is a paucity of high-quality direct evidence (i.e., trials conducted in actual patients with heatstroke) to guide clinical decision-making. Existing clinical data are limited by small sample sizes and trials that were conducted in controlled environments or laboratory settings, specifically those assessing cooling methods. Although randomized studies exist, they were conducted in healthy volunteers where the degree of hyperthermia is not as severe as that encountered in true heatstroke patients. Remaining data originate from observational studies, case series or case reports. A treatment algorithm and examples of common therapies appear in **Figures 2** and **3**.

Cooling Methods

Regardless of the classification, it is imperative that the treatment process is simple to initiate, rapidly available and implemented as soon as heat stroke has been identified (34). External cooling methods should begin immediately. In the prehospital setting, patients should be moved to the shade and active cooling initiated. Organized athletic events typically have educated staff monitoring participants for possible injury and heatstroke with cooling stations, ice sheets and equipment for cold-water immersion (CWI) therapy readily available (35). Multiple studies, associations and organizations recommend for patients suffering from EHS, that rapid cooling to temperature less than or equal to 40°C be achieved before transport to a hospital, embracing the concept of "Cool First, Transport Second" (35–37).

The gold standard treatment for EHS treatment has been ice or CWI to create cooling by conduction (38, 39). Circulation of the ice/cold water will further enhance the rate of cooling (38). Managing patients submerged in ice/cold water is challenging. Acquiring large enough containers, monitoring, access to patients to perform procedures, entry and egress from containers, environmental safety for potential electrical issues, and risk of slipping for both patients and staff are just some of the challenges with this method of cooling (40). One solution is the use of a body bag as the container, with the water/ice level to the patient's anterior axillary line, allowing for containment of water/ice and easier patient access (34, 41). EHS patients tolerate submersion in

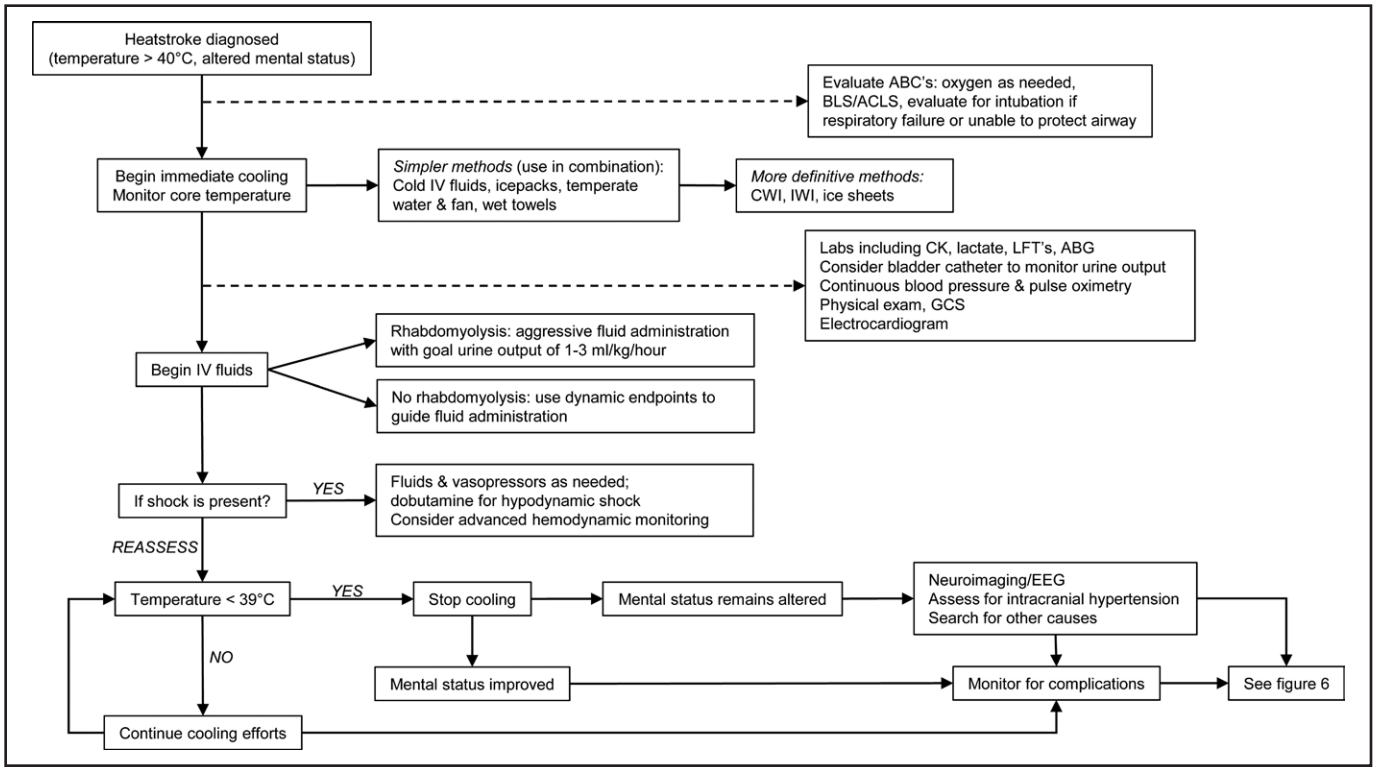

Figure 2. Generalized treatment approach for inpatient management. ABC = airway-breathing-circulation, ABG = arterial blood gas, ACLS = advanced cardiac life support, BLS = basic life support, CWI = cold-water immersion, CK = creatine kinase, EEG = electroencephalogram, GCS = Glasgow Coma Score, IWI = ice water immersion, LFT = liver function tests.

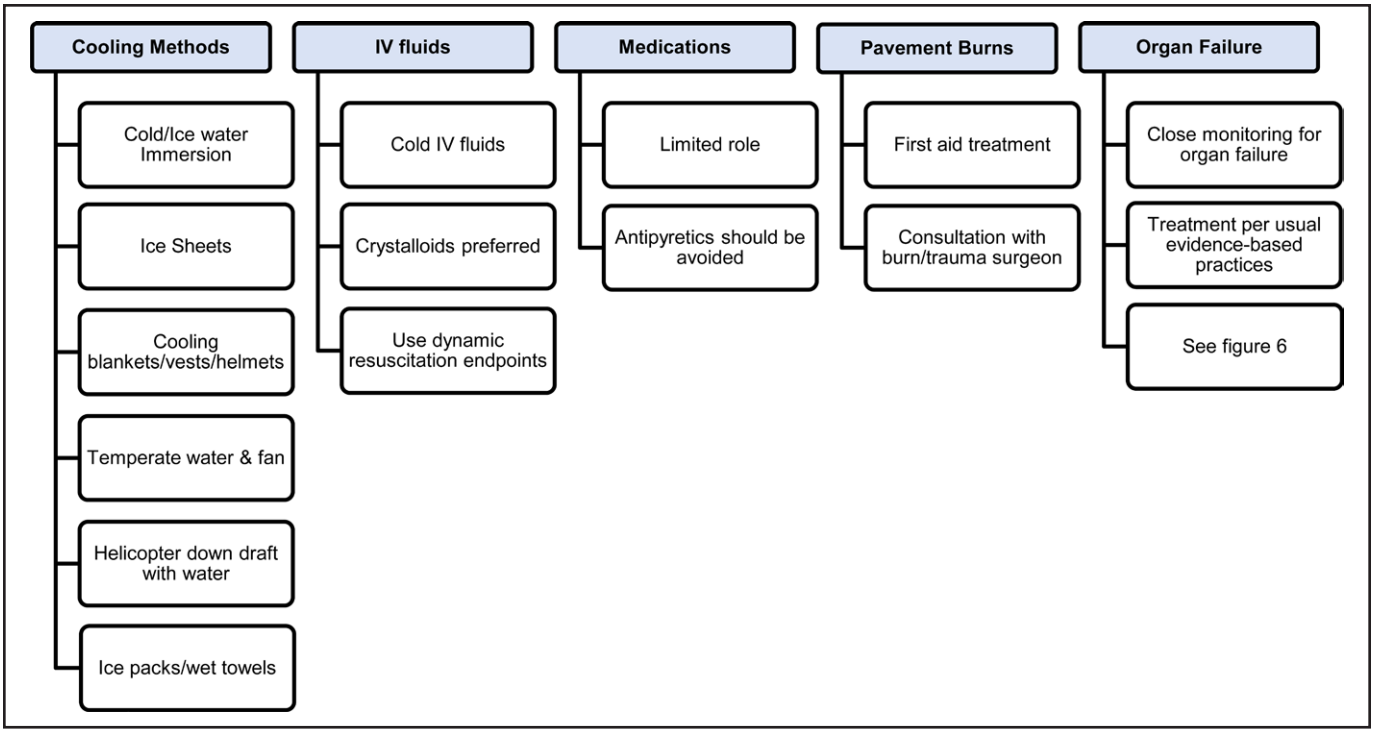

Figure 3. Summary of common therapies for heat-related illness and injury.

cold/ice water more readily than the usual patient who suffers from CHS. Nevertheless, given some of the difficulties with CWI, it is common to use several simpler cooling methods simultaneously (i.e., cold IV fluids plus cold water to wet patients plus fans blowing air to aid in evaporation). The key principle is to employ whatever means necessary to rapidly lower the patient's temperature to minimize end-organ damage.

Patients with CHS experience a more protracted rise in temperature; thus, multiple literature reviews have found that there is no standardized approach to cooling (15, 34, 38). Less stressful measures of conduction heat loss such as cooling blankets, cooling vests, cooling helmets, automated surface cooling devices, wetting sheets and clothing, and ice packs to palms and soles and areas of large vessels (groin and neck) should be used in these individuals (14). Evaporation and convection measures are also therapeutically effective, such as wetting the patient with cool or tepid water and using fans to blow air across the patient (15, 27, 38). These methods should be used in combination to facilitate rapid cooling. For evaporation on a larger scale or in remote settings, helicopter downdrafts with patients underneath being wet with water, have been used in both military and civilian settings (34). In patients whose temperature is not rapidly responding to therapy, invasive modalities, if available, should be readily considered and rapidly employed. However, cold-water irrigation of nasogastric/orogastric tubes or bladder irrigation is not recommended in isolation as these methods have shown only minimal changes in core body temperature with substantial time commitment on behalf of staff (17, 34). Case reports have documented the use of endovascular/intravascular cooling catheters to improve the rate of achieving goal temperature (40, 42–44). Extracorporeal circulation has also been shown to be effective for temperature management in patients with heat stroke (45, 46). Other practical aspects for implementing cooling therapies are listed in **Figure 4**.

The time frame for lowering core body temperature is ideally within 30 minutes. The literature has a range of initial target temperatures between 38° and 40°C. Exceeding a time of 60 minutes to reach initial target temperature is associated with adverse outcomes, including increased mortality (15, 17, 34, 36, 38). In a systematic review, McDermott et al (47) evaluated rates of cooling methods for lowering temperature in EHS patients. A rate of cooling at less than 0.078°C/min was determined to be unacceptable, a rate between 0.078°C/min and 0.154°C/min was considered acceptable, and a

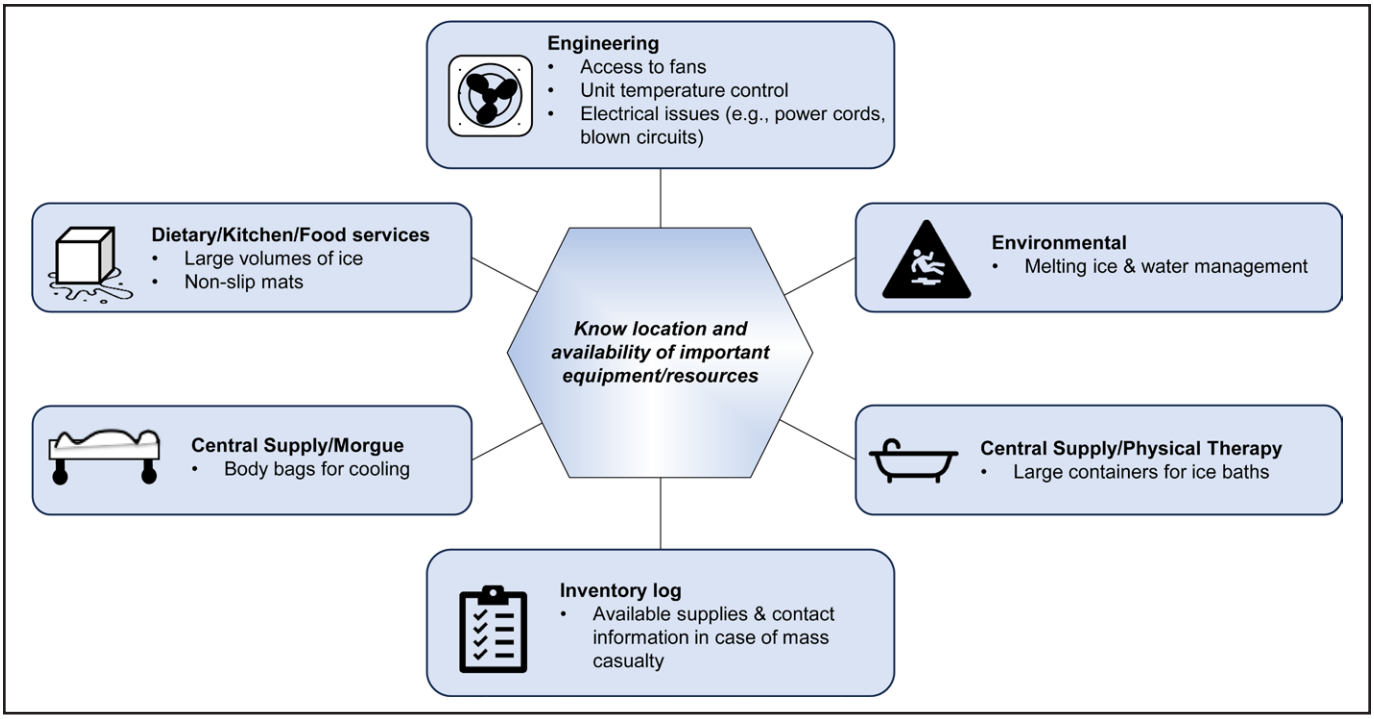

Figure 4. Practical considerations when constructing a heatstroke protocol.

rate of greater than or equal to 0.155°C/min was ideal. A systematic review of 521 patients by Filep et al (36) determined that a cooling rate of greater than 0.15°C/min for EHS yielded 100% survival. Survivors who were cooled at an inadequate rate had a 4.57 times risk of medical complications. A recent study by DeGroot et al (48) demonstrated the effectiveness of ice sheets (bed sheets soaked in ice water) as a possible alternative for EHS when CWI is not available. A cooling rate of 0.16°C/min was achieved, which is comparable to CWI. Examples of cooling rates for several different methods are included in **Figure 5** (17, 47).

An accurate temperature is imperative for diagnosing and managing patients with heatstroke. Tactile, axillary, tympanic, infrared, temporal, and oral methods are frequently inaccurate, especially once therapy has been initiated. Every effort should be made to initiate core temperature monitoring as soon as possible (15, 27). A small

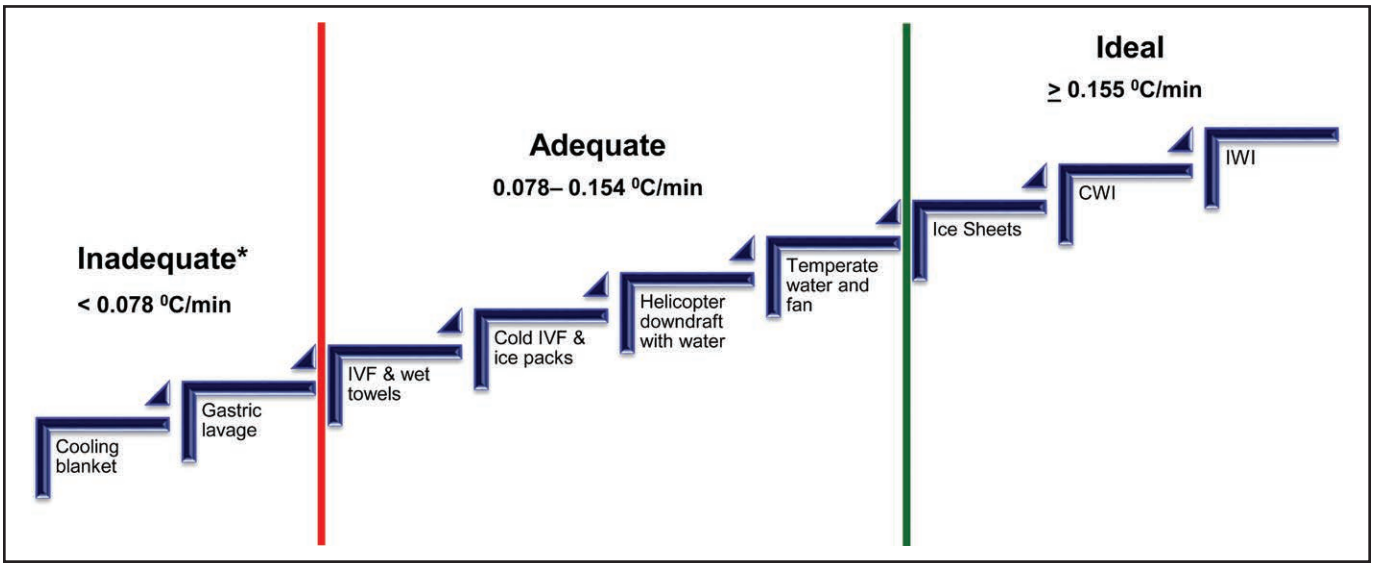

Figure 5. Cooling methods and rates of cooling. *Cooling strategies categorized as inadequate should not be used alone but combined with other methods of cooling. CWI = cold-water immersion, IVF = IV fluids, IWI = ice water immersion.

prospective pilot study by Yokobori et al (49) suggested that intravascular temperature management may have greater utility in obtaining more rapid temperature lowering with less variation than using traditional core temperature measurements such as esophageal, bladder, or rectal methods. Furthermore, a significant decline in Sequential Organ Failure Assessment scores was noted with no difference in adverse events compared with patients who used traditional monitoring techniques.

Fluid Therapy

Heat-related illness can be associated with hypovolemia secondary to excessive sweating, loss of salt and dehydration. Additionally, redistribution of blood flow to the skin to increase heat dissemination can decrease preload and compromise perfusion to vital organs. Circulatory failure caused by distributive shock is common, and hypovolemia can be a contributing factor (38). Appropriate volume therapy is crucial, as hypotension necessitating vasopressor therapy has been associated with increased mortality (38).

Studies with fluid therapy that are specific to heat-related illness are sparse. Basic tenets used in distributive shock should apply. Fluid administration should be carefully monitored and adjusted using dynamic endpoints. Although there are no data specific to heat-related illness, point-of-care ultrasound may be beneficial to assess volume status and responsiveness. Hypovolemia can be mild in some cases and respond to as little as 1 L of IV crystalloid (50, 51). One study reported 65% of patients had central venous pressure values greater than or equal to 3 on arrival, and of those 27% were greater than or equal to 10 (51). Caution with aggressive fluid administration is warranted especially when right heart failure is detected. On the other hand, aggressive fluid delivery is indicated when creatine kinase (CK) is elevated, and concern for rhabdomyolysis exists. Advanced hemodynamic monitoring should be performed to minimize complications recognizing that cooling will lead to vasoconstriction and redistribution of blood flow.

Crystalloid fluids are preferred in patients with heat-related injury but there are no studies evaluating the specific type of fluid (i.e., normal saline vs. balanced salt solutions). In theory, normal saline may be preferred because of the prevalence of hyponatremia and hypochloremia due to sweat loss but prospective trials are necessary.

Cold IV solutions (4°C) have been widely described in the setting of cardiac arrest but trials evaluating their use in heat-related illness are limited. Most studies were performed in controlled environments with healthy volunteers (52–54). One randomized, crossover trial was conducted in volunteers with exercise-induced hyperthermia who were treated with cold IV saline or passive cooling (e.g., sat in the shade) (55). Cold IV saline was associated with more rapid cooling, but the rate was insufficient to be considered as a primary treatment strategy. When used in addition to other therapies, one retrospective study observed less morbidity with cold IV saline (56). Cold IV fluids can, therefore, be considered an adjunct to other cooling methods.

Drug Therapy

The role of medications to accelerate cooling and minimize organ damage is minimal. Dantrolene is used to manage malignant hyperthermia and has been considered in heat-related illnesses, given some overlap in pathophysiologic mechanisms. Dantrolene interferes with the release of calcium from the sarcoplasmic reticulum and can attenuate the heat generated through muscle contractions. The literature evaluating dantrolene, though, for heat-related illness is limited to animal studies, case reports and two small randomized controlled trials (57–61). In these trials, the effects of dantrolene on temperature control were mixed with neither study demonstrating an improvement in clinical outcome (57, 58).

Antipyretics (e.g., nonsteroidal anti-inflammatory drugs, acetaminophen) are widely used for fever control in the ICU but they are ineffective in patients with heat-related illness (62, 63). This is largely due to pathophysiologic differences and the mechanism by which these agents work. Furthermore, risks for added morbidity exist, which include coagulopathy, kidney, and hepatic injury.

Numerous experimental therapies exist that are aimed toward the inflammatory and coagulopathic response to heat-related illness (14). Animal studies, thus far, show promise but further research is required before these agents can be considered.

Heat-Related Injury From Pavement/Surface Burns

Hot ambient temperatures may also lead to heat-related cutaneous injuries. The three leading causes

of pavement burns are patients found lying on pavement—21.6% ("found down"), walking on pavement—21.6% (particularly diabetics, who have insensate feet), and mechanical falls—15.1% (64). Burns caused by contact with hot pavement/surfaces have an increased need for surgical excision, higher overall cost per percent burn, longer hospital stays and higher mortality than other types of burn injury (64). A single center study of 195 patients documented that 50.5% of those sustaining pavement burns required burn excision, and 35.9% required grafting (64).

Initial management of pavement/surface burns consists of cool tap water irrigation for 20 minutes, airway assessment, and fluid resuscitation. A thorough evaluation for the cause of the fall and associated injuries should be performed. Patients may develop extremity compartment syndrome or rhabdomyolysis from heat-related muscle damage, edema, or pressure injuries. If extremity compartment syndrome is suspected or rhabdomyolysis develops, a qualified burn/trauma surgeon should immediately evaluate the patient for muscle compartment fasciotomy. Wounds should be dressed in the appropriate antimicrobial dressing. Although the definitive treatment of full thickness (third-degree) burns is excision and grafting, the optimal timing of burn excision in hot pavement burns, in which the extent is not always clearly defined at the time of injury, has not been identified. Last, patients should be monitored for cutaneous damage secondary to cooling therapy (i.e., frostbite) and immobility (i.e., pressure sores).

Treatment of Complications

Multisystem organ failure, presenting as shock, acute renal failure, encephalopathy, cerebral edema, acute respiratory distress syndrome, disseminated intravascular coagulation, heart failure, intestinal ischemia, and liver failure, is a major complication of heat-related illness. The treatment of these complications, particularly when it involves neurologic and organ dysfunction, requires a multidisciplinary approach to address specific issues and manage the patient's overall condition.

The CNS is particularly vulnerable to excessive heat; therefore, neurologic dysfunction is a hallmark complication of heatstroke. Brain injury is typically concentrated in the cerebellum but other areas can be affected (e.g., hippocampus, midbrain, thalamus) (14). As temperatures rise, permeability of the blood–brain barrier increases exposing the brain to systemic and intestinal toxins. Intracranial hypertension, cerebral edema, and decreased cerebral blood flow may ensue. Appropriate therapies, aimed toward optimizing cerebral hemodynamics, should be implemented if cerebral hypoperfusion is suspected. Seizures are common, which should be managed with benzodiazepines.

Acute kidney injury (AKI) occurs in roughly 25–35% of patients with a higher prevalence in patients with EHS (65, 66). Although the cause of AKI can be multifactorial, rhabdomyolysis is commonly encountered especially in patients with EHS. Aggressive fluid therapy should be provided to maintain an adequate urine output and resolution of CK concentrations. Other causes of AKI include renal hypoperfusion, tubular necrosis, and direct thermal injury (14). Continuous renal replacement therapy may be beneficial (67).

Cardiovascular collapse can occur because of hypovolemia, vasoplegia, and redistribution of blood flow. Studies have predominantly shown hemodynamic values consistent with distributive shock but hypodynamic shock has also been reported (38). Dobutamine may be useful in the latter. Hypotension requiring the need for vasopressors has been associated with higher mortality and poor neurologic recovery (38).

Coagulation disorders can range from subclinical coagulation and fibrinolysis to widespread microthrombosis and fatal hemorrhage. Coagulopathy may worsen in the first few days after cooling (66). Disseminated intravascular coagulation, resulting from thermal injury to the endothelium, has been reported in up to 45% of patients with heatstroke and is a noted factor leading to mortality (18, 19). In fact, postmortem evaluations have reported end-organ failure is primarily due to heat-induced necrotic and apoptotic cell death, widespread microthrombisis, hemorrhage, and inflammation (15).

Clinical management should prioritize prevention of multiple organ dysfunction as the options for treatment beyond cooling techniques are limited and directed toward supportive care. A summary of ICU management appears in **Figure 6**. Upon recovery, clinicians must assure patients can be discharged to a safe environment to minimize complications or

BOX 1. RESEARCH GAPS

- In classic heat stroke, what are the best methods to achieve rapid cooling?
- What is the best water temperature to use for immersion therapy?
- When should cooling efforts cease?
- What is the role of novel cooling devices (e.g., endovascular, intranasal cooling devices, blankets engineered from heat-conductive biomaterials, etc.)
- How should cooling therapies be prioritized across large patient cohorts when mass-casualties occur?
- What are the best cooling therapies to use in mass casualty situations?
- What is the best approach for treatments in resource limited regions?
- What is the role of immunomodulators to treat the inflammatory response?
- What is the role of anticoagulant medications?
- What is the role of biomarkers to identify organ failure and prognosticate outcomes?
- What is the role of artificial intelligence to create a system that creates rapid, predictable cooling and minimize shivering?
- Can precision medicine (e.g., genotypical profiles) be used to identify patients at higher-risk for heat-related illness or severe disease?
- What are the long-term outcomes for patients with heat-related injuries?
- What is the optimal timing for excision and grafting of heat-related pavement burns?

readmission. Long-term rehabilitation, including physical therapy, occupational therapy, and neuropsychologic support, may be required to address residual deficits or complications.

Prognosis

Mortality rates in patients requiring ICU care for heatstroke approach 60% and long-term complications, such as inability to return to work or severe functional limitations, range between 3.7% and 40.7% (14, 15, 68). Approximately 30% of heatstroke survivors experience some form of cognitive or motor dysfunction, many of which are permanent (14, 19). Recovery of neurologic function during cooling is a positive prognostic indicator (18). In fact, if treatment is prompt, symptoms may abate after several days emphasizing the importance of aggressive cooling. Prognosis is worse if organ dysfunction persists beyond 96 hours (15).

Other prognostic indicators have been identified to predict which patients have the greatest risk of mortality (4, 64, 69–85). The most established include advanced age, maximum temperature, duration of hyperthermia, cooling rate, hypotension, and presence of organ dysfunction (68) (**Fig. 7**). Scoring systems exist (e.g., Early Risk Assessment Tool for Detecting Clinical Outcomes in Patients with Heat-related Illness [J-ERATO] score) for the identification of adult patients with higher risk of heat-related hospitalization and in-hospital mortality (86). Biomarkers may play a role in identifying patients at risk for death or neurologic damage (15). Further research is needed.

Research Gaps

Despite decades of research aimed toward prevention and treatment of heat-related illnesses, there are several research questions that require attention (**Box 1**). Most data are either derived from case series, small retrospective studies, or extrapolations from alternative research models, given the ethical challenges of conducting studies in this environment. Furthermore, optimal strategies in resource-limited areas are not clearly established. These gaps represent a blueprint for future studies to better guide decision-making and evidence-based practice.

CONCLUSIONS

The prevalence of heat-related illness and injury is increasing, and rapid initiation of appropriate therapies is necessary to optimize outcomes. The foundation of treatment consists of early identification of heatstroke, rapid cooling, and appropriate critical care therapies for the maintenance of organ support. High-level evidence, however, is sparse. Complications of

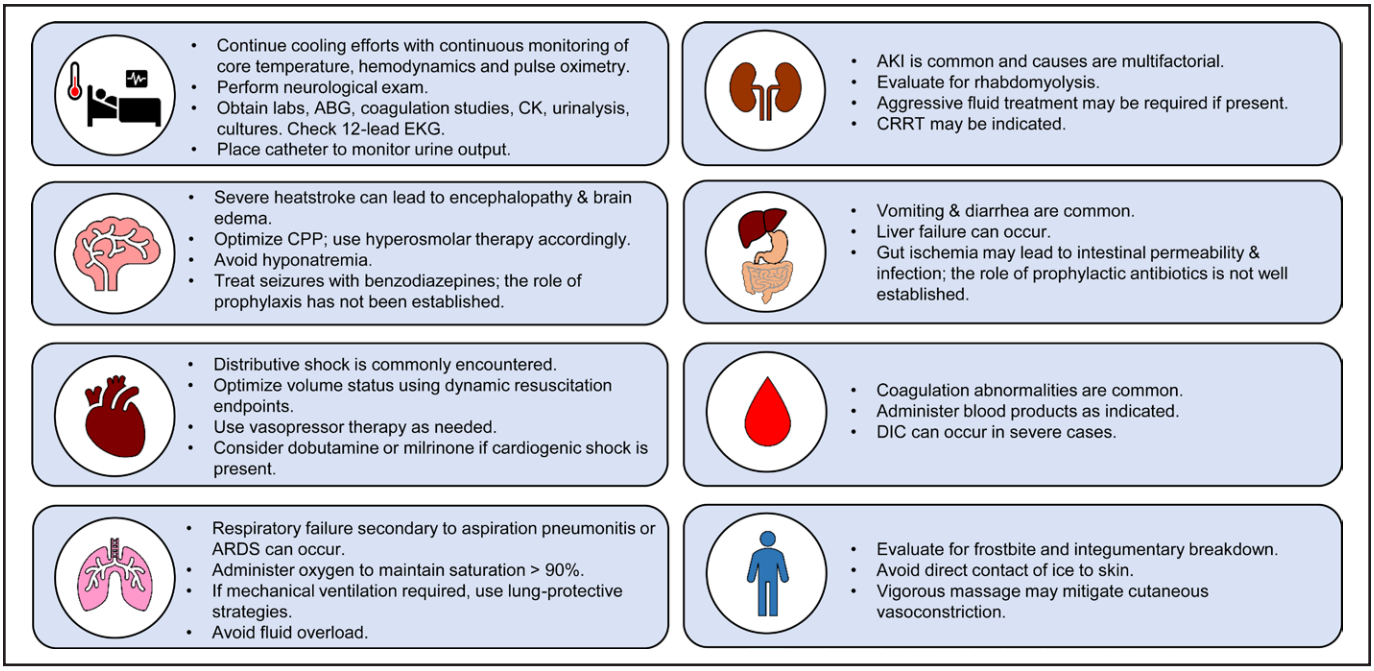

Figure 6. Overview of ICU management. ABG = arterial blood gas, ARDS = acute respiratory distress syndrome, AKI = acute kidney injury, CK = creatine kinase, CPP = cerebral perfusion pressure, CRRT = continuous renal replacement therapy, DIC = disseminated intravascular coagulation, EKG = electrocardiogram.

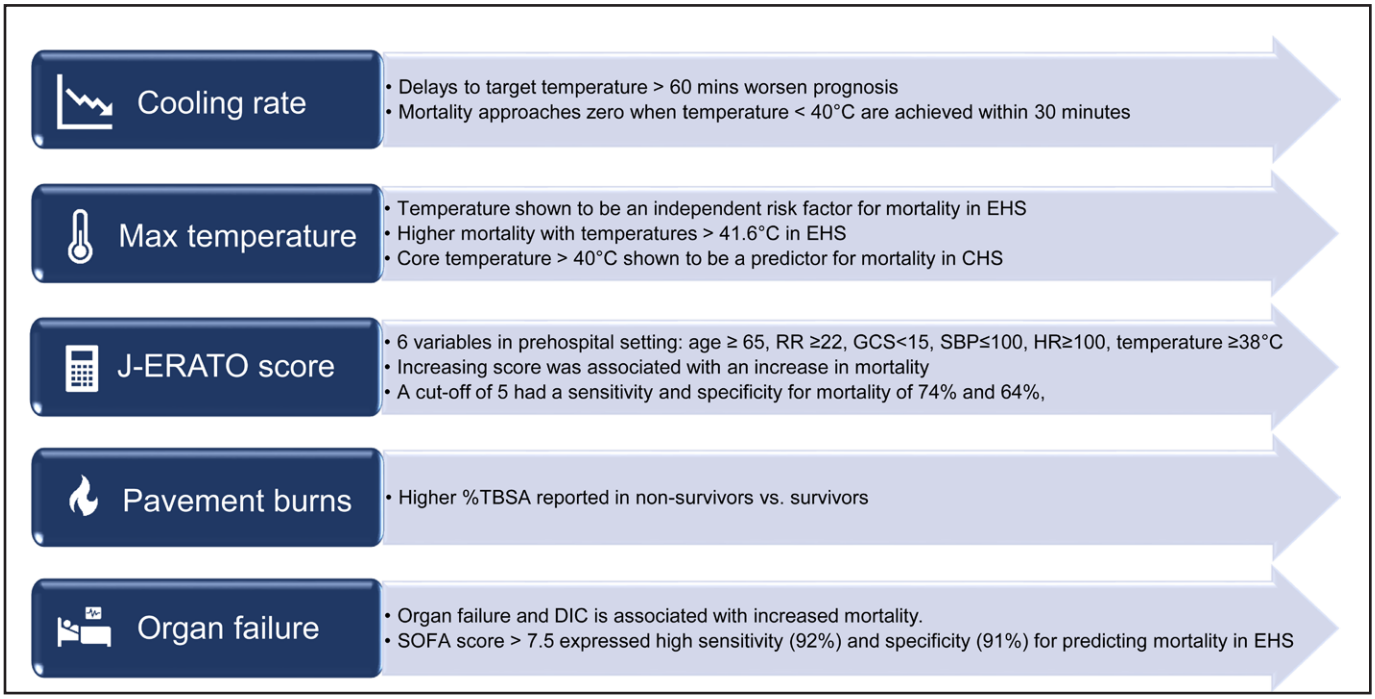

Figure 7. Prognostic indicators in heat-related illness. CHS = classic heat stroke, DIC = disseminated intravascular coagulation, EHS = exertional heat stroke, GCS = Glasgow Coma Score, HR = heart rate, J-ERATO score = Early Risk Assessment Tool for Detecting Clinical Outcomes in Patients with Heat-related Illness, RR = respiratory rate, SBP = systolic blood pressure, SOFA = sequential organ failure assessment. Data from references: (25, 36, 77, 83, 86–90).

heat-related illness can impact practically every organ system; prompt recognition and intervention are crucial determinants for survival.

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