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In the Clinic®

Heat-Related Illnesses

Climate change is anticipated to continue to adversely affect public health, with heat stress the predominant threat. Accordingly, heat-related illness is predicted to increase as extremely hot days become more frequent. Heat stroke, the most serious heat-related illness, is a medical emergency that may be fatal if it is not promptly recognized, addressed with early and rapid cooling, and accompanied by multidisciplinary supportive care as clinically indicated. Heat stroke is a preventable illness that occurs in 2 distinct forms–classic and exertional–that have distinct demographic profiles and clinical courses but similar management paradigms.

Recognition and Prevention

Diagnosis

Management

Practice Improvement

CME/MOC activity available at Annals.org.

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Recognition and Prevention

What is heat-related illness, and why is it important?

Biological processes depend on a narrow temperature range for optimal function. Humans maintain a relatively constant body temperature by generating heat internally through increased metabolism with exercise while dissipating heat through evaporative and nonevaporative mechanisms. However, global warming is expected to challenge the limits of thermoregulation with more extreme heat (1). Although moderate heat load may promote physiologic acclimation, excessive load can increase morbidity and mortality, particularly in active populations (2, 3). Environmental heat and related illnesses account for more weather-related deaths than all other exposures combined, including hurricanes, tornadoes, floods, and earthquakes (4).

Heat-related illness can be classified as minor or serious (5–7). Minor disorders, including exercise-associated muscle cramps (EAMC), heat syncope, heat edema, and miliaria rubra, are well described and generally self-limited. Serious heat illness (SHI), the focus of this article, includes heat exhaustion (HE), heat injury (HI) (HE with clinical evidence of injury to vital organs), and heat stroke (HS). HS occurs in 2 forms: exertional (EHS) and classic (CHS). CHS results from passive exposure to high temperatures and humidity, whereas EHS typically occurs during vigorous exercise in hot or temperate environments. In addition, CHS and EHS tend to have distinct demographic profiles. CHS occurs in prepubertal, elderly, and compromised persons (for example, those experiencing homelessness or those with severe mental illness), largely while they are engaged in sedentary activity during heat waves. Persons with CHS frequently have underlying comorbidities with chronic illnesses, which are complicated by use of multiple medications or alcohol or drug dependency. Persons with EHS tend to be young, generally healthy postpubertal people participating in vigorous exercise or activity. EHS may occur at any time of the year but primarily occurs in warm or hot months (8).

How does heat cause health problems?

Environmental heat results in predictable and well-described patterns of physiologic strain that the body can compensate for and ultimately adapt to, but it may cause heat-related illness (3, 6). The primary physiologic response to heat stress is an increase in cardiac output to the skin (for thermoregulation) and muscle (for work), with compensatory decreases in renal and splanchnic blood flow (3). High sweat losses can induce dehydration if not fully replaced, which further elevates cardiovascular and thermal strain. If physiologic strain is not excessive, multiple heat exposures will stimulate heat acclimation and acquired thermal (heat) tolerance (9, 10). If physiologic strain is excessive, a series of pathologic events can develop, resulting in HI or HS. Physiologic strain disturbances include an increase in gut permeability with endotoxemia, decreased cerebral blood flow resulting in central nervous system (CNS) injury, decreased mesenteric blood flow and resultant acute liver and kidney injury, and an exaggerated systemic inflammatory response with concomitant coagulopathy and subsequent cell death (8). Heat waves (a period of abnormally hot weather lasting >2 days) are strongly associated with increased emergency department (ED) visits and hospitalizations (11).

A retrospective study linked databases to identify cause-specific Medicare hospitalizations on heat wave days over 10 years. The authors identified 11 diagnoses with a higher admission risk on heat wave days, including HS and sunstroke (relative risk [RR], 22.5 [95%

1. Zhao Q, Guo Y, Ye T, et al. Global, regional, and national burden of mortality associated with non-optimal ambient temperatures from 2000 to 2019: a three-stage modelling study. Lancet Planet Health. 2021;5:e415-e425. [PMID: 34245712]
2. Pal Lubinsky H, Blondin DP, Jay O. A double-edged sword: risks and benefits of heat for human health. Trends Endocrinol Metab. 2024;35:277-279. [PMID: 38593784]
3. Sawka MN, Leon LR, Montain SJ, et al. Integrated physiological mechanisms of exercise performance, adaptation, and maladaptation to heat stress. Compr Physiol. 2011;1:1883-1928. [PMID: 23733692]
4. Luber G, McGeehin M. Climate change and extreme heat events. Am J Prev Med. 2008;35:429-435. [PMID: 18929969]
5. Headquarters, Department of the Army. Heat Stress Control and Heat Casualty Management. Technical Bulletin, Medical. TB MED 507. 12 April 2022. Accessed at www.govinfo.gov/content/pkg/GOVPUB-D101-PURL-gpo216616/pdf/GOVPUB-D101-PURL-gpo216616.pdf on 13 May 2025.
6. Roberts WO, Armstrong LE, Sawka MN, et al. ACSM expert consensus statement on exertional heat illness: recognition, management, and return to activity. Curr Sports Med Rep. 2023;22:134-149. [PMID: 37036463]
7. O'Connor FG, Nye NS, DeGroot D, et al. Clinical Practice Guideline for the Prevention, Diagnosis, and Management of Exertional Heat Illness. Consortium for Health and Military Performance (CHAMP); June 2024.
8. Bouchama A, Knochel JP. Heat stroke. N Engl J Med. 2002;346:1978-1988. [PMID: 12075060]
9. Taylor NA. Human heat adaptation. Compr Physiol. 2014;4:325-365. [PMID: 24692142]
10. Horowitz M. Heat acclimation, epigenetics, and cytoprotection memory. Compr Physiol. 2014;4:199-230. [PMID: 24692139]
11. Vaidyanathan A, Gates A, Brown C, et al. Heat-related emergency department visits - United States, May–September 2023. MMWR Morb Mortal Wkly Rep. 2024;73:324-329. [PMID: 38635484]

CI, 14.9 to 34.2]), fluid and electrolyte disorders (RR for hyperosmolality, 1.4 [CI, 1.1 to 1.3]; RR for hypoosmolality, 1.2 [CI, 1.1 to 1.3]), and acute kidney failure (RR, 1.1 [CI, 1.1 to 1.2]) (12).

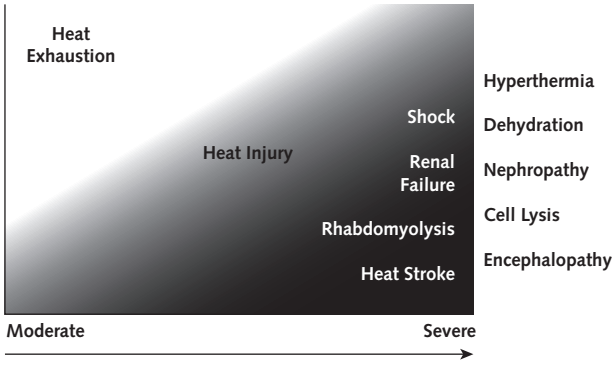
What is the scope of heat-related illnesses?

SHI is most appropriately described as consisting of a spectrum of disorders (**Figure**) (5–7). The diagnostic conditions of HE, HI, and HS often share overlapping features that blur their interpretation as discrete disorders with their own distinct pathogeneses. Importantly, the disorders do not necessarily function as a continuum, with a direct line from HE to HI and then HS; although patients may progress, they may also abruptly present with the profound collapse of HS. Any patient presenting with a clinical picture suggesting a potential SHI should be assumed to have HS and emergently and actively cooled until a more thorough evaluation can be done and a final diagnosis can be made.

HE is the inability to sustain the required cardiac output and blood pressure to continue physical activity due to high skin blood flow requirements and/or dehydration related to heat stress. Body temperature is elevated by the metabolic heat produced during exercise but is usually below 40 °C (104 °F). HE has no chronic health effects, and after body cooling and restoration of fluid–electrolyte balance, most patients can return to activity in 1 to 2 days. HI is characterized by evidence of organ (for example, gastrointestinal, kidney, liver, muscle) damage and dysfunction in the presence of hyperthermia without the CNS changes seen in EHS and requires formal laboratory testing to establish the diagnosis. The exact clinical pathway to tissue or organ injury is unknown, but it may result from dehydration, reduced blood flow, or a direct thermal injury during a more severe HE episode. HI typically causes tissue and organ dysfunction that may persist for several weeks, including acute kidney injury (AKI), transient diarrhea (gut injury), and/or transaminitis associated with liver injury (13).

HS is defined as a life-threatening condition characterized by CNS disturbances and hyperthermia, usually with a rectal temperature above 40 °C. The term "heat stroke," which implies the presence of focal "stroke-like" symptoms associated with hyperthermia, is misleading as the clinical presentation in most patients is more global (encephalopathic) than focal. CNS changes associated with HS vary from mild personality deviations to confusion, delirium, stupor, or unconsciousness; seizure activity can be observed. In addition to

Figure. **Spectrum of exertional heat illness with associated categories of physiological dysfunction.**



Reproduced from Headquarters, Department of the Army, Heat Stress Control and Heat Casualty Management, Technical Bulletin, Medical, TB MED 507, 12 April 2022 (www.govinfo.gov/content/pkg/GOVPUB-D101-PURL-gpo216616/pdf/GOVPUB-D101-PURL-gpo216616.pdf), with permission.

12. Hopp S, Dominici F, Bobb JF. Medical diagnoses of heat wave-related hospital admissions in older adults. Prev Med. 2018;110:81-85. [PMID: 29428173]
13. Ward MD, King MA, Gabrial C, et al. Biochemical recovery from exertional heat stroke follows a 16-day time course. PLoS One. 2020;15:e0229616. [PMID: 32130237]

CNS dysfunction, HS is almost always associated with a body temperature above 40°C as well as signs and symptoms of cardiovascular and other organ system distress. Organ and tissue damage can occur with prolonged hyperthermia, but the damage may not be clinically evident until later in the disease process. The current accepted definition of HS, first proposed by Bouchama and Knochel in 2002, is "a form of hyperthermia associated with a systemic inflammatory response leading to a syndrome of multiorgan dysfunction in which encephalopathy predominates" (8).

Who is at risk for heat-related illnesses in the United States, and what are the risk factors for poor outcomes?

Intrinsic (for example, individual susceptibility) and extrinsic (for example, environmental conditions and sociocultural factors) risk factors contribute to heat-related illness; these factors represent cornerstone efforts for targeted risk mitigation strategies. Although EHS and CHS share many risk factors, they have distinct demographic patterns. Table 1 presents a summary of common intrinsic and extrinsic risk factors associated with both EHS and CHS.

Health care disparities are increasingly recognized as a critical risk factor in the United States, with significant morbidity and mortality. Although HS occurs with equal prevalence in African Americans and White persons, African Americans have increased morbidity and mortality. A recent review identified race, ethnicity, income, education, occupation, access to air conditioning at home, and neighborhood characteristics (including crime) as affecting individual vulnerability to heat-related illness (14).

Finally, although chronic disease is a risk factor for SHI prognosis, environmental heat stress also results in significant physiologic strain that exacerbates underlying disease.

In a case-only retrospective analysis involving review of medical records for nearly 11 million deaths, persons with preexisting conditions were more vulnerable to extreme heat, with a 6% (CI, 4% to 8%) increase in mortality on

14. Gronlund CJ. Racial and socioeconomic disparities in heat-related health effects and their mechanisms: a review. Curr Epidemiol Rep. 2014;1:165-173. [PMID: 25512891]

Table 1. **Risk Factors for Classic and Exertional Heat Stroke***

Classic Heat Stroke	*Exertional Heat Stroke*
Intrinsic risk factors (individual susceptibility)	
Chronic disease: Obesity and cardiovascular, cerebrovascular, endocrine, neurologic, mental, and pulmonary illnesses	
Recent or concurrent viral and/or bacterial infection	
Long-term medication use: β-Blockers, diuretics, calcium-channel blockers, laxatives, anticholinergic drugs, salicylates, thyroid agonists, benztropine, trifluoperazine, butyrophenones, α-agonists, monoamine oxidase inhibitors, sympathomimetic medications, tricyclic antidepressants, SSRIs	
Drug misuse: Amphetamines and amphetamine-like agents (e.g., ephedra), MDMA, cocaine, PCP, LSD, synthetic stimulants of the cathinone class (e.g., α-PHP), alcohol	
Elderly	Overmotivation
Prepubertal	Peer/coach pressure
Social isolation	Functional disorders: Lack of acclimation, poor physical fitness
Inability to care for oneself	Congenital disorders: Anhidrosis, ectodermal dysplasia, RYR1 myopathy
Confinement to bed	Acquired disorders: Dehydration, sleep deprivation, sweat gland dysfunction (e.g., deep burns, scarred skin on >40% of total body surface area)
	Prior heat illness
Extrinsic risk factors (environmental and sociocultural)	
Heat waves	
High humidity, high thermal radiation, low wind speed (high heat index)	
Air pollution	
Socioeconomic and racial health care disparities	
Unventilated/non–air-conditioned living space	Protective clothing

LSD = lysergic acid diethylamide; MDMA = 3,4-methylenedioxymethamphetamine; PCP = phencyclidine; PHP = pyrrolidinohexiophenone; SSRI = selective serotonin reuptake inhibitor.
** Information from references 5 and 19.*

extremely hot days in those with a prior hospitalization for atrial fibrillation, an 8% (CI, 4% to 12%) increase in those with Alzheimer disease, and a 6% (CI, 3% to 9%) increase in those with dementia. ZIP code–level and personal characteristics were also associated with increased susceptibility to temperature (15).

What is the role of prevention in managing heat-related illness?

A comprehensive public health and clinical approach is needed to prevent heat-related illness. Effective management of SHI requires a functional "chain of survival," similar to the American Heart Association's paradigm for out-of-hospital cardiac arrest. The chain of survival for SHI includes 5 linked steps: prevention, prehospital management, emergency medical transport, advanced clinical management in a medical treatment facility, and finally return to activity (5, 7). Communication and speed are critical, as the morbidity and mortality associated with SHI have repeatedly been shown to depend on the duration of hyperthermia ("it's not how hot the patient gets, but how long the patient has been hot" [16]).

In a systematic review of 521 patients, no patients died when treatment involved an adequate cooling rate, whereas 23 (4.4%) died with inadequate cooling. One hundred seventeen patients (22.5%) survived with medical complications when treatment involved an insufficient cooling rate, whereas only 4 (0.77%) had complications when there was adequate cooling. Cooling rates faster than 0.15 °C (0.27 °F) per minute were associated with survival of patients with EHS without medical complications (16).

What are strategies for prevention of heat-related illness?

Strategies for prevention of SHI are tailored to the population at risk, with specific recommendations for those who work or exercise in the heat (EHS risk) versus those who may be sedentary during periods of prolonged exposure (CHS risk) (17). Clinicians have the responsibility to advise not only at-risk patients but also individuals, leaders, and organizations that have an opportunity to mitigate risk for others (such as coaches, trainers, family caregivers, and city planners) (18).

Prevention of EHS requires an integrated public health approach that focuses on both the individual and the greater community. Advice on adjustments in workload, clothing, hydration, training, and work schedules should match individual fitness levels, heat acclimation status, and changing environmental heat loads. In preventing CHS, clinicians, family members, and community leaders need to be particularly alert to anticipated heat waves and individual patient vulnerabilities. Valuable prevention strategies include early warning systems, air conditioning at home or in designated locations, home use of fans or misting, decreasing exertion, and engaging friends or family to assess those who are at risk to ensure their well-being (18–20). Appendix

Table 2. **Common Signs and Symptoms of Serious Heat-Related Illness**

Signs
Vital signs
Weak or rapid pulse
Hyperthermia
Hypotension
Skin/muscles
Sweaty or dry skin
Warm or cool to touch
Pale or flushed
Flaccid, rigid, or visible fasciculations
Symptoms
Central nervous system
Fatigue, weakness
Headache
Altered gait
Combative, aggressive
Personality change, delirium, coma
Gastrointestinal
Nausea, vomiting
Diarrhea, stool incontinence

15. Zanobetti A, O'Neill MS, Gronlund CJ, et al. Susceptibility to mortality in weather extremes: effect modification by personal and small-area characteristics. Epidemiology. 2013;24:809-819. [PMID: 24045717]
16. Filep EM, Murata Y, Endres BD, et al. Exertional heat stroke, modality cooling rate, and survival outcomes: a systematic review. Medicina (Kaunas). 2020;56:589. [PMID: 33167534]
17. Jacklitsch B, Williams WJ, Musolin K, et al. NIOSH criteria for a recommended standard: occupational exposure to heat and hot environments. DHHS (NIOSH) Publication No. 2016-106. National Institute for Occupational Safety and Health; February 2016.
18. Sorensen C, Hess J. Treatment and prevention of heat-related illness. N Engl J Med. 2022;387:1404-1413. [PMID: 36170473]
19. Epstein Y, Yanovich R. Heatstroke. N Engl J Med. 2019;380:2449-2459. [PMID: 31216400]
20. Bouchama A, Abuyassin B, Lehe C, et al. Classic and exertional heatstroke. Nat Rev Dis Primers. 2022;8:8. [PMID: 35115565]

Table 1 (available at Annals.org) identifies prevention strategies for both EHS and CHS.

How should physicians advise caregivers?

SHI can present with a wide range of signs and symptoms. Caregivers should have a high index of suspicion for HS with a low threshold for assessing rectal temperature, as HE can routinely occur at lower core temperatures if the skin is warm (3). Serious EHS or CHS does not necessarily present with profound collapse; a subtle personality change or gait disturbance may be an initial diagnostic clue. In addition, cognitive dysfunction may not permit patients to assess their own decline. Accordingly, caregivers, training staff, or an assigned activity "buddy" need to be alert for changes that prompt a medical assessment by an appropriate provider. Table 2 identifies common signs and symptoms of SHI.

Recognition and Prevention... Serious heat illness includes heat exhaustion, heat illness (heat exhaustion with clinical evidence of injury to vital organs), and heat stroke (which includes CNS dysfunction). Classic and exertional heat stroke have different demographic profiles. Prevention begins with identification of the population at risk and a detailed assessment of intrinsic and extrinsic risk factors. Prevention requires a team approach with involvement of patients, clinicians, supervisors, trainers, coaches, caregivers, administrators, and leaders.

CLINICAL BOTTOM LINE

Diagnosis

What are clinical signs and symptoms to look for on the history and physical examination?

SHI is a clinical diagnosis. In HS, the clinical triad of hyperthermia, evidence of CNS dysfunction, and the setting of either hot weather (CHS) or physical exertion (EHS) is core to the diagnosis (19). The focus of the initial history, therefore, is to ascertain the details of the heat exposure (for example, exertional stress or prolonged exposure) and observed changes in function with particular attention to the CNS. Changes in the patient's CNS status can range from relatively minor, with reports of dizziness or headache, to profound presentations that include confusion, combativeness, delirium, and coma. Consistent with the nature of the CNS dysfunction, the history is frequently obtained from family members, bystanders, or caregivers who witnessed the initial event. The remaining focus of the history is to assess the patient's medical history, including comorbidities, medications, and supplements, and perform a pertinent review of systems. Clinical clues (for example, history of diabetes or cardiovascular disease; history of baseline CNS dysfunction, such as autism, seizures, or recent infection; use of new medications; nutritional or hydration strategies) can assist the provider with the differential diagnosis and initial management.

The initial physical examination in a patient with suspected SHI should start with basic life support recommendations of sequentially assessing responsiveness, airway and breathing, and presence of a pulse (21). Absence of a pulse should immediately move the provider to a cardiac arrest algorithm with actions as appropriate to sustain life; otherwise, the assessment of vital signs should continue with blood pressure and temperature measurement. Patients with SHI are frequently but not always tachypneic, tachycardic, and hypotensive. In the setting of SHI, a rectal temperature evaluation is the only valid and practical method of core body

21. Greif R, Bray JE, Djärv T, et al. 2024 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations: summary from the Basic Life Support; Advanced Life Support; Pediatric Life Support; Neonatal Life Support; Education, Implementation, and Teams; and First Aid Task Forces. Circulation. 2024;150:e580-e687. [PMID: 39540293]

temperature assessment (22, 23). Aural, oral, tympanic, axillary, and temporal measurements can be invalid, particularly in persons with warm skin or those exercising in the heat (22). Proper measurement of rectal temperature with a thermistor requires an insertion depth of 15 cm (24). The remainder of the physical examination should focus on establishing the patient's baseline and carefully assessing for a secondary cause (such as cellulitis or occult pneumonia) or complications (for example, petechia or bloody diarrhea consistent with disseminated intravascular coagulation [DIC]). Examination of the skin can be helpful; profuse sweating and wet skin are typical of EHS, whereas the skin is often dry in CHS. Warm and flushed skin may reflect excessive peripheral vasodilation, whereas cool and pale skin may indicate vascular collapse. Finally, the clinician should assess and document the patient's initial mental status using a tool such as the Glasgow Coma Scale.

What else should clinicians consider in the differential?

Appendix Table 2 (available at Annals.org) includes a list of differential diagnoses for patients with brain dysfunction and hyperthermia. However, differential diagnoses should be considered only after HS is ruled out and emergent cooling is initiated, as delay can have catastrophic consequences. Categories of disorders that clinicians should consider in the differential diagnosis include but are not limited to infectious disease; metabolic, water, and electrolyte disorders; cerebrovascular disease; seizures; and intoxications (Appendix Table 2). Clinicians should remember that although these disorders are in the differential diagnosis, they can also be comorbid with the HS presentation.

What is the role of other laboratory or imaging studies in making the diagnosis?

There is currently no specific biomarker or clinical imaging study to diagnose HS (20). However, the natural history of HS has shown a predictable 3-phase clinical pattern: a hyperthermic–neurologic acute phase, a hematologic–enzymatic phase that peaks 24 to 48 hours after the event, and a late renal–hepatic phase if clinical symptoms are sustained for 96 hours or longer (19). These patterns, more clearly identified in EHS, highlight 2 important observations: 1) HS, particularly when cooling is delayed, is potentially a multiorgan critical illness, and 2) clinicians must not be misled by early normal laboratory values and should be alert for subsequent deterioration. Some biomarkers have been associated with HS (10, 25, 26). Appendix Table 3 (available at Annals.org) identifies common clinical scenarios encountered in the management of HS, with suggested potential laboratory studies and adjunctive tests that may be used.

Diagnosis... Clinical presentation of heat-related illnesses can initially be nonspecific, with potential multiorgan dysfunction in later stages if the patient is not rapidly cooled. Core diagnostic features include the clinical triad of hyperthermia, evidence of CNS dysfunction, and the setting of either hot weather (CHS) or physical exertion (EHS). Differential diagnoses should be considered only after heat-related illness has been ruled out. Conditions in the differential may also be comorbid with heat-related illness.

CLINICAL BOTTOM LINE

22. Casa DJ, Becker SM, Ganio MS, et al. Validity of devices that assess body temperature during outdoor exercise in the heat. J Athl Train. 2007;42:333-342. [PMID: 18059987]
23. Casa DJ, DeMartini JK, Bergeron MF, et al. National Athletic Trainers' Association position statement: exertional heat illnesses. J Athl Train. 2015;50:986-1000. [PMID: 26381473]
24. Miller KC, Hughes LE, Long BC, et al. Validity of core temperature measurements at 3 rectal depths during rest, exercise, cold-water immersion, and recovery. J Athl Train. 2017;52:332-338. [PMID: 28207294]
25. Hashim IA. Clinical biochemistry of hyperthermia. Ann Clin Biochem. 2010;47:516-523. [PMID: 20926467]
26. Schlader ZJ, Davis MS, Bouchama A. Biomarkers of heatstroke-induced organ injury and repair. Exp Physiol. 2022;107:1159-1171. [PMID: 35654394]

Management

What is the approach to management of minor heat-related illness?

Minor heat-related illness includes EAMC, heat syncope, heat edema, and miliaria rubra (5). These conditions are common, can be recognized by their distinct clinical features, and are generally self-limited.

Muscle cramping is an unexpected, involuntary, and painful contraction of a single muscle or a muscle group that is relieved by stretching the cramping muscle. Although the cause is unknown, cramps are believed to arise from spontaneous ectopic discharges of motor nerves or the terminal branches of motor axons (27). EAMC, also called heat cramps, are a form of cramping directly associated with exercise, particularly in the heat in unacclimated persons (28). Cramping in an individual muscle is often preceded by palpable or visible fasciculations that generally last 2 to 3 minutes but can be prolonged. Diffuse cramping can occur with severe EAMC, which can also occur during recovery after prolonged, intense sweating. There is no specific treatment, but most authorities recommend passive static stretching of the affected muscle groups, cryotherapy, and hydration, which may include intravenous fluids in refractory cases (27, 28). Other than muscle soreness and minor muscle strain, no significant complications have been reported from EAMC; an episode of EAMC does not imply any predisposition to exertional heat illness (5). Acclimation, hydration, and dietary salt supplementation may reduce the incidence of EAMC in at-risk populations. Patients with recurrent cramping that interferes with performance should be considered for further evaluation by an appropriate specialist to rule out an underlying medical condition (5, 29).

Heat syncope, or parade syncope, is commonly seen in persons who are inadequately acclimated (military recruits, marching band artists), elderly persons, and those who are standing with minimal leg movement for venous return, who may be predisposed to postural hypotension (5, 30, 31). Heat syncope is believed to result from a temporary circulatory insufficiency due to pooling of blood in the peripheral veins, especially those of the lower extremities, with consequent inadequate cerebral perfusion. Symptoms can range from lightheadedness to loss of consciousness. Heat syncope most commonly occurs during prolonged standing in hot weather and during the initial period of heat acclimation and is aggravated by dehydration. Patients with heat or parade syncope are generally treated with Trendelenburg positioning and recover rapidly, although complete recovery of stable blood pressure and heart rate (resolution of orthostasis) in some patients may take an hour or two or may require rehydration (5). Patients presenting with heat syncope should be thoroughly evaluated for injury resulting from a fall and should be carefully assessed for a cardiac, neurologic, or other potentially serious cause of their collapse (32).

Heat edema presents as swelling and uncomfortable tightness in the hands and/or feet. The physiologic mechanism is unknown but probably includes venodilation and extravascular fluid shifts with subsequent pooling of increased interstitial fluid (5). Symptoms usually resolve within a few days or weeks as the person becomes acclimated to the heat. Treatment for this self-limited condition consists of reassurance; elevation of the extremities; and, in severe cases, application of compression stockings. Administration of diuretics can exacerbate volume depletion and should be avoided (32).

Miliaria rubra, or heat rash, occurs when occluded sweat gland pores trap

27. Miller TM, Layzer RB. Muscle cramps. Muscle Nerve. 2005;32:431-442. [PMID: 15902691]
28. Miller KC, Stone MS, Huxel KC, et al. Exercise-associated muscle cramps: causes, treatment, and prevention. Sports Health. 2010;2:279-283. [PMID: 23015948]
29. Dijkstra JN, Boon E, Kruijt N, et al. Muscle cramps and contractures: causes and treatment. Pract Neurol. 2023;23:23-34. [PMID: 36522175]
30. Gauer R, Meyers BK. Heat-related illnesses. Am Fam Physician. 2019;99:482-489. [PMID: 30990296]
31. Merchant RK, Grundstein A, Yeargin S, et al. Exertional heat illnesses in marching band artists: a case series. Int J Biometeorol. 2021;65:2181-2188. [PMID: 34424411]
32. Lugo-Amador NM, Rothenhaus T, Moyer P. Heat-related illness. Emerg Med Clin North Am. 2004;22:315-327, viii. [PMID: 15163570]

eccrine sweat in the skin, resulting in erythematous papules or pustules (30). Skin covered by clothing is most often affected, and the condition may be complicated by a secondary staphylococcal infection. Because miliaria skin cannot fully contribute to thermoregulatory sweating, the risk for exertional heat illness increases in proportion to the amount of skin surface involved. Miliaria rubra is treated through cooling and drying of affected skin, avoiding conditions that induce sweating, controlling infection, and relieving pruritus. Treatment can also be managed with chlorhexidine lotion or cream with or without salicylic acid or with low-dose topical corticosteroids. For a diffuse pustular rash complicated by infection, systemic antibiotics may be prescribed. Prevention includes proper skin hygiene; wearing clean, loose-fitting clothes; and avoiding talc and creams (30). Miliaria rubra generally improves with heat acclimation.

What is the approach to management of severe heat-related illness?

SHI includes the clinical spectrum of HE, HI, and HS. HE is the mildest of the severe heat-related illnesses and is by definition readily reversible by removal from activity in the heat with active cooling and rehydration. However, the distinction among HE, HI, and HS at the time of injury can be challenging and is often best made retrospectively, so the illness should initially be managed as HS.

The morbidity and mortality associated with SHI are associated with the duration of exposure to high temperatures and treatment delay (6–8, 18–20). Management of severe SHI accordingly necessitates a rapid, systematic, and integrated approach. In 2016, the Korey Stringer Institute convened a meeting with experts in the fields of emergency and sports medicine and developed a paradigm for the management of EHS, including rapid recognition, rapid assessment, rapid cooling, and rapid advanced care (33). Although the identified steps were designed for prehospital care of EHS, they can be applied to CHS and also to management in the ED and inpatient setting (as not all patients will have access to prehospital care) or detailed interventions in the ED, with distinct diagnostic tools and interventional methods at each level of care (33, 34).

What are the goals of management in the prehospital setting?

The best outcomes occur with early identification of persons with severe heat illness, emergent cooling, and transport to facilities that are prepared for the next steps in clinical management (20). Accordingly, a preplanned response in the prehospital setting is critical, with the well-recognized "golden hour" of opportunity (recently optimized to the "golden half hour") and the accepted paradigm of "cool first, transfer second" (19, 23, 35).

Goal 1: Rapid recognition

Because the differential diagnosis in suspected SHI is extensive (Appendix Table 2), with overlapping clinical presentations, alertness to common signs and symptoms is critical (Table 2). It is important to remember that although a patient with severe SHI may present with CNS dysfunction, in some patients a lucid interval can occur, with CNS function subsequently declining rapidly (23).

Goal 2: Rapid assessment

Accurate temperature assessment with, ideally, a properly measured rectal temperature is key to the diagnosis in patients with a suspected SHI. However, whole-body cooling should not be delayed for a core temperature measurement when SHI is suspected based on the initial clinical assessment (6, 23).

Goal 3: Rapid cooling

Body cooling has been shown to perform 3 critical tasks: It reduces organ and tissue temperature, it redirects blood from the skin (cutaneous vasoconstriction) to the central circulation to support organ tissue and perfusion, and it supports blood pressure regulation

33. Belval LN, Casa DJ, Adams WM, et al. Consensus statement–prehospital care of exertional heat stroke. Prehosp Emerg Care. 2018;22:392-397. [PMID: 29336710]
34. Jacobsen RC, Beaver B, Abo B. Out-of-hospital cold water immersion for classic (non-exertional) heat stroke guided by real-time core temperature monitoring: a case series. Prehosp Emerg Care. 2023;27:832-837. [PMID: 36377966]
35. Heled Y, Rav-Acha M, Shani Y, et al. The "golden hour" for heatstroke treatment. Mil Med. 2004;169:184-186. [PMID: 15080235]

(5). Because morbidity and mortality are directly associated with the duration of hyperthermia after collapse and likely underperfusion, cooling is ideally done on site before transport ("cool first, transport second"). Cold water immersion (CWI) provides the fastest cooling rates and has consistently been identified as best practice. When CWI is unavailable or impractical, initiating cooling on site with any available strategy, such as removing clothing; repeatedly dousing the person with cold water; putting ice in the axilla, in the groin, or all over the body; or applying a fan, is superior to passive cooling (5–8, 20, 23). **Appendix Table 4** (available at Annals.org) identifies prehospital cooling methods and rates that are commonly used, although many factors may contribute to variability, such as body weight and habitus, gender, and duration of hyperthermia (36–38).

The primary goal of cooling is to decrease the body temperature, with prioritization of a core temperature reduction to below 39 °C (102.2 °F) within 30 minutes (ideally) to 60 minutes to protect the critical organs. An evidence-based minimum cooling rate has not been established; however, cooling rates above 0.10 °C (0.18 °F) per minute seem to be acceptable, and cooling rates above 0.15 °C per minute are preferred to reduce morbidity and mortality (16, 19, 24). Patients with an indwelling rectal thermistor can be monitored continuously without interrupting body cooling. When a rectal thermistor is not available, repeated measurement of rectal temperature can interrupt body cooling and reduce the overall cooling rate. The recent American College of Sports Medicine (ACSM) consensus recommendation is to continue uninterrupted cooling until the patient shows improved CNS function or "wakes up" (eyes open, normal behavior, and conversational). If the rectal temperature confirms cooling to the goal level at this point, rectal temperature measurement can be discontinued. If a patient does not improve in 30 to 40 minutes, clinical reassessment is indicated (6).

A recent study compiled data from laboratory and field studies and found that patients with EHS must be immersed for 11 to 12 minutes when water temperature is 9 °C (48.2 °F) or lower and for 18 to 19 minutes when water temperature is 10 to 26 °C (50 to 78.8 °F) (39).

Current guidance from the National Athletic Trainers' Association, the ACSM, and the Wilderness Medical Society is to stop active cooling at approximately 38.3 to 38.9 °C (101 to 102 °F) to prevent hypothermic overshoot (6, 23, 40). However, there have been no studies on the exact target at which to stop cooling, and there are no known disadvantages or adverse outcomes from cooling below 38 °C (100.4 °F). Most "overcooled" athletes are in the range of 35 to 37 °C (95 to 97 °F), which causes no significant adverse physiologic effects (41).

Goal 4: Transfer to advanced medical care

There are currently no evidence-based guidelines to assist providers in the important decision about which patients need transfer to the ED for further evaluation and which can be safely sent home. In general, once cooling has been completed on site, most patients should be triaged and transported to a facility that is equipped to evaluate and manage the complications of SHI (6). However, several studies examining the experience at the Falmouth Road Race have shown that many persons with exertional heat illness who are promptly recognized, emergently cooled, and treated and are clinically stable can be discharged to home with family (42, 43). Critical elements of the prehospital discharge to home decision in this cohort included emergent early cooling, patient education, and a physician involved with clinical decision making. In addition, it should be noted that athletes who sustain EHS typically

36. McDermott BP, Casa DJ, Ganio MS, et al. Acute whole-body cooling for exercise-induced hyperthermia: a systematic review. J Athl Train. 2009;44:84-93. [PMID: 19180223]
37. Friesen BJ, Carter MR, Poirier MP, et al. Water immersion in the treatment of exertional hyperthermia: physical determinants. Med Sci Sports Exerc. 2014;46:1727-1735. [PMID: 24784433]
38. Lemire BB, Gagnon D, Jay O, et al. Differences between sexes in rectal cooling rates after exercise-induced hyperthermia. Med Sci Sports Exerc. 2009;41:1633-1639. [PMID: 19568196]
39. Flouris AD, Notley SR, Stearns RL, et al. Recommended water immersion duration for the field treatment of exertional heat stroke when rectal temperature is unavailable. Eur J Appl Physiol. 2024;124:479-490. [PMID: 37552243]
40. Eifling KP, Gaudio FG, Dumke C, et al. Wilderness Medical Society clinical practice guidelines for the prevention and treatment of heat illness: 2024 update. Wilderness Environ Med. 2024;35:112S-127S. [PMID: 38425235]
41. Sawka M, O'Connor F. Disorders due to heat and cold. In: Goldman L, Schafer AI. Goldman-Cecil Medicine. Elsevier/Saunders; 2019:659-663.
42. Stearns RL, Hosokawa Y, Belval LN, et al. Exertional heat stroke survival at the Falmouth Road Race: 180 new cases with expanded analysis. J Athl Train. 2024;59:304-309. [PMID: 37655801]
43. Demartini JK, Casa DJ, Stearns R, et al. Effectiveness of cold water immersion in the treatment of exertional heat stroke at the Falmouth Road Race. Med Sci Sports Exerc. 2015;47:240-245. [PMID: 24983342]

do not have the comorbidities identified in persons with nonexertional HS.

In a cohort of 274 runners with suspected EHS at the Falmouth Road Race, the survival rate was 100%, with 93% of the runners able to be cleared for home discharge by a medical tent physician (43).

Conversely, if on-site cooling was not started or completed in a timely fashion in a patient with suspected EHS or CHS, the patient would be best managed at the nearest medical facility with the capability for emergent cooling. Cooling should be maintained during transport from the field to the ED (33, 34).

What are the goals of treatment in the ED?

In 2021, Rublee and colleagues proposed an evidence-based treatment pathway to be used in the ED, with 3 critical treatment goals (recognition, rapid cooling, and supportive care), reflecting a similar approach to the goals of prehospital care as not all patients will have had the advantage of prehospital interventions. Specific provider steps and tasks are shown in Table 3 (44).

The cornerstone of Rublee and colleagues' algorithm is recognition of the importance of time, with a goal of initiation of cooling within 10 minutes and cooling to a core temperature below 39.2 °C (102.5 °F) within 30 minutes. After recognition, the team should obtain supplies (ice, ice packs, mist bottle, fan, cooling blanket, Foley catheter [ideally temperature-sensing], intravenous fluids, cooling devices) and decide on external or internal cooling, or both (44). Evaporative cooling techniques are also advocated in the ED, particularly in the management of CHS (20, 45). These techniques, which typically use fans to blow warmed air over the patient as they are sprayed with cooled, atomized water, produce a slightly slower rate of cooling but with fewer impracticalities and a decreased shivering response (45). Evaporative cooling combined with convective

44. Rublee C, Dresser C, Giudice C, et al. Evidence-based heatstroke management in the emergency department. West J Emerg Med. 2021;22:186-195. [PMID: 33856299]
45. Graham BS, Lichtenstein MJ, Hinson JM, et al. Nonexertional heatstroke. physiologic management and cooling in 14 patients. Arch Intern Med. 1986;146:87-90. [PMID: 3942468]

Table 3. **Emergency Management of Heat Stroke in the Emergency Department: An Evidence-Based Proposal***

Treatment Goals	*Timeline Goals*	*Provider Key Steps*	*Team Tasks*
Recognition	Initiate algorithm within 5 min	• Assess airway, breathing, and circulation • Document core temperature • Consider alternative/concomitant diagnoses and need for neuroimaging	• Obtain core temperature • Place patient on the monitor • Establish large-bore IV access • Perform laboratory tests, electrocardiography, and chest radiography
Rapid cooling	Initiate cooling within 10 min	• Begin external cooling and consider internal cooling • Consider deep sedation followed by neuromuscular blockade to reduce metabolic heat production • Consider use of benzodiazepines to prevent shivering	Begin external cooling • Perform cold water immersion • Apply ice packs to axilla, groin, and neck • Mist patient with water and direct fan • Apply cooling blanket (if available) Consider internal cooling • Hang chilled fluids • Place 3-way Foley catheter for bladder irrigation • Sedate and paralyze • Place intravascular cooling device • Perform body cavity lavage (rare) • ECMO/transfer to liver transplant center for patients with more severe illness (rare)
Supportive care	Cool to 39 °C (102.2 °F) by 30 min	• Continuous temperature monitoring • Correct electrolyte abnormalities • Arrange for disposition (likely ICU) • Consider transfer to transplant center if in acute liver failure	• Serial neurologic and hemodynamic reassessments • Keep defibrillator and pads at bedside • Resupply ice as needed • Active temperature management

ECMO = extracorporeal membrane oxygenation; ICU = intensive care unit; IV = intravenous.
** Information from reference 44.*

methods in elderly patients may offer several advantages, including greater patient comfort and easier access for potential advanced monitoring or resuscitation. However, these advantages need to be balanced against the value of CWI. A review of cooling methods in HS identified the absence of high-quality clinical trials for elderly patients and concluded that elderly patients must be judged clinically on an individual basis (46).

The use of chilled intravenous saline in patients with SHI has not been evaluated in large randomized controlled trials. However, a recent laboratory study performed in athletes did show that hyperthermic persons could be effectively cooled with chilled saline (4 °C [39 °F]) (47). Intravenous fluid administration can help support the cardiovascular system, as most patients with SHI are hypovolemic from systemic vasodilation and/or dehydration.

Further supportive care focuses on circulation, airway, and breathing, with identification and correction of metabolic derangements. The medical team must be aware of common SHI sequelae, including AKI, rhabdomyolysis, liver failure, and DIC, and should ideally have preplanned advanced care and organ system protocols in place. A core task at this point is clinical consultation with the hospital critical care team for further disposition and management.

When should patients be hospitalized?

Hospitalization from the ED is a relatively easy decision for patients who have severe presentations or require advanced airway management, but those who are clinically stable and received excellent prehospital care can be more challenging. Accordingly, there is no one-size-fits-all recommendation for deciding on hospitalization or discharge home, particularly because patients who are successfully cooled and survive the hyperthermic–neurologic phase are at high risk for progression to the hematologic–enzymatic and late hepatic–renal phases (19). The pattern of biomarker release associated with HS is well described, with the enzymatic and hepatic renal phases occurring 48 to 96 hours after the initial event (19).

A study evaluating 2529 EHS episodes in soldiers identified that indicators of AKI, including serum electrolyte disturbances and abnormal urinalysis findings, were most prevalent on the day of the injury but normalized within 24 to 48 hours, while muscle damage and liver function–associated markers peaked 24 to 96 hours after injury and persisted outside their respective reference ranges for 2 to 16 days (13).

Although there are no evidence-based guidelines for clinical decision making on who may safely be discharged home, ED release would ideally be considered if the patient was successfully cooled in a prehospital setting, if the patient has access to caregivers in case the clinical condition were to change, and if there is a clear plan for outpatient follow-up. If, on the other hand, active cooling interventions were required in the ED or if the patient has significant comorbidities or no support structure at home, hospitalization for observation should be strongly considered (5–8, 19, 20, 48).

What are the goals of management in the initial inpatient setting?

There are currently no specific therapies for management of HS; beyond emergent cooling, treatment is largely supportive. There is no current evidence for use of pharmacotherapy to facilitate cooling, such as acetaminophen, nonsteroidal anti-inflammatory drugs, or dantrolene (20). The 3 principal goals for inpatient management of serious HS are continued cooling and temperature surveillance, support of the cardiovascular system, and identification and treatment of complications. Multiple excellent resources provide detailed guidance for the management of HS and related complications, and prognostic

46. Gaudio FG, Grissom CK. Cooling methods in heat stroke. J Emerg Med. 2016;50:607-616. [PMID: 26525947]
47. Morrison KE, Desai N, McGuigan C, et al. Effects of intravenous cold saline on hyperthermic athletes representative of large football players and small endurance runners. Clin J Sport Med. 2018;28:493-499. [PMID: 29112514]
48. Savioli G, Zanza C, Longhitano Y, et al. Heat-related illness in emergency and critical care: recommendations for recognition and management with medico-legal considerations. Biomedicines. 2022;10:2542. [PMID: 36289804]

Table 4. **ACSM Return to Activity Considerations Following a Diagnosis of EHS or EHI***

- A detailed history and physical examination including unique intrinsic and extrinsic risk factors, the timing of treatment, and the rate of cooling must be considered in the RTA decision.
- The athlete should refrain from exercise for at least 7 d after release from initial medical care, at which time the clinician will address the clinical course of the heat stroke incident and carefully assess the status of end-organ function (neurocognitive, renal, hepatic, muscle, hematologic as clinically indicated).
- The clinician should carefully address any intrinsic and extrinsic risk factors associated with the EHS event.
- When medically eligible for RTA/RTP based on the return of normal end organ function, an individual can begin exercise in a cool environment and gradually increase the duration, intensity, and heat exposure over 2 to 4 wk to initiate environmental acclimation, improve fitness, and demonstrate heat tolerance.
- If return to vigorous activity and evidence of the patient's ability to adapt to exercise-heat stress over several days is not accomplished within 4 to 6 wk, consider referral to a physician with experience in heat related disorders for further evaluation that may include HTT in a controlled setting.
- An athlete may be allowed to resume full competition between 2 and 4 wk after demonstrating sports-specific exercise acclimation and heat tolerance with no abnormal symptoms or test results during the re-acclimation period.

ACSM = American College of Sports Medicine; EHI = exertional heat injury; EHS = exertional heat stroke; HTT = heat tolerance testing; RTA = return to activity; RTP = return to play.
** Reproduced from Roberts WO, Armstrong LE, Sawka MN, et al, ACSM expert consensus statement on exertional heat illness: recognition, management, and return to activity,* Current Sports Medicine Reports*, 2023, vol. 22, pages 134-149 (http://doi.org/10.1249/JSR.0000000000001058), with permission.*

inpatient scoring systems are also available (19, 20, 49–55).

Goal 1: Continued cooling

Monitoring for rebound hyperthermia is recommended for the initial stages of inpatient care, with maintenance of esophageal, bladder, or rectal thermistors. Although there is no consensus guideline on the duration of monitoring for hypothermia or rebound hyperthermia, surveillance should be considered for the first days of hospitalization (54). Hypothermia is a concern with the potential for associated arrhythmias and a hypercoagulable state, while rebound hyperthermia warrants consideration of occult infection or other potential causes (Appendix Table 2). Endovascular cooling can be maintained during hospitalization and offers an alternative to cooling blankets (56, 57).

Goal 2: Support of the cardiovascular system

Cooling directly assists with cardiovascular support; by vasoconstricting blood vessels in the skin and superficial tissues, intravascular volume is mobilized from the peripheral to the central circulation. Dehydration can be associated with SHI and should be corrected, and hypovolemia also occurs from extensive cutaneous vasodilation. Although most consensus recommendations include early intravenous access with fluid therapy as clinically indicated to support the cardiovascular system, caution is also recommended to prevent fluid overload with complications that include hyponatremia and pulmonary edema (58, 59).

Goal 3: Management of complications

As previously discussed, HS is associated with a systemic inflammatory response leading to a syndrome of multiorgan dysfunction in which encephalopathy predominates. End-organ disorders that may be seen in HS include rhabdomyolysis, acute hepatic injury, AKI, and DIC. Although there are no specific therapeutic agents to treat HS, several strategies have been shown to prevent or mitigate complications. Aggressive intravenous fluid therapy to achieve a urine output greater than 300 mL/h can mitigate rhabdomyolysis-induced AKI, and *N*-acetylcysteine has been used for prevention of liver failure in HI-mediated ischemic hepatitis (60, 61). Although a complete guide to managing these complications is beyond the scope of this review, Appendix Table 5 (available at Annals.org) provides initial recommendations. Current medical resources should be consulted as appropriate (7, 8, 19, 49, 62).

49. Liu SY, Song JC, Mao HD, et al; Expert Group of Heat Stroke Prevention and Treatment of the People's Liberation Army, and People's Liberation Army Professional Committee of Critical Care Medicine. Expert consensus on the diagnosis and treatment of heat stroke in China. Mil Med Res. 2020;7:1. [PMID: 31928528]
50. Barletta JF, Palmieri TL, Toomey SA, et al. Management of heat-related illness and injury in the ICU: a concise definitive review. Crit Care Med. 2024;52:362-375. [PMID: 38240487]
51. Patel J, Boyer N, Mensah K, et al. Critical illness aspects of heatstroke: a hot topic. J Intensive Care Soc. 2023;24:206-214. [PMID: 37260431]
52. Hifumi T, Kondo Y, Shimizu K, et al. Heat stroke. J Intensive Care. 2018;6:30. [PMID: 29850022]
53. Li P, Yang L, Liu R, et al. The value of the exertional heat stroke score for the prognosis of patients with exertional heat stroke. Am J Emerg Med. 2021;50:352-355. [PMID: 34454398]
54. Chen L, Xu S, Yang X, et al. Association between cooling temperature and outcomes of patients with heat stroke. Intern Emerg Med. 2023;18:1831-1842. [PMID: 37133728]
55. Bursey MM, Galer M, Oh RC, et al. Successful management of severe exertional heat stroke with endovascular cooling after failure of standard cooling measures. J Emerg Med. 2019;57:e53-e56. [PMID: 31005365]
56. DeHan PJ, Flores SA, Rhodehouse BB, et al. Rebound hyperthermia in exertional heat stroke. Mil Med. 2025;190:e881-e885. [PMID: 39212949]
57. Broessner G, Beer R, Franz G, et al. Case report: severe heat stroke with multiple organ dysfunction - a novel intravascular treatment approach. Crit Care. 2005;9:R498-R501. [PMID: 16285034]
58. Oh RC, Malave B, Chaltry JD. Collapse in the heat - from overhydration to the emergency room - three cases of exercise-associated hyponatremia associated with exertional heat illness. Mil Med. 2018;183:e225-e228. [PMID: 29365179]
59. Bouchama A, Dehbi M, Chaves-Carballo E. Cooling and hemodynamic management in heatstroke: practical recommendations. Crit Care. 2007;11:R54. [PMID: 17498312]

What are considerations for a return to physical activity?

The final step in the chain of survival is return to physical activity (RTA). RTA is frequently the principal concern of young athletes, recreational exercisers, or occupational laborers, who are eager to return to previous competition, activities, or employment, respectively. Unfortunately, clinicians confront real concerns of ensuring medical recovery, the return of acclimation, and the ability to thermoregulate in a warm environment in order to ensure safety in the absence of substantial evidence-based guidance. As previously noted, exertional heat illness can recur, and clinicians should therefore optimize the RTA process to minimize this risk.

Most persons with HS recover completely within a few weeks, especially if the incident was recognized promptly and the patient was cooled appropriately (63). However, some with more serious cases can experience long-term complications that may include some combination of multisystem organ dysfunction (liver, kidney, muscle), neurologic damage, reduced exercise capacity, and heat intolerance (64, 65). The ACSM recently published guidance detailing criteria for RTA and a return to play for athletes (**Table 4**), which mirror those recommended by the Department of the Army (5, 6). No specific guidance is available for persons with sustained CHS (20).

Management... Morbidity and mortality associated with severe heat-related illness have been associated with the duration of exposure to high temperatures and treatment delay. Management of severe heat-related illness therefore necessitates a rapid, systematic, and integrated approach, including rapid recognition and cooling, cardiovascular support, and management of complications. Because SHI involves multiorgan dysfunction, it requires careful assessment before a return to prior activities.

CLINICAL BOTTOM LINE

Practice Improvement

What do professional organizations recommend regarding prevention, diagnosis, and screening of heat-related illnesses?

The Centers for Disease Control and Prevention maintains extensive resources on its website that pertain to heat-related illness, including specific recommendations for travelers, children, athletes, and occupational laborers, with further resources from the National Institute for Occupational Safety and Health. Unfortunately, there are currently no federal protections and very few state protections for laborers, who represent the highest-risk demographic group for exertional heat illness.

Several sports medicine organizations provide specific prevention and treatment guidance, including the National Athletic Trainers' Association and the ACSM. These organizations detail recommendations for acclimation, prehospital care, and return to play. The Wilderness Medical Society provides several clinical practice guidelines on environmental challenges encountered by outdoors enthusiasts, with specific attention to recommendations in resource-constrained environments. The military also provides extensive resources for prevention and management of exertional heat illness in warfighters.

Finally, several professional organizations are strong advocates for improvement in public health efforts to mitigate heat illness, including the American Public Health Association, the Medical Society Consortium on Climate and Health, and the American Nurses Association.

60. Long B, Koyfman A, Gottlieb M. An evidence-based narrative review of the emergency department evaluation and management of rhabdomyolysis. Am J Emerg Med. 2019;37:518-523. [PMID: 30630682]
61. Will JS, Snyder CJ, Westerfield KL. N-acetylcysteine (NAC) for the prevention of liver failure in heat injury-mediated ischemic hepatitis. Mil Med. 2019;184:565-567. [PMID: 30811527]
62. Farkas J. Hyperthermia & heat stroke: management of complications. Internet Book of Critical Care (IBCC). 20 June 2021. Accessed at https://emcrit.org/ibcc/hyperthermia/#management_of_complications on 19 April 2025.
63. McDermott BP, Casa DJ, Yeargin SW, et al. Recovery and return to activity following exertional heat stroke: considerations for the sports medicine staff. J Sport Rehabil. 2007;16:163-181. [PMID: 17923722]
64. Mehta AC, Baker RN. Persistent neurological deficits in heat stroke. Neurology. 1970;20:336-340. [PMID: 5534966]
65. Royburt M, Epstein Y, Solomon Z, et al. Long-term psychological and physiological effects of heat stroke. Physiol Behav. 1993;54:265-267. [PMID: 8372119]

In the Clinic
Tool Kit

Heat-Related Illnesses

Patient Information

https://medlineplus.gov/heatillness.html
Information on heat-related illness from the National Institutes of Health's MedlinePlus.

www.nia.nih.gov/health/safety/hot-weather-safety-older-adults
www.nia.nih.gov/espanol/hipertermia/hipertermia
Hot weather safety tips for older adults in English and Spanish from the National Institute on Aging.

https://familydoctor.org/condition/heat-exhaustion-heatstroke
https://es.familydoctor.org/condicion/agotamiento-por-calor-y-golpe-de-calor-es
Information on heat exhaustion and heat stroke in English and Spanish from the American Academy of Family Physicians.

Information for Health Professionals

www.hprc-online.org/resources-partners/whec/clinical-care/clinical-practice-guideline-prevention-diagnosis-and
Clinical practice guideline on the prevention, diagnosis, and management of exertional heat illness from United States Military Joint Service Medical Providers.

www.cdc.gov/niosh/docs/2016-106
Criteria for a recommended standard on occupational exposure to heat and hot environments from the National Institute for Occupational Safety and Health.

https://journals.sagepub.com/doi/10.1177/10806032241227924
Clinical practice guidelines for the prevention and treatment of heat illness from the Wilderness Medical Society.

https://journals.lww.com/acsm-csmr/fulltext/2021/09000/acsm_expert_consensus_statement_on_exertional_heat.10.aspx
Consensus statement on exertional heat illness from the American College of Sports Medicine.

WHAT YOU SHOULD KNOW ABOUT HEAT-RELATED ILLNESSES

What are the heat-related illnesses?

Heat-related illness occurs when your body overheats, resulting in disruption of normal bodily functions. Minor heat-related illnesses include muscle cramps and heat rash. Serious heat-related illnesses include heat exhaustion, heat injury, and heat stroke, which may be life-threatening. Your body normally maintains a steady temperature, but extreme heat and humidity can overwhelm its ability to cool down. Heat stroke, the most severe form of heat-related illness, is characterized by high body temperature (usually >104 °F [>40 °C]) and neurologic symptoms, such as confusion, seizures, or coma. Heat stroke is a medical emergency that requires immediate treatment.

Who is at risk for serious heat-related illness?

Young children and elderly people are more vulnerable to serious heat-related illness. Certain health conditions, medications, and environmental factors may also increase risk, including:

- Obesity
- Heart problems
- Medications such as beta-blockers, diuretics, and antihistamines
- Inability to care for oneself
- Prior heat illness
- A period of unusually hot weather
- Limited access to air conditioning or other cooling measures
- Coach or supervisor pressure

What are the signs and symptoms of serious heat-related illnesses?

Heat stroke involves overheating of the body along with neurologic symptoms and is a medical emergency requiring immediate treatment. Other serious heat-related illnesses, such as heat exhaustion, may present similarly at first. Signs and symptoms may include:

- Dizziness or weakness
- Low blood pressure
- Fast heartbeat
- Headache
- Nausea and vomiting
- Confusion, seizures, and loss of consciousness

Most people recover completely if treated early, but others, particularly when recognition or treatment with emergent cooling is delayed, may progress to kidney, liver, or other organ damage.

How can serious heat-related illnesses be prevented?

- Stay hydrated. Drink plenty of fluids, especially water, throughout the day.
- Avoid strenuous activity during the hottest part of the day. Exercise in the early morning or late evening.
- Wear loose-fitting, light-colored clothing, which helps your body stay cool.
- Take breaks in a cool place. Seek shade or air conditioning.
- Gradually acclimate to the heat. Slowly increase your exposure to hot weather.
- Monitor yourself and others for symptoms. Check on elderly or vulnerable people during heat waves.

How are heat-related illnesses treated?

Immediate cooling is crucial. This may involve cold water immersion, ice packs, or cooling blankets. Hospitalization is often necessary for supportive care, including fluids, electrolytes, and monitoring for complications.

Questions for My Doctor

- Am I at increased risk for heat-related illness?
- What precautions should I take to prevent heat-related illness?
- What are the signs and symptoms of heat-related illness that I should watch out for?
- What should I do if I think I have heat-related illness?
- What are the potential long-term complications of heat-related illness?

For More Information

ACP℠
American College of Physicians
Leading Internal Medicine, Improving Lives

MedlinePlus
https://medlineplus.gov/heatillness.html

National Institute on Aging
www.nia.nih.gov/health/safety/hot-weather-safety-older-adults
www.nia.nih.gov/espanol/hipertermia/hipertermia

American Academy of Family Physicians
https://familydoctor.org/condition/heat-exhaustion-heatstroke
https://es.familydoctor.org/condicion/agotamiento-por-calor-y-golpe-de-calor-es

Patient Information

Appendix Table 1. **Strategies for the Prevention of Exertional Heat Illness***

EHS Prevention	*CHS Prevention*
Education • Heat stress education to participants, supervisors, coaches, and leaders should include risk factors, mitigation strategies, and recognition of heat strain • Emergency action planning should include provision of on-site cooling	**Education** • Inform the public about heat illnesses and first aid measures • Conduct professional CHS management sessions for paramedics
Risk assessment and enhancement • Heat acclimation status • Physical fitness, obesity • Adequate fluid and salt consumption	**Population risk assessment and enhancement** • Identify isolated vulnerable populations, establish contact, and ensure adequate ventilation of their spaces • Prepare air-conditioned public transport and increase shaded waiting areas • Ensure hospital preparedness to receive high number of patients with heat-related illnesses, including heat stroke
Event mitigation strategies • Modification of event workload based on local WBGT • WBGT assessment • Provision of intraevent hydration and cooling methods	**Mitigation strategies** • Avoid scheduled or habitual sport activities • Prohibit nonessential laborious work • Encourage spending a few hours in a cold environment • Take additional showers or baths • Avoid direct sun exposure • Keep hydrated

CHS = classic heat stroke; EHS = exertional heat stroke; WBGT = wet-bulb globe temperature.
** Information from references 5, 18, 19, and 20.*

Appendix Table 2. **Differential Diagnosis of Heat-Related Illness**

Metabolic disorders
- Hypoglycemia
- Hyponatremia
- Thyroid storm

Hematologic
- Exertional collapse associated with sickle cell trait (ECAST)
- Tumor lysis syndrome

Infectious disease
- Encephalitis
- Meningitis
- Malaria
- Sepsis

Central nervous system
- Intracranial hemorrhage
- Status epilepticus

Medication
- Serotonin syndrome
- Anticholinergic toxicity
- Sympathomimetic toxicity
- Salicylate toxicity
- Alcohol or benzodiazepine withdrawal

Systemic
- Pheochromocytoma
- Malignant hyperthermia
- Neuroleptic malignant syndrome
- Pulmonary embolus

Appendix Table 3. **Laboratory and Adjunctive Testing to Be Considered in the Management of Heat-Related Illness**

Clinical Indication	*Suggested Laboratory and Adjunctive Testing*
Cardiac status	Cardiac troponin I Creatine kinase–MB B-type natriuretic peptide Electrocardiography Echocardiography
Pulmonary status	Oximetry Arterial blood gas Chest radiography
Fluid and electrolyte status	Potassium Sodium Chloride Calcium Glucose
Acid–base balance	Arterial blood gas Lactate
Blood/clotting function	Complete blood count Sickle cell trait status International normalized ratio Prothrombin time Activated partial thromboplastin time D-dimer Fibrinogen
Acute kidney injury	Urinalysis Blood urea nitrogen Creatinine Urea
Neurocognitive status	Cranial computed tomography or magnetic resonance imaging Electroencephalography
Infection	Aerobic and anaerobic blood cultures C-reactive protein
Liver function	Aspartate aminotransferase Alanine aminotransferase Lactate dehydrogenase Total bilirubin
Muscle function	Creatine kinase Myoglobin

Appendix Table 4. **Prehospital Cooling Strategies***

Body Cooling Strategy	*Reported Range of Cooling Rates, °C/min*
Cold water immersion	0.13–0.35
Cold water dousing	0.04–0.20
Tarp-assisted water/ice immersion	0.14–0.17
Ice water–soaked towels	0.11–0.16
Ice cold/water-soaked sheets	0.05–0.16
Water spray mister	0.03–0.17
Passive cooling	0.03

** Information from reference 6; from Douma MJ, Aves T, Allan KS, et al, First aid cooling techniques for heat stroke and exertional hyperthermia: a systematic review and meta-analysis,* Resuscitation*, 2020, vol. 148, pages 173-190; and from DeGroot DW, Henderson KN, O'Connor FG, Cooling modality effectiveness and mortality associate with prehospital care of exertional heat stroke casualties,* The Journal of Emergency Medicine*, 2023, vol. 64, pages 175-180.*

Appendix Table 5. **Select Inpatient System Management Considerations in Patients With Serious Heat–Related Illness***

Illness	*Considerations*
Rhabdomyolysis	Administer IV fluid infusion, 1–2 L/h (aggressive fluid treatment in the first hour), then 300 mL/h IV furosemide (10–20 mg in patients without previous diuretic treatment; follow-up dose depends on urine output) in case of fluid overload Sodium bicarbonate, 30 mmol/h (to achieve urine pH >6.5) Myoglobinuria is expected Hypercalcemia and metabolic alkalosis (pH >7.5) should be avoided
Liver failure	Monitor liver function and mental status for ≥4 d Provide supportive treatment: hemodynamic stability, *N*-acetylcysteine IV (bolus dose, 150 mg/kg in 200 mL of 5% glucose solution for 20 min, then 50 mg/kg in 500 mL of 5% glucose solution for 4 h, then 100 mg/kg in 1000 mL of 5% glucose solution for 16 h) Administer hypertonic saline 3% IV or mannitol IV (0.25–2 g/kg in 30 min in 20% solution), hemofiltration, laxatives (e.g., oral lactulose, 30 mL every 2 h until diarrhea occurs), oral rifaximin (400 mg 3 times a day) in case of fulminant liver failure Liver transplant rarely needed, and there is no evidence that it is effective
Acute kidney injury	Administer crystalloid solution to maintain urine output >50 mL/kg/h Administer IV furosemide (10–20 mg in patients without previous exposure to diuretics; follow-up dose depends on urine output) Provide hemodialysis or CVVH in cases of volume overload, severe acidosis, hyperkalemia, or uremia Adjust fluid infusion rate according to blood pressure and urine output Monitor electrolytes and correct as needed
Disseminated intravascular coagulation	For bleeding and thrombosis, administer fresh-frozen plasma (bolus dose, 10–15 mL/kg, then 200–400 mL according to coagulation indices) Administer cryoprecipitate (5–10 U each time) for fibrinogen level <180 mg/dL Administer platelet concentrates (infusion of 1 therapeutic dose) if platelet count $<0.020 \times 10^9$/L or if there is bleeding and platelet count $<0.050 \times 10^9$/L In patients with hepatic failure, consider PCCs to achieve a target INR ≤1.5 Inject PCC dose according to INR and patient's weight Avoid heparin Beware of hypothermia and metabolic acidosis

CVVH = continuous venovenous hemofiltration; INR = international normalized ratio; IV = intravenous; PCC = prothrombin complex concentrate.
** Information from reference 19.*