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Exertional Heat Stroke — Recognition and Cooling Protocols: A Narrative Review

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Abstract

Background. Exertional heat stroke (EHS) is the most severe form of exertional heat illness and one of the leading causes of non-traumatic sudden death in athletes, soldiers and outdoor workers. It is defined by central nervous system dysfunction combined with severe hyperthermia, and its outcome depends almost entirely on how quickly the diagnosis is made and body cooling is begun. With rising global temperatures and growing participation in endurance and team sports, timely recognition and correct cooling have become increasingly relevant to clinical practice.

Aim. This narrative review synthesises the current evidence on the recognition of EHS and on cooling protocols, with a focus on medical aspects, and critically compares diagnostic tools and cooling modalities across sporting, military, occupational and community settings.

Material and methods. A thematic narrative synthesis was performed of original studies, systematic reviews, case reports and consensus-based reviews addressing the pathophysiology, recognition, cooling and prevention of EHS.

Results. EHS diagnosis rests on the combination of neurological dysfunction and elevated core temperature; rectal thermometry is the reference standard, whereas peripheral methods are unreliable and frequently unavailable. Rapid whole-body cold-water immersion is the gold-standard treatment; cooling rates above 0.15 °C·min^{-1} and the principle of "cool first, transport

second" are consistently associated with survival without complications, yet these practices remain incompletely implemented. Alternative field methods differ substantially in efficacy.

Conclusions. Recognition and aggressive cooling are the decisive, modifiable determinants of survival in EHS. Persistent gaps in temperature assessment, prehospital protocols and provider knowledge must be closed to reduce preventable morbidity and mortality.

Key words: exertional heat stroke, hyperthermia, recognition, cold-water immersion, cooling rate, core temperature, prehospital care

1. Introduction

Exertional heat stroke (EHS) is the most severe condition on the continuum of exertional heat illness, which extends from muscle cramps and heat syncope through heat exhaustion to the life-threatening state of heat stroke [1]. It is a medical emergency characterised by central nervous system (CNS) dysfunction — confusion, delirium, ataxia, seizures or coma — accompanied by a core body temperature that is frequently, but not always, in excess of 40 °C [1,2]. Unlike classic heat stroke, which affects vulnerable individuals passively exposed to environmental heat, EHS typically strikes young, healthy and physically fit people during vigorous exercise, most often, though not exclusively, in hot and humid conditions [1,2]. The paradox of EHS is that it targets precisely those individuals who appear least at risk, and that it does so in circumstances — competition, military training, occupational exertion — where the drive to continue can override the body's protective signals [1].

The clinical and public-health significance of EHS is difficult to overstate. In competitive sport it is the second most common cause of non-traumatic death, after cardiac conditions, and in a retrospective analysis of long-distance running events it was estimated that for every serious cardiac adverse event there occurred ten serious cases of EHS [1]. The condition therefore represents a far more frequent life-threatening event in athletic settings than is generally appreciated. Its incidence is not static: with the accelerating pace of climate change and more frequent, intense and prolonged heatwaves, the global burden of heat-related illness — including EHS — is expected to increase, extending the risk to broader populations and geographies [3–5]. Contemporary reviews frame heat stroke not merely as a thermoregulatory failure but as a complex, multi-system inflammatory syndrome, a conceptual shift that has direct implications for how the condition is recognised and managed [5,6].

What makes EHS both tragic and, in principle, largely preventable is the tight relationship between the speed of treatment and the outcome. Timely whole-body cooling initiated within the first hour of symptom onset — and ideally within thirty minutes of collapse — confers a survival rate approaching 100 % [3,4]. Conversely, when recognition or cooling is delayed, mortality rises steeply and survivors are exposed to residual organ damage, including chronic kidney disease, cardiovascular sequelae, heat intolerance and long-term neurological impairment [3,6–8]. The margin between full recovery and death or lifelong disability is thus measured in minutes, and it is determined chiefly by two modifiable factors: whether the condition is recognised promptly and whether cooling is applied aggressively and correctly. These two pillars — recognition and cooling — constitute the practical core of EHS management and the focus of the present review.

Both pillars, however, remain fragile in real-world practice. Accurate diagnosis depends on the measurement of core temperature by rectal thermometry, yet this is rarely available at the point of collapse, and the peripheral methods that are used instead — oral, tympanic, temporal or axillary — systematically underestimate the internal temperature of an exercising, hyperthermic individual and may lead clinicians to falsely exclude EHS [9,10]. The picture is further complicated because active on-site cooling may lower the measured temperature before it can be recorded, so that a value below 40 °C does not exclude the diagnosis [1,2]. On the treatment side, surveys of emergency medical services (EMS) have revealed poor knowledge of EHS best practices, limited access to appropriate cooling equipment, and prehospital protocols that frequently fail to recommend cold-water immersion or even instruct providers to prioritise rapid transport over on-site cooling [9–12]. These deficiencies stand in stark contrast to the strength of the underlying evidence and represent a persistent gap between what is known and what is done.

This narrative review therefore aims to synthesise the current medical evidence on the recognition and cooling of EHS. It first situates the condition within its definitions, classification and pathophysiology, because the mechanisms of injury explain both why the diagnosis is elusive and why rapid cooling is decisive. It then examines the recognition of EHS in detail — the clinical picture, the measurement of core temperature, the differential diagnosis, the role of biomarkers, and the emerging contribution of wearable and predictive technologies. The central part of the review is devoted to cooling protocols: the physical principles of heat transfer, the gold standard of cold-water immersion, the comparative efficacy of alternative field and hospital modalities, and the practical adaptation of cooling to different settings, from elite sport and the military to community events in low- and middle-income countries. Finally, the review considers prevention and heat mitigation, return-to-activity decision-making and the long-term sequelae that follow the acute event. Throughout, the intention is not merely to describe the evidence but to compare and critically appraise it, and to identify where practice diverges from the best available science.

2. Material and Methods

This work was conceived as a narrative review, and the methods reflect that design. A comprehensive, multidisciplinary body of literature was consulted, comprising original research articles, case reports and case series, systematic reviews and meta-analyses, and consensus-oriented narrative reviews addressing the epidemiology, pathophysiology, recognition, cooling, prevention and long-term outcomes of exertional heat stroke and, where relevant, of heat-related illness more broadly. Sources spanned the sporting, military, occupational and clinical emergency-medicine literature, reflecting the fact that the evidence base for EHS is distributed across these overlapping fields and that important lessons in one setting frequently illuminate practice in another.

Consistent with a narrative rather than a systematic design, no formal quality-assessment instrument or meta-analytical pooling was applied to individual studies; instead, the evidence was organised thematically and synthesised critically, with attention to the strength of the supporting data and to areas of disagreement. Studies were grouped under the principal themes of definition and classification; thermoregulatory physiology and pathophysiology; multi-organ

involvement; risk factors; recognition and diagnosis, including core-temperature measurement, differential diagnosis and biomarkers; cooling protocols and modalities; prevention and heat mitigation; and return to activity, recurrence and long-term sequelae. Where the literature permitted, quantitative findings — incidence rates, cooling rates, survival proportions and biomarker thresholds — were extracted and compared directly, so that the relative performance of diagnostic and therapeutic strategies could be appraised. The synthesis deliberately privileges the medical dimensions of recognition and cooling, in keeping with the aim of the review, while acknowledging the behavioural, environmental and organisational context in which these medical decisions are made.

3. Definitions and Classification

A recurring difficulty in the field, and one that has direct consequences for recognition, is the absence of a single universally accepted definition of heat stroke. The most widely used clinical definition, attributed to Bouchama, describes heat stroke as a core body temperature rising above 40 °C accompanied by hot, dry skin and CNS abnormalities such as delirium, convulsions or coma, resulting from exposure to a high environmental temperature or from strenuous exercise [2]. Bouchama additionally proposed a pathophysiological definition, framing heat stroke as a form of hyperthermia associated with a systemic inflammatory response that leads to a syndrome of multi-organ dysfunction, predominantly encephalopathy [2]. This second formulation is important because it anticipates the modern understanding of the condition as more than a simple thermal insult.

The classic dichotomy divides heat stroke into two forms according to the presence or absence of exertion [2]. Classic (non-exertional) heat stroke develops during passive exposure to high environmental temperatures, characteristically affecting the elderly, infants and individuals with comorbidities such as obesity, diabetes, cardiovascular or renal disease, dementia and alcoholism, whose thermoregulatory reserves are insufficient to compensate for the heat load [2]. Exertional heat stroke, by contrast, arises from excessive endogenous heat production during vigorous physical activity that overwhelms the body's capacity to dissipate heat, and it affects predominantly young, otherwise healthy athletes, soldiers and outdoor workers [1,2]. The two forms share a common core — hyperthermia with CNS dysfunction and the risk of multi-organ injury — but differ in onset, population and prognosis: classic heat stroke tends to have a gradual onset and higher mortality, largely because of delayed recognition in vulnerable patients, whereas EHS has a sudden, rapid onset and a more favourable outcome when managed promptly [5]. Sweating is another point of divergence, being often absent in classic heat stroke but usually present, at least initially, in EHS [5]. These distinctions matter for recognition because they shape the index of suspicion: collapse with altered mental status during or immediately after strenuous exercise in a young, previously healthy person should be treated as EHS until proven otherwise.

The insistence on a strict temperature threshold, however, is a recognised weakness of the classic definitions. A body temperature above 40 °C is a cardinal feature of EHS, but it is not diagnostic in isolation, and — critically — a lower measured temperature does not exclude the condition [1,6]. Two circumstances explain this. First, core temperature can fall rapidly after collapse, so that by the time it is measured the value may already have declined; second, on-

site cooling may have been initiated before measurement, artificially lowering the recorded temperature [1,2]. In one instructive community case, a patient with clear encephalopathy, seizure and multi-organ injury after a 5-km run was recorded at only 38.5 °C on tympanic measurement after partial cooling had already been applied, a value that plainly underestimated the true core temperature [13]. The lesson, repeated across the literature, is that the diagnosis of EHS must rest on the full clinical picture — exertional collapse, CNS dysfunction and evidence of systemic injury — rather than on a single temperature threshold [1,13].

In recognition of these limitations, alternative diagnostic frameworks have been proposed that de-emphasise the absolute temperature. In Japan, the Japanese Association for Acute Medicine (JAAM) established criteria that deliberately omit a fixed body-temperature threshold, precisely because fatal cases were observed in patients whose temperatures were below 40 °C [2]. The JAAM criteria define heat stroke by exposure to high environmental temperature together with at least one of: CNS manifestations (impaired consciousness, cerebellar symptoms, convulsions or seizures); hepatic or renal dysfunction requiring inpatient care; or a coagulation disorder diagnosed as disseminated intravascular coagulation (DIC) [2]. A subsequent working-group refinement simplified these into a Glasgow Coma Scale score of 14 or less, creatinine or total bilirubin of 1.2 mg/dL or more, or a JAAM DIC score of 4 or more [2]. The contrast between Bouchama's temperature-anchored definition and the JAAM's organ-dysfunction-anchored criteria encapsulates a genuine tension in the field: the former is conceptually clear but risks missing hyperacute or already-cooled cases, whereas the latter is more sensitive to the systemic reality of the disease but less immediately operational at the point of collapse. For the practical purposes of recognition in the field, the pragmatic synthesis that emerges from the literature is to combine a high index of suspicion based on context (exertion in the heat) with the two clinical anchors of neurological dysfunction and, wherever obtainable, an accurately measured core temperature [1,2,9].

4. Thermoregulation and the Physiology of Heat Balance

Understanding why EHS develops, why it is difficult to recognise and why cooling is decisive requires a grasp of the physiology of thermoregulation, because EHS is fundamentally a failure of this system under load. In humans, core temperature is regulated within a narrow range around 37 °C by a control network centred on the anterior hypothalamus, which integrates information from cutaneous thermoreceptors sensing the ambient environment and from central thermoreceptors sensing internal temperature, and then coordinates autonomic, hormonal and behavioural effector responses to conserve or dissipate heat [9,14]. During exercise, heat is generated as a by-product of metabolism, and the great majority of the energy liberated by muscle contraction — of the order of three-quarters or more — does not perform mechanical work but appears as heat, driving core temperature upward unless it is dissipated [15]. Thermal balance is therefore maintained through the equation between metabolic heat production and heat loss, the latter occurring by the four physical avenues of conduction, convection, radiation and evaporation [9,15].

The relative contribution of these avenues shifts with the thermal environment. At rest in temperate conditions, more than half of heat production is dissipated by radiation and a further fraction by evaporation through sweating and respiration; but as ambient or body temperature

rises above skin temperature, the dry avenues of radiation, conduction and convection become progressively ineffective, and evaporation of sweat becomes the principal — and eventually the only — effective mechanism of heat loss [9,15]. This dependence on evaporation is the Achilles' heel of human thermoregulation, because it is critically limited by ambient humidity: in a hot, humid environment the water-vapour gradient between skin and air narrows, sweat evaporates poorly, and the capacity to lose heat collapses even as heat production continues [1,9]. The consequence is uncompensable heat stress, in which the evaporative heat-loss requirement exceeds the evaporative capacity of the environment, and core temperature rises without check [1]. This physiological reality explains why EHS clusters in hot and humid conditions and why the wet-bulb globe temperature (WBGT), which integrates temperature, humidity, radiant heat and air movement, has become the standard index of environmental heat-stress risk [1].

The immediate thermoregulatory response to a rising core temperature is a redistribution of the circulation: active cutaneous vasodilation increases skin blood flow to carry heat to the surface, while sweating provides the fluid for evaporative cooling [14]. This redistribution, however, carries a cost. Diverting blood to the skin reduces the effective circulating volume and can precipitate heat syncope, and the loss of salt and water in sweat produces dehydration and salt depletion that, if uncorrected, further impair thermoregulation [2]. As thermal and cardiovascular strain compound one another, visceral perfusion falls because blood is shunted from the central circulation to the skin and muscles, setting the stage for organ injury [2]. Severe dehydration is itself a recognised amplifier of risk, because it reduces the sensitivity of the sweating and skin-blood-flow responses to a given core temperature, hastening the development of thermal strain [1]. The physiology thus describes a self-reinforcing spiral in which heat load, cardiovascular strain and fluid loss accelerate one another toward decompensation.

At the cellular level, the same rise in temperature that overwhelms the macroscopic control system also triggers protective responses. Heat shock proteins, produced by nearly all cells in response to thermal and other stresses, are necessary for acquired heat tolerance; overexpression of HSP70, for example, has been shown to protect against organ dysfunction and to reduce mortality in animal models of heat stroke [2]. Adaptive processes at the whole-body level underlie the phenomenon of heat acclimatisation and acclimation, whereby repeated heat exposure produces a lower resting core temperature, earlier and greater sweating, expansion of plasma volume, improved cardiovascular stability and acquired cellular thermotolerance [1,15]. Detailed partitional calorimetry in trained endurance athletes has shown that, following acclimation, the body shifts a greater proportion of heat dissipation onto the evaporative avenue: evaporative heat loss increases while dry heat loss falls, and the net effect is a lower internal temperature for a given metabolic load [15]. These adaptations are central to prevention and are considered in a later section; here they serve to emphasise that thermotolerance is not fixed but modifiable, and that the same individual may be far more or far less vulnerable to EHS depending on their acclimatisation state.

Two broader points about thermoregulatory physiology deserve mention because they recur in the recognition and management of EHS. First, the hypothalamic control network is highly integrated with the endocrine, immune and metabolic systems, and molecular thermosensitivity

is mediated by transient receptor potential channels that translate temperature into neural activity; this integration means that thermoregulatory disturbance is rarely an isolated event but tends to propagate into inflammatory, metabolic and neuroendocrine derangement [5,14]. Second, the body's physiological response to heat can be modulated deliberately, both adaptively through acclimation and acutely through cooling. Controlled exposure of the body to low temperatures — whether by cold-water immersion, cold showers, whole-body cryotherapy or the ingestion of cold fluids and ice — activates a cascade of vasomotor, cardiovascular, endocrine and metabolic responses that can be harnessed either to enhance recovery and performance or, in the emergency setting, to reverse dangerous hyperthermia [16–18]. The recognition that the same physical principle of heat transfer that governs the loss of body heat can be exploited therapeutically is the conceptual bridge between the physiology of thermoregulation and the practice of cooling.

5. Pathophysiology of Exertional Heat Stroke

The modern account of EHS pathophysiology has moved decisively away from the view of the condition as pure thermal injury and toward a model of a systemic, sepsis-like inflammatory syndrome in which hyperthermia is the trigger rather than the sole cause of injury [5,6]. This conceptual evolution matters for recognition and cooling because it explains both why organ injury can progress even after temperature has been normalised and why the duration of hyperthermia — the "area under the temperature curve" — rather than the peak temperature alone determines the outcome [4]. Several overlapping mechanisms have been characterised: direct thermal cytotoxicity, systemic inflammation and immune dysregulation, disruption of the gastrointestinal barrier with microbial translocation, endothelial injury and coagulopathy, and neuroinflammation with selective vulnerability of specific brain regions [2,5,6].

Direct thermal cytotoxicity is the most intuitive mechanism. Elevated intracellular temperature damages cell membranes, enzymatic systems, mitochondria and protein-synthetic machinery, and this damage is aggravated by the relative hypoxia that develops as metabolic demand outstrips supply [9]. Above a threshold of heat cytotoxicity — placed at roughly 42–44 °C in experimental models — thermal injury becomes overwhelming and largely independent of other mechanisms, whereas at the somewhat lower core temperatures characteristic of many EHS cases the systemic mechanisms assume greater importance [3]. This gradient underpins the clinical observation that survival is far higher when core temperature is brought below the cytotoxic range promptly, and it provides the pathophysiological rationale for the emphasis on cooling speed.

The systemic inflammatory response is now regarded as central to severity. Hyperthermia facilitates a coordinated stress response involving endothelial, leukocyte and epithelial cells, with heat shock proteins attempting to preserve protein homeostasis while activated cells release a surge of pro-inflammatory cytokines — interleukin (IL)-1β, IL-6, tumour necrosis factor-α and others — counter-regulated by anti-inflammatory mediators [5,6]. A key amplifier of this cascade is high-mobility group box 1 (HMGB1), a nuclear protein that, once released extracellularly, acts as a damage-associated molecular pattern, binding to pattern-recognition receptors such as Toll-like receptor 4 and the receptor for advanced glycation end-products, activating NF-κB signalling and driving further cytokine release [5,6]. The resulting cytokine

storm recruits and activates leukocytes and promotes cell death through apoptosis, necroptosis and other regulated pathways, sustaining inflammation and driving progression toward multi-organ dysfunction [5,6]. The parallel with sepsis is more than superficial: comparative appraisal of the mechanisms suggests that systemic inflammation and coagulopathy carry the strongest human clinical evidence and the greatest immediate clinical applicability, with elevations in IL-6, TNF-α and HMGB1 and abnormalities of D-dimer, prothrombin time and platelet count offering value in monitoring, risk stratification and prognosis [5].

The gastrointestinal, or "endotoxaemia," paradigm supplies a mechanistic link between exertional-heat strain and systemic inflammation. Sustained heat and exercise, through localised hyperthermia, splanchnic hypoperfusion and neuroendocrine–immune change, damage the intestinal barrier and increase its permeability, permitting the translocation of microbial products such as lipopolysaccharide from the gut lumen into the systemic circulation [3]. When the liver's reticuloendothelial detoxification capacity is exceeded — a capacity itself impaired at high core temperatures — these products bind pattern-recognition receptors and initiate the same systemic inflammatory cascade, potentially culminating in DIC, multi-organ failure and death [3]. This paradigm is considered most relevant when core temperature remains below the threshold of frank heat cytotoxicity, and it reframes the gastrointestinal tract from a passive victim to an active participant in disease progression [3]. It also carries a preventive corollary, in that measures to preserve gut-barrier integrity have been proposed as adjunctive countermeasures [3].

Endothelial injury has emerged as a unifying lesion tying inflammation to coagulopathy and organ failure. The vascular endothelium maintains barrier function, regulates vascular tone and controls coagulation, and heat stress injures it both directly, through thermal cytotoxicity, and indirectly, through the systemic inflammatory response [6]. Characteristic mechanisms include degradation of the endothelial glycocalyx, dysregulation of vascular tone through altered nitric oxide production, disruption of intercellular tight-junction proteins, and activation of regulated cell-death pathways [6]. The shedding of glycocalyx components — syndecan-1, heparan sulfate and hyaluronan — increases vascular permeability, promotes tissue oedema and exposes adhesion molecules that amplify leukocyte–endothelial interaction, while the release of pro-coagulant factors such as von Willebrand factor drives microthrombosis [6]. The clinical translation of this endothelial pathology is coagulopathy: severe heat stroke is complicated by DIC in a substantial proportion of cases — clinical series have reported an incidence around 48 % — and patients who develop DIC have markedly higher rates of multi-organ dysfunction and death [6]. Circulating biomarkers of endothelial injury, including syndecan-1, endothelin-1 and von Willebrand factor, correlate with disease severity and outcome and are being investigated for prognostic use [6].

Finally, the CNS is both the seat of the defining clinical feature of EHS and a principal target of injury. Acute encephalopathy — agitation, delirium, seizures, cerebellar ataxia or altered consciousness — arises from a combination of direct hyperthermic neuronal injury, blood–brain barrier breakdown, cerebral oedema and endotoxin-mediated neuroinflammation [5]. Under severe hyperthermia the tight junctions of the blood–brain barrier are compromised, in part through activation of matrix metalloproteinases such as MMP-9, permitting the extravasation

of plasma proteins and inflammatory mediators and exacerbating cytotoxic oedema [8]. Key mediators of the neuroinflammatory cascade include HMGB1, the NLRP3 inflammasome, IL-1β and IL-18, and the resulting injury shows a selective regional vulnerability, affecting the cerebellum, hippocampus, thalamus and basal ganglia [5,8]. The cerebellum is especially sensitive, with early and selective degeneration of Purkinje cells that is strongly associated with the long-term neurological deficits discussed later in this review [8]. This regional selectivity, and the demonstration that even brief delays in cooling are associated with irreversible neuronal damage, provide the pathophysiological foundation for treating EHS as a time-critical neurological emergency in which the speed of cooling determines not only survival but the preservation of brain function [8].

A further layer of the pathophysiology, and one increasingly targeted by experimental therapeutics, is the activation of regulated cell-death pathways in the injured endothelium and parenchyma. Beyond classical apoptosis, heat stress triggers pyroptosis — an inflammatory form of programmed cell death mediated by gasdermin D and caspase-1 that releases IL-1β, IL-18 and HMGB1 — as well as necroptosis, driven by the RIPK1–RIPK3–MLKL axis, and ferroptosis, characterised by iron accumulation and lipid peroxidation [6]. These pathways are not merely of mechanistic interest: in exertional heat stroke, endothelial pyroptosis with HMGB1 release has been shown to propagate immune dysregulation, and ferroptosis has been implicated in the rhabdomyolysis and acute kidney injury that complicate the syndrome, so that neutralising HMGB1 or inhibiting specific death pathways attenuates tissue damage in experimental models [6]. This mechanistic granularity matters for the present review chiefly because it reinforces the temporal logic of treatment: each of these pathways is engaged and amplified by continued hyperthermia, so that the longer the core temperature remains elevated, the more extensively these self-reinforcing cascades of inflammation, coagulation and cell death are activated, and the greater the burden of organ injury [5,6]. It also situates the several biomarkers of endothelial and glycocalyx injury — syndecan-1, heparan sulfate, hyaluronan, von Willebrand factor and soluble thrombomodulin — within a coherent mechanistic framework, since their circulating concentrations reflect the intensity of these processes and correlate with organ-failure scores and mortality [6]. Although the experimental interventions suggested by this work — inhibition of cell-death pathways, protection of the endothelial glycocalyx, thrombomodulin administration and early fluid resuscitation — remain investigational, they converge with the clinical evidence in identifying the reduction of the hyperthermic insult as the intervention most likely to interrupt the cascade at its origin [5,6]. A comparative appraisal of the several mechanisms concludes that systemic inflammation and coagulopathy carry the strongest and most clinically applicable human evidence, that neuroinflammation and CNS injury constitute an emerging domain with a strong clinical phenotype, and that intestinal-barrier dysfunction, though supported by robust experimental and early human biomarker data, remains of limited routine clinical applicability — a hierarchy that helps the clinician weight the various markers and mechanisms encountered at the bedside [5].

6. Multi-Organ Involvement

EHS is, in its severe form, a disease of multiple organs, and the pattern of organ involvement both aids recognition — because systemic markers of injury help confirm the diagnosis when

temperature is equivocal — and shapes the intensive-care management that follows successful cooling. The organs most consistently affected are the CNS, skeletal muscle, kidneys, liver, heart and the coagulation system, and their involvement is driven by the same combination of direct heat cytotoxicity, systemic inflammation and endothelial and coagulation disturbance described above [2,5]. A well-documented community case illustrates the constellation vividly: a previously healthy 30-year-old man who collapsed during a 5-km run presented with encephalopathy and a generalised seizure, metabolic acidosis, acute kidney injury, rhabdomyolysis and myocardial injury, yet, with rapid cooling and supportive care, achieved complete normalisation of all biomarkers and cardiac findings at one-month follow-up [13]. This case demonstrates both the multi-system reach of EHS and its potential for full reversibility when treated correctly.

Rhabdomyolysis — the breakdown of skeletal muscle with release of its contents into the circulation — is among the most frequent and dangerous complications, occurring in roughly one in six to one in three hospitalised EHS patients [19]. It is diagnosed by markedly elevated creatine kinase and, when severe, by myoglobinuria and acute kidney injury; myoglobin released from damaged muscle can injure the kidneys through tubular obstruction, direct toxicity and vasoconstriction, and the accompanying hyperkalaemia raises the risk of fatal arrhythmia [19,20]. EHS and exertional rhabdomyolysis share a hypermetabolic pathophysiology and frequently co-occur, and their combination with acute kidney injury, particularly under the hot and humid conditions typical of endurance events, is especially hazardous [19,20]. The renal dimension is important in its own right: acute kidney injury is a common finding after prolonged exertion, and although its incidence varies widely with the diagnostic criteria used, its combination with dehydration, heat strain and myoglobinuria in EHS can progress to severe renal failure requiring intensive support [20]. The recognition that a substantial minority of survivors carry an elevated long-term risk of chronic kidney disease underscores that renal injury in EHS is not always transient [3,20].

Cardiac involvement is increasingly recognised and has particular relevance to recognition because it can mimic acute coronary syndrome. Mechanisms include direct thermal damage, systemic inflammation, catecholamine surge and endothelial dysfunction, which may provoke ischaemia, arrhythmias or transient systolic impairment [13]. Clinically this typically manifests as troponin elevation, conduction changes or tachy- and bradyarrhythmias during the acute phase [13]. In most young, previously healthy patients the troponin rise reflects reversible stress-related injury rather than structural heart disease; in the community case described above, high-sensitivity troponin I peaked markedly yet echocardiography showed preserved ventricular function and all cardiac markers normalised within a month [13]. However, population data from heatwave cohorts have linked troponin elevation with worse prognosis, and the possibility that a troponin rise unmasks genuine acute coronary syndrome cannot be dismissed, so that serial biomarker measurement, electrocardiography and echocardiography are recommended to differentiate transient myocardial stress from structural injury and to guide safe return to activity [13]. This pragmatic, serial-assessment approach is of particular value in resource-limited settings where advanced cardiac imaging may be unavailable [13].

Hepatic injury, coagulopathy and encephalopathy complete the picture of multi-organ dysfunction. Transaminase elevation is characteristic and may progress, in severe cases, to fulminant hepatic failure; coagulation disturbance ranges from mild abnormality to overt DIC, which as noted is a marker of severe disease and poor outcome; and CNS injury, expressed acutely as the defining encephalopathy of EHS, may leave lasting sequelae [2,5,6]. The severity and reversibility of this multi-organ involvement are strongly conditioned by the promptness of cooling and supportive care. Where these are delivered rapidly, as in the community case, even severe simultaneous dysfunction of muscle, kidney, heart and brain can resolve completely; where they are delayed, the same constellation may prove fatal or leave permanent damage [13]. This dependence on treatment timing is the thread that connects the pathophysiology of multi-organ injury back to the twin imperatives of recognition and cooling.

7. Risk Factors for Exertional Heat Stroke

Susceptibility to EHS is not uniform; it is determined by the interaction of environmental, individual, health-related, behavioural and organisational factors, and appreciating these factors sharpens recognition by identifying who is at risk and in what circumstances [1]. The environmental contribution is the most obvious. A linear relationship has been demonstrated between EHS incidence and the severity of the thermal environment as quantified by WBGT, and the risk rises with high temperature and humidity, low air movement and radiant heat load [1]. Yet environment alone is an incomplete predictor: EHS can occur in cool or temperate conditions when exercise intensity is high, and the exact prevalence across sports is obscured by inconsistent terminology, diagnostic criteria and reporting [1]. The interaction between environment and the demands of the activity is therefore crucial, and it is captured by the concept, discussed later, of matching the evaporative heat-loss requirement of the participant to the evaporative capacity of the environment [1].

The nature of the activity strongly modulates risk. In sport, American football has been identified as the discipline with the greatest number of EHS fatalities, a risk driven by high exercise intensity, protective equipment that creates an insulating microclimate and adds mass, and the static position-related activity of linemen that reduces self-generated air flow [1]. Endurance running carries the highest incidence of exertional heat illness of any sport, and, counter-intuitively, the risk of EHS increases as race distance decreases, because runners in shorter races sustain a faster pace and a higher rate of metabolic heat production, driving core temperature up more rapidly [1]. Team and intermittent sports are not exempt: a systematic review of core temperatures measured in competition across eleven intermittent sports found that the large majority of studies documented hyperthermia, with roughly seven in ten reporting "modest" hyperthermia (peak core temperature between 38.5 and 39.5 °C) and nearly one in five reporting "marked" hyperthermia at or above 39.5 °C, magnitudes associated with impaired performance and elevated heat-illness risk [21]. Elite athletes generate core temperatures above 39 °C during competition regardless of ambient conditions, and values up to 40.5 °C have been observed in hot conditions [21]. The relevance of these magnitudes to recognition is heightened by the effect of hyperthermia on cognition: experimental work in team-sport athletes has shown that heat exposure during prolonged intermittent exercise impairs complex decision-making, and a core-temperature threshold around 38.5 °C has been proposed as the point at which

cognitive deficits begin to appear, so that the same thermal strain that endangers the athlete may also erode the judgement needed to recognise and respond to it [22].

The quantitative epidemiology, though hampered by inconsistent terminology and reporting, provides a useful sense of scale and of the settings in which recognition and cooling capacity are most needed. In endurance running the reported rate of EHS varies by race and rises as distance falls: rates of roughly 3 to 4 per 10,000 athlete-exposures have been reported at marathons, but considerably higher rates — of the order of 10 to 21 per 10,000 — at 10-km road races, consistent with the higher intensities sustained over shorter distances [1]. The highest exertional-heat-illness incidence rates of all appear in endurance running and cycling, whereas team sports such as soccer and field hockey report much lower rates, and American football, though of intermediate incidence, accounts for the greatest number of fatalities [1]. In the military, where standardised case definitions and mandatory reporting permit more reliable surveillance, the overall EHS incidence has been reported at around 0.37 per 1,000 person-years, with markedly higher rates among recruits and in the Marine Corps and Army than in the Air Force and Navy, and with recognised risk factors including poor fitness, high body-mass index and initial-entry training [1]. A recurring and sobering observation from military data is that a large share of EHS casualties occurs during mandatory timed events such as runs and foot marches, driven by the extrinsic motivation to meet a standard, and that traditional risk factors are entirely absent in a substantial proportion of cases — a finding that underlines both the multifactorial nature of the condition and the impossibility of predicting every case in advance, and hence the enduring primacy of prompt recognition and cooling over risk prediction alone [1].

A striking and consistent finding across the literature is the magnitude of inter-individual variation in the physiological response to heat, which implies that risk cannot be predicted from environmental exposure alone. In a large controlled study of elite athletes exercising under simulated Tokyo 2020 conditions, peak gastrointestinal temperature ranged from 37.6 to 40.4 °C across individuals performing the same protocol, and the exercise-induced rise in core temperature and the loss of performance were highly variable and unrelated to one another [23]. Endurance-trained athletes reached higher peak core temperatures than mixed- or skill-trained athletes, a difference attributable to their higher metabolic heat production per unit mass, but the absence of a consistent association between core temperature and performance loss led the authors to conclude that a "one-size-fits-all" heat-mitigation strategy is untenable and that individual testing is required to identify those at greatest risk [23]. This heterogeneity is a recurring theme with direct implications for recognition: the same conditions that are safe for most participants may be dangerous for a susceptible minority, and vigilance cannot be relaxed simply because environmental thresholds appear tolerable.

Individual and behavioural factors further shape susceptibility. Poor physical fitness, high body-mass index, lack of heat acclimatisation, dehydration, sleep deprivation, recent illness and the use of certain medications and drugs are all recognised contributors [1]. Youth athletes represent a specific vulnerable group: a study of youth American football players documented the coexistence of extrinsic risk factors (disproportionate work-to-rest ratios and stressful environmental conditions, with most events observed in the higher WBGT flag categories) and

intrinsic factors (higher body-mass index, with roughly a third of players overweight or obese), and noted that prepubertal children thermoregulate differently from adults, with lower sweat rates and a longer acclimatisation process that render them potentially more vulnerable [24]. Paralympic and para-athletes may be at increased risk because impairments such as spinal cord injury disrupt sudomotor and vasomotor pathways and blunt thermoregulation; wheelchair athletes with cervical injury can experience hyperthermia even in temperate environments [25]. Reassuringly, a survey of Paralympic athletes at the hot and humid Tokyo 2020 Games found no medically diagnosed exertional heat illness despite the extreme conditions, an outcome attributed to the widespread use of heat acclimation and cooling strategies and to organisational countermeasures, demonstrating that susceptible populations can compete safely when appropriately protected [25].

Behavioural and psychological factors merit particular emphasis because they help explain the paradox that EHS strikes the fit and motivated. The motivation to excel — driven by competition, peer pressure, the desire to win or, in the military, the requirement to meet a standard — can lead individuals to override the sensory feedback from hyperthermia that would normally moderate their behaviour, sustaining an effort unmatched to their fitness [1,26]. This over-motivation has long been suspected as a risk factor, and a cognitive model of EHS proposes that the condition results from a disrupted cost–benefit trade-off during prolonged effort, in which the benefits of performance are overvalued and the physiological costs undervalued [26]. Empirical testing of this model found that individuals with a history of EHS reported significantly lower interoceptive awareness and reduced trait mindfulness than controls, suggesting that a diminished ability to sense and heed the body's internal warning signals may be an intrinsic vulnerability, even though a difference in global motivation traits was not confirmed [26]. In the cohort of patients reviewed by Kruijt and colleagues, peer pressure to perform was self-reported as a major contributor by more than a quarter of participants, and the sense of not being well acclimatised to the heat by more than half [19]. These findings converge on the conclusion that recognition of EHS begins before collapse, in the awareness that highly motivated individuals pushing beyond safe limits in the heat are the archetypal victims.

A final and clinically important risk factor is a previous episode of EHS, which has traditionally been regarded as predisposing to recurrence but which, until recently, rested on limited data. Analysis of seventeen years of records from a warm-weather road race with a high EHS rate identified sixteen individuals who suffered repeated episodes, accounting for 11 % of all EHS cases at that event, and demonstrated that the risk of a second episode was significantly elevated in the first two years following the initial event [27]. Whether this reflects a persistent, possibly genetic, susceptibility, a lingering impairment from the initial injury, or the same behavioural and environmental exposures recurring, remains debated, but the practical implication is clear: a history of EHS should heighten suspicion and prompt more conservative management and closer surveillance, particularly in the two years after the index event [27].

8. Recognition and Diagnosis of Exertional Heat Stroke

8.1. Clinical recognition

Because the outcome of EHS depends on the speed of treatment, and because treatment cannot begin until the condition is suspected, recognition is the first and arguably the most important step in the entire management pathway. The two clinical anchors of the diagnosis are CNS dysfunction and elevated core temperature, but in the field the former is often the more immediately available and the more discriminating [9,10]. Any alteration of mental status — confusion, agitation, irritability, disorientation, combativeness, ataxia, collapse, seizure or coma — arising during or immediately after strenuous exercise in the heat should raise the suspicion of EHS [9,10]. The clinical picture is completed by hot skin, which may be either dry or, in the exertional form, sweaty, together with tachycardia, tachypnoea and, in severe cases, hypotension [9]. Importantly, in exertional heat stroke sweating is frequently still present, and the classic teaching of "hot, dry skin" derived from classic heat stroke should not be used to exclude the exertional diagnosis; failure to sweat is a late rather than an early sign [9,10]. In the cohort of EHS and rhabdomyolysis patients reviewed by Kruijt and colleagues, the most frequently reported signs of CNS dysfunction were collapse, ataxia and confusion, confirming that neurological features dominate the presentation [19].

A crucial principle of recognition is that a core temperature threshold should never override the clinical context. Heat exhaustion, the milder condition with which EHS is most often confused, is characterised by volume depletion under heat stress with weakness, headache, nausea, dizziness, hypotension and tachycardia, but — critically — mental status remains intact [9]. The presence of any CNS alteration therefore distinguishes EHS from heat exhaustion, and the literature is explicit that any patient with neurological dysfunction after exertion in the heat must be treated as having EHS even if the measured temperature does not reach 41 °C [9]. Conversely, where collapse occurs during or immediately after heavy exertion in the heat and a valid core temperature cannot be obtained, EHS should be the presumptive diagnosis and treatment should not be delayed for confirmation [28]. This principle — treat first on suspicion, confirm later — is the field counterpart of the "cool first, transport second" imperative that governs the therapeutic side of management.

8.2. Measurement of core temperature

The single most consequential technical issue in the recognition of EHS is the measurement of core temperature, because the choice of measurement site determines whether the diagnosis is confirmed or falsely excluded. Rectal thermometry is the clinical reference standard and is the method endorsed by consensus and position statements for the diagnosis of EHS, because it provides a valid estimate of internal temperature in an exercising, hyperthermic individual and can be monitored continuously during treatment [9,10]. Gastrointestinal (ingestible telemetric pill) and oesophageal thermometry are similarly valid but are largely confined to research and specialised field settings [9]. The peripheral alternatives that dominate routine practice — oral, tympanic, temporal (temporal-artery) and axillary — are, by contrast, unreliable in this population: they are systematically affected by sweat, ambient conditions, wind and skin temperature, and they consistently underestimate the true core temperature of an exercising individual in the heat, with the potential to place a genuinely hyperthermic patient in a falsely reassuring temperature range [9–11]. A meta-analysis of aural thermometry in hyperthermic

exercising individuals found that it consistently underestimated core temperature relative to the rectal standard [28].

The clinical consequences of relying on invalid methods are not hypothetical. A survey of EMS providers in the United States found that temporal, tympanic and oral thermometers were the most commonly available devices, while only about one in ten providers had access to a rectal thermometer, and the use of these inaccurate devices risks the false exclusion of EHS when the measured temperature falls below 40.5 °C [10]. The same survey documented that fewer than a fifth of providers correctly identified rectal thermometry as the most acceptable method, and that overall knowledge of EHS best practices was poor despite years of experience and self-rated comfort with the condition [10]. This gap between confidence and competence is a recurring and troubling theme, and it implies that the barrier to correct recognition is often not the science but its dissemination and the provision of appropriate equipment [10].

Even with a valid method, the interpretation of temperature must account for the natural history of the condition. Because core temperature can decline rapidly after collapse, and because on-site cooling may already have been applied, a value below the diagnostic threshold does not exclude EHS, and the full clinical picture must take precedence [1,2,13]. This is particularly relevant in settings where rectal thermometry is unavailable for logistical or privacy reasons; in such circumstances the literature advises reliance on the complete clinical presentation rather than deferring treatment while awaiting an unobtainable confirmatory temperature [13]. The pragmatic synthesis is therefore twofold: obtain a rectal temperature wherever possible, and never allow the absence or the reassuring appearance of a peripheral temperature to delay the recognition and cooling of a patient whose clinical picture is consistent with EHS [9,10,13].

8.3. Differential diagnosis

Recognition of EHS also requires its differentiation from other causes of collapse with altered mental status during endurance events, because several of these conditions are life-threatening and some require management that is directly opposed to that of EHS [8]. The principal differential diagnoses are exercise-associated hyponatraemia, severe hypoglycaemia, sudden cardiac arrest and acute ischaemic stroke [8]. Exercise-associated hyponatraemia is the most important mimic, because it can present with confusion, seizures and coma indistinguishable from EHS at the bedside, yet it is typically associated with weight gain and a low serum sodium and is managed by fluid restriction or hypertonic saline rather than by cooling and rehydration [8,20]. The distinction matters greatly: exercise-associated hyponatraemia and exertional rhabdomyolysis, another concurrent condition, may require opposite fluid strategies, since hyponatraemia demands restriction of water intake while rhabdomyolysis demands aggressive hydration [20]. Hypoglycaemia usually presents with sweating, tremor and confusion that respond rapidly to glucose; sudden cardiac arrest requires immediate cardiopulmonary resuscitation and defibrillation; and acute ischaemic stroke is suggested by focal neurological signs [8]. The severe neurohumoral and cardiovascular stress of hyperthermia can itself provoke dangerous blood-pressure fluctuations and acute cardiovascular events, further complicating field assessment [8].

In the hospital, the differential broadens to include meningitis, encephalitis, malaria, thyroid storm, sepsis, and drug-related syndromes such as malignant hyperthermia, neuroleptic malignant syndrome and serotonin syndrome, none of which can be excluded by a single laboratory test [9]. Laboratory features that support a diagnosis of heat stroke include leucocytosis, elevated urea and creatinine, raised transaminases and creatine kinase, metabolic acidosis and, on urinalysis, myoglobinuria [9]. Hyperglycaemia is more often associated with classic heat stroke, whereas hypoglycaemia, though rare, tends to occur in the exertional form [9]. The practical message is that recognition of EHS in the field is essentially clinical, resting on the triad of context, neurological dysfunction and — where obtainable — core temperature, and that the differential-diagnostic refinement afforded by laboratory testing should never delay the initiation of cooling when EHS is suspected [8,9].

8.4. Biomarkers

The limitations of temperature as a stand-alone diagnostic have stimulated interest in biomarkers that might aid the recognition, risk stratification and prognostication of EHS, particularly in the hospital phase. A wide array has been investigated, reflecting the multi-organ nature of the injury: markers of muscle damage (creatine kinase, myoglobin), cardiac injury (high-sensitivity troponin I), coagulation disturbance and DIC risk (D-dimer, thrombin–antithrombin complex, platelet count, prothrombin and partial thromboplastin times), systemic inflammation (IL-6, TNF-α, HMGB1), CNS injury (S100β, neuron-specific enolase), renal injury (neutrophil gelatinase-associated lipocalin, kidney injury molecule-1), hepatic injury (transaminases), intestinal barrier injury (intestinal fatty-acid-binding protein) and endothelial dysfunction or glycocalyx shedding (von Willebrand factor antigen, soluble thrombomodulin, syndecan-1) [5,6]. A useful appraisal groups these into practical tiers: routinely available but non-specific markers such as creatine kinase, troponin, D-dimer and coagulation times, which support early recognition and severity assessment but lack heat-stroke specificity; markers with growing evidence such as HMGB1, S100β, neutrophil gelatinase-associated lipocalin and intestinal fatty-acid-binding protein, which show prognostic potential but are limited by inter-study variability and the absence of standardised cut-offs; and experimental indicators of endothelial and glycocalyx injury that presently illuminate pathophysiology but require validation before bedside adoption [5].

Two limitations temper the enthusiasm for biomarkers. First, a core temperature above 40 °C, though a defining feature, is itself not diagnostic in isolation, and no biomarker has yet supplanted the clinical and temperature-based recognition of EHS [5]. Second, the clinical utility of even the most promising markers is constrained by inter-individual variability and the lack of standardised thresholds, so that they currently serve to complement rather than to replace clinical judgement [5,6]. Where advanced markers and imaging are unavailable, as in many low- and middle-income and field settings, structured serial measurement of routinely available biomarkers — creatine kinase, creatinine, troponin — together with electrocardiography and echocardiography offers a pragmatic and cost-effective alternative for monitoring organ recovery and guiding return to activity [13]. Biomarkers, in short, are a valuable adjunct to recognition and, especially, to the assessment of severity and recovery, but they do not alter the fundamental principle that EHS is recognised clinically and treated urgently.

8.5. Wearable and predictive technologies for early detection

A conceptually distinct approach to recognition seeks to detect dangerous hyperthermia before overt collapse, using wearable sensors and predictive algorithms to monitor core temperature or heat-stress risk continuously in at-risk individuals. The rationale is compelling: because mortality falls from as high as 80 % with delayed cooling to essentially zero when an elevated core temperature is detected and reduced below 40 °C within thirty minutes of symptom onset, any technology that shortens the interval to recognition could be lifesaving [29]. The central obstacle is that valid core-temperature measurement during exercise currently requires invasive or obstructive methods — rectal, oesophageal or ingestible telemetric devices — that are impractical for routine continuous monitoring, while the peripheral and skin-based alternatives are unreliable in the exercising individual [29].

Two strategies have been pursued. The first estimates core temperature non-invasively from physiological signals — most commonly heart rate, alone or combined with skin temperature and skin heat flux — using predictive algorithms, frequently based on Kalman filters or machine learning. A systematic review of twenty studies yielding twenty-five algorithms found that non-invasive prediction of core temperature is achievable with clinically acceptable accuracy under controlled conditions, with seventeen of eighteen algorithms reporting a root-mean-square error at or below the accepted threshold of 0.5 °C and an average error around 0.28 °C after exclusion of an outlier [29]. However, the review also exposed important limitations: few algorithms incorporated individual and environmental variables despite the known influence of these factors, validation was largely confined to laboratory settings and homogeneous, predominantly young male subjects, and there was a conspicuous lack of validation in the high-school athletes who constitute the largest group presenting to emergency departments with heat illness [29]. The second strategy monitors environmental and physiological parameters to compute a heat-stroke risk index rather than an absolute temperature. Prototype devices integrating ambient temperature and humidity, body temperature and heart rate with a heat-index calculation have been developed for athletes, and supervised machine-learning systems using personalised vital signs have been designed for occupational settings, where a personalised heat-strain temperature index combined with heart rate and activity achieved a prevention accuracy of around 85 % in a hot working environment [30,31]. These systems face their own constraints — the reliability of individual sensors, the need for individualisation, connectivity requirements and, above all, the gap between controlled validation and dynamic real-world use — and most remain at the prototype or pilot stage [30,31]. Nonetheless, they represent a plausible future adjunct to recognition, and the broader literature envisages the integration of biomarker profiles with artificial-intelligence tools to enhance pre-hospital triage and individualised risk prediction in vulnerable populations [6]. For the present, however, these technologies supplement rather than replace the clinical recognition of EHS, and their promise should not distract from the immediate and proven priority of measuring core temperature correctly and cooling aggressively when EHS is suspected [11].

9. Cooling Protocols

9.1. The governing principles: cooling rate, the therapeutic window and "cool first, transport second"

Cooling is the definitive treatment of EHS, and the evidence that governs it can be distilled into a small number of robust principles. The first is that the determinant of outcome is not the peak temperature reached but the duration for which the patient remains dangerously hyperthermic — the area under the temperature-versus-time curve — so that the goal of treatment is to minimise this area by lowering core temperature as rapidly as possible [4,28]. The second is that a therapeutic window of approximately thirty minutes exists, within which reduction of core temperature below the critical threshold prevents irreversible organ damage; cooling to below 40 °C within thirty minutes of collapse is repeatedly associated with survival, and mortality that may reach 80 % with delayed cooling falls toward zero when this window is respected [3,9,11]. The third, and most quantitatively important, is that the rate of cooling must be adequate: systematic analysis has established a threshold of 0.15 °C per minute, above which cooling is considered "adequate" and below which it is "insufficient," and this threshold discriminates survival with striking clarity [4].

The evidence for the cooling-rate threshold is among the strongest in the field and deserves emphasis because it directly guides the choice of modality. In the largest dataset assembled on the question, a systematic review of thirty-two case reports representing 521 EHS patients from sporting and military settings, no patient died when treatment achieved an adequate cooling rate above 0.15 °C per minute, whereas all twenty-three deaths occurred among patients cooled insufficiently [4]. Moreover, among survivors, those cooled insufficiently carried a 4.57-fold increased risk of medical complications, and 22.5 % of them survived with complications compared with only 0.77 % of adequately cooled patients [4]. The same evidence, independently appraised in the community-cooling literature, is summarised as a 0 % case-fatality rate when the cooling rate exceeded 0.15 °C per minute against 4.4 % when it did not, and a complication rate among survivors of 0.77 % versus 22.5 % [11]. The convergence of these analyses provides an unusually clear, quantitatively defined target for treatment: any modality selected for EHS should be capable of a cooling rate above 0.15 °C per minute, and modalities that cannot achieve this should not be relied upon as primary treatment [4,11].

The operational corollary of these principles is the mantra "cool first, transport second." Because the therapeutic window is short and cooling is the intervention that saves life, the priority at the scene is to lower core temperature on site, before and, if necessary, during transport, rather than to rush the patient to hospital uncooled [4,9,12]. EHS is, in this respect, unusual among emergencies in that definitive treatment can and should be delivered in the field. The principle has been endorsed by consensus statements and incorporated into prehospital protocols for major events, yet its implementation is inconsistent, as discussed below [9,12]. Where on-site cooling to a target core temperature — commonly around 38.9 °C — can be achieved before transport, this is the recommended approach; where it cannot, the most aggressive available cooling should be continued during transport [9,11].

9.2. Water as the optimal cooling medium

The pre-eminence of water-based cooling rests on the physical properties of water, which make it a far more effective medium for heat transfer than air. Water has a thermal conductivity roughly twenty-four times that of air, a high specific heat capacity, and a density far exceeding that of air, so that it can absorb large quantities of heat with minimal temperature rise and can cool the body approximately four times faster than air at the same temperature [11]. When the body is immersed, water contacts nearly the entire body surface, maximising conductive heat loss, and continuous movement or circulation of the water prevents the formation of a warmed boundary layer at the skin, maintaining the steep thermal gradient that drives cooling [4,11,32]. A further, physiologically favourable feature is that in a genuinely hyperthermic patient the large temperature gradient between body and water overwhelms any brief cutaneous vasoconstriction, so that the theoretical concern that cold water might impede heat dissipation through vasoconstriction is not borne out in practice [4]. These properties explain why every high-efficiency cooling modality identified in the literature involves water applied to a large body-surface area, and why air-based methods are comparatively feeble.

9.3. Cold-water immersion: the gold standard

Cold-water immersion (CWI) is the gold-standard treatment of EHS, endorsed across the sporting, military and emergency-medicine literature [4,9,11,12]. Its cooling rate, typically in the range of 0.15 to 0.35 °C per minute, comfortably exceeds the adequacy threshold, and it maximises convective and conductive heat loss over a large surface area simultaneously [4,11]. The clinical evidence for its efficacy is exemplified by the experience of a summertime road race with a high EHS rate, at which whole-body CWI was associated with a 100 % survival rate among 274 EHS patients presenting with initial rectal temperatures between 40.0 and 42.8 °C [4]. In the pooled dataset of Filep and colleagues, every one of the 348 patients treated with CWI was cooled below 40 °C and survived without medical complications [4]. Supervised settings that combine rapid recognition with immediate CWI have repeatedly achieved case-fatality rates at or near zero: outcomes from long-distance running events over eighteen years demonstrated a 0 % fatality rate when CWI at around 10 °C was begun within two minutes of collapse, and a military cohort recorded a case-fatality below 2 % with a high proportion of patients receiving prehospital cooling [11]. On the strength of such data, CWI has been adopted into the prehospital protocols of major sporting events and expert guidelines [11].

9.4. Body-surface area and depth of immersion

Because cooling by water depends on the proportion of the body surface exposed and on the maintenance of the thermal gradient, the depth of immersion and the circulation of the water materially affect the cooling rate — a point of considerable practical importance when full CWI is impractical. A systematic review of water-immersion cooling in firefighters, examining fifty-five treatments, contrasted hand-and-forearm immersion (HFI) with multi-segment immersion (MSI) of the torso, pelvis and lower body [32]. The findings were unequivocal: MSI achieved cooling rates spanning approximately 0.01 to 0.35 °C per minute, generally classified as ideal or acceptable, whereas every HFI treatment produced an unacceptably slow rate of only 0.01 to 0.05 °C per minute [32]. The highest rate, 0.35 °C per minute, was obtained with immersion of about 93 % of the body surface in circulated 2 °C water, and the review concluded that

maximising the immersed surface area — ideally "neck-out" immersion — in circulated water is the key to rapid cooling [32]. Although the hands and forearms are highly vascular, their small surface area (roughly 19 % of the body) limits their cooling power, and the recommendation to reassess the reliance on HFI in favour of MSI follows directly [32]. Circulation of the water is a further amplifier, roughly doubling the cooling rate for a given water temperature and immersion depth by preventing warming of the boundary layer, and it becomes particularly important when only temperate water is available [32]. These principles — large surface area, adequate depth, cold and circulated water — define the ideal application of immersion cooling and inform the design of practical field alternatives.

Two physiological considerations refine the interpretation of immersion-cooling data and, occasionally, its practice. The first is the "afterdrop" phenomenon, whereby the redistribution of body heat to previously cooled peripheral tissues continues to lower core temperature after immersion has ceased; because cooling rates are frequently reported from the temperature measured immediately at the end of immersion, they may underestimate the true cooling power of a modality when the afterdrop is not accounted for [32]. In one firefighter study, lower-body immersion reduced core temperature by around 0.5 °C during cooling but by a further 0.8 °C after cooling ended, so that less than half of the total decrease occurred during the immersion itself, and the reported rate was correspondingly conservative [32]. The afterdrop is proportional to the cooling stimulus — water temperature, duration and body-surface area cooled — and is therefore more relevant to multi-segment than to hand-and-forearm immersion, but it does not rescue the inadequate cooling rates of the latter, which remain unacceptable even when the afterdrop is considered [32]. The second consideration is the frequently voiced concern that immersion in cold water might paradoxically impede cooling through cutaneous vasoconstriction, trapping heat in the core. The evidence indicates that this concern is misplaced in the genuinely hyperthermic patient: the large temperature gradient between the body and the water overwhelms any brief vasoconstriction, no case series has demonstrated attenuated cooling attributable to it, and the theoretical objection has repeatedly been shown not to compromise the cooling rate in practice [4]. These two points together correct a common under-appreciation of immersion cooling — that its true power is sometimes understated in the literature by the unmeasured afterdrop, and that a longstanding physiological objection to it is not borne out — and they reinforce the case for immersion as the treatment of choice [4,32].

A related practical question concerns the influence of pre-treatment core temperature and immersion duration on the cooling rate, which bears directly on the interpretation of field data. Because cooling proceeds down a temperature gradient, immersion that begins at a higher core temperature establishes a steeper gradient and therefore a faster initial cooling rate; conversely, when cooling begins at a relatively low temperature, as is common when a delay has allowed some spontaneous decline, the measured rate is lower [32]. This gradient effect explains part of the variation in reported cooling rates and cautions against comparing rates measured from very different starting temperatures. It also carries a reassuring corollary for the sickest patients: immersion tends to be most effective precisely when it is most urgently needed, that is, when the core temperature is highest and the gradient steepest [32]. Immersion duration interacts with this, since a large increase in duration can produce a disproportionately smaller gain in cooling, so that short, cold, high-surface-area immersions of the hyperthermic patient deliver the most

rapid cooling [32]. These nuances do not alter the central recommendation, but they help the clinician interpret the heterogeneous cooling-rate literature and set realistic expectations for the time required to bring a patient safely out of the dangerous range.

9.5. Field alternatives to cold-water immersion

When ample cold water and a suitable tub are unavailable — as is common in remote, occupational and low-resource settings — alternative field modalities must be used, and these differ substantially in efficacy. The literature provides comparative cooling rates that allow these methods to be ranked against the 0.15 °C per minute threshold (Table 1) [4,11,28]. Tarp-assisted cooling with oscillation (TACO), which improvises immersion using a tarpaulin and locally available water, achieves cooling rates of approximately 0.14 to 0.17 °C per minute — at or near the adequacy threshold — and is recommended as a feasible alternative when a dedicated tub is not available, although it requires several personnel and a substantial volume of water [4,28]. The rotation or repeated application of ice-cold wet towels or sheets over the torso and limbs is a portable, single-operator method that has produced cooling rates around 0.11 °C per minute in athletes and up to 0.16 °C per minute for ice-sheet cooling of casualties with an initial core temperature at or above 39 °C in military settings [28]. Continuous dousing with cold water combined with ice-bag massage and fanning has also achieved adequate rates and features among the CWI-equivalent combinations in survival series [4].

Below these effective methods lies a group of modalities with cooling rates too slow to be relied upon as primary treatment. Ice packs applied to the major arteries at the neck, axillae and groin, cold intravenous fluids, cooling blankets, evaporative cooling with fans and spray used alone, and single-fan cooling all produce insufficient cooling rates and are associated with worse outcomes when used as the main modality [4,11]. Kielblock and colleagues' classic comparison found that topical ice-pack application cooled far more slowly than either whole-body ice coverage or evaporative cooling, and combined splashing, fanning and cold-pack application to the groin, neck and axillae achieved only around 0.036 °C per minute, roughly half the minimum acceptable rate [28]. Evaporative cooling — spraying the patient with atomised water while fanning warm air over the body — has a cooling rate of approximately 0.08 °C per minute, slower than immersion but with the practical advantages of fewer logistical demands, easier concurrent monitoring and resuscitation, and a reduced shivering response, which is why it retains a role in the emergency department [9]. These modalities are best regarded as adjuncts or as interim measures during transport when superior methods are unavailable, rather than as substitutes for immersion [4,9].

Table 1. Approximate core-temperature cooling rates of modalities used for exertional heat stroke and exertional hyperthermia, ranked against the adequacy threshold of 0.15 °C·min⁻¹ [4,9,11,28].

Cooling modality	Approximate cooling rate (°C·min⁻¹)	Classification
Ice-water immersion (~2 °C, large surface area, circulated)	up to 0.35	Ideal

Cold-water immersion (~10 °C)	0.15–0.35	Ideal / adequate
Multi-segment (neck-out) immersion, cold circulated water	0.10–0.35	Ideal / acceptable
Tarp-assisted cooling with oscillation	0.14–0.17	Adequate
Ice sheets (initial core temperature ≥ 39 °C)	~0.16	Adequate
Ice/wet towels rotated over torso and limbs	~0.11	Borderline
Evaporative cooling (mist and fan)	~0.08	Insufficient
Chemically activated cooling vest	~0.06	Insufficient
Cold intravenous saline	~0.07	Insufficient
Combined splashing, fanning and cold packs	~0.036	Insufficient
Ice packs to major arteries only	~0.02–0.05	Insufficient
Hand-and-forearm immersion	0.01–0.05	Insufficient
Passive rest	~0.004–0.03	Insufficient

The place of cooling vests warrants specific comment because their portability makes them attractive and their limitations are instructive. A randomised crossover study evaluated a chemically activated cooling vest against passive rest in individuals rendered hyperthermic (rectal temperature above 39 °C) by exercise in a hot environment [33]. Although the vest cooled significantly faster than passive rest, its cooling rate was only about 0.06 °C per minute — far below the 0.15 °C per minute standard — and the authors calculated that, extrapolated to a patient presenting at 41.4 °C, the vest would require some 41 minutes to cool to a safe temperature, and 64 minutes from 42.8 °C, well beyond the therapeutic window [33]. Notably, anthropometric differences between individuals did not confer additional benefit, and the study concluded unambiguously that the vest should not replace whole-body CWI in the treatment of EHS [33]. This finding generalises to the broader category of small-surface-area and convection-dependent devices: their convenience does not compensate for their inadequate cooling power, and they are appropriate only as a bridge to definitive cooling, for example while a patient is transported to a location where immersion can be performed [33].

A practical occupational case study illustrates how these principles translate into the field. In a remote industrial setting, a worker with suspected EHS was cooled by a single colleague using

the ice-towel method — submerging towels in iced water and applying them to the torso — from a portable "heat-stroke kit" consisting of an insulated bin and towels carried in each field vehicle [28]. The method proved cost-effective, portable, scalable and simple enough to be applied by one person under the stress of an emergency, and paramedics judged that the prompt cooling had likely averted a far more serious outcome [28]. This case demonstrates the value of matching the cooling modality to the constraints of the setting: where CWI is impractical, an effective, transportable alternative that can be applied immediately by available personnel is preferable to delaying cooling until a superior method arrives [28]. The general lesson is that the best cooling modality is the most effective one that can actually be applied without delay in the specific circumstances of the collapse [21,28].

9.6. Internal and invasive cooling methods

Internal cooling methods have a role chiefly in the hospital setting or when external cooling is constrained, but they are generally slower and more resource-intensive than immersion. Cold intravenous fluid infusion cools at approximately 0.07 °C per minute and is limited by the need for medical personnel and venous access; its combination with high-dose diazepam has been shown to enhance core cooling in volunteers without adverse cardiovascular effects, and rapid infusion of chilled saline is a practical component of ambulance-based internal cooling where immersion is impossible [11]. Gastric lavage with ice-cold saline can achieve cooling rates up to 2.6 °C per hour and is valuable when external cooling is constrained, for example in the confined space of an ambulance [11]. Peritoneal lavage has a marked cooling capacity, up to 0.5 °C per minute, but is invasive and should not be a first-line therapy when non-invasive methods can be effective [9]. Endovascular (intravascular balloon-catheter) cooling devices have been used in small numbers of severe cases and can provide rapid, accurate core cooling — one report achieved a mean rate of 0.1 °C per minute, reaching 38.8 °C in seventeen minutes — and a prospective feasibility study of a convection-based intravascular device combined with conventional cooling reported a significant reduction in organ-failure scores and favourable neurological outcomes, but no prospective comparative study has confirmed the superiority of any particular internal cooling method, and these devices require catheter placement and specialised resources [2]. For the community-acquired patient in whom the thirty-minute window has been missed, aggressive cooling on arrival at the emergency department — ideally ice-water immersion, which at around 2 °C can achieve at least 0.2 °C per minute — remains imperative and should never be delayed for diagnostic procedures such as intubation or computed tomography [11].

Beyond cooling itself, blood-purification therapies have been explored as means of removing the inflammatory mediators and toxic metabolites that drive multi-organ injury. Continuous renal replacement therapy, continuous venovenous haemofiltration, plasma exchange and plasma diafiltration have been reported to aid recovery in severe heat stroke complicated by multi-organ failure, with one retrospective study reporting significantly lower thirty-day mortality with continuous renal replacement therapy than with routine treatment [2]. Continuous renal replacement therapy can reduce the inflammatory response, clear toxic metabolites and correct fluid, electrolyte and acid–base disturbance [9]. Anticoagulant strategies — antithrombin and recombinant thrombomodulin — have shown promise in case

reports and animal models of heat-stroke-induced DIC [2]. However, the evidence for all of these adjuncts rests on case reports and retrospective series; no prospective comparative study has confirmed the efficacy of blood purification, anticoagulation or specific vasoactive strategies in heat stroke, and their use remains inconclusive [2]. This uncertainty stands in sharp contrast to the robust evidence for rapid external cooling and reinforces the point that, whatever the potential of adjunctive therapies, the proven determinant of survival remains the speed with which core temperature is lowered [2,4].

9.7. Cooling across settings: from elite sport to the community

The application of these cooling principles must be adapted to the setting, and the distinction between supervised and community-acquired EHS is fundamental to understanding where the greatest gains — and the greatest failures — occur. In supervised settings such as athletic events and military training, where medical personnel and cooling resources are present, immediate CWI can be applied and case-fatality falls to near zero; the sub-classification of heat stroke into supervised and community-acquired forms captures precisely this influence of healthcare accessibility on outcome [11]. Community-acquired heat stroke, by contrast, arises where immediate medical assistance and cooling resources are lacking, so that patients depend on emergency medical services that may take ten to thirty minutes or longer to arrive, on ambulances frequently ill-equipped for cooling, and on emergency departments where unfamiliarity with heat-stroke management may further delay effective cooling [11]. The strategies proposed to close this gap — public education in the recognition and first-aid cooling of heat stroke, dispatcher-guided cooling instructions to bystanders, and the equipping of ambulances with practical internal-cooling capability such as chilled saline for wiping and infusion and ice-saline gastric lavage — are all directed at bringing effective cooling forward in time to the community setting where it is currently weakest [11].

Occupational settings present their own constraints. Emergency personnel — firefighters, military personnel and others — who work in high-temperature environments while wearing personal protective equipment experience substantial thermal strain, and the encapsulating equipment that protects them also impairs heat dissipation and complicates cooling [34]. A systematic review and meta-analysis of cooling strategies for such personnel found that cooling interventions significantly reduced core temperature, heart rate and sweat rate and improved tolerance time, with intermittent and per-cooling approaches, and particularly mixed-method strategies combining cooling vests with immersion or ice-slurry ingestion, yielding the greatest benefit [34]. Yet the same review noted that the most effective interventions require time, equipment and logistical planning that may not be feasible during operations, and that fewer than 15 % of emergency personnel in surveys report using advanced cooling strategies, relying instead on basic measures such as resting in shade, drinking cold water and removing equipment [34]. This gap between demonstrated efficacy and field adoption echoes the broader problem in EHS and points to organisational and cultural barriers as much as scientific ones [34].

Community events in low- and middle-income countries constitute a setting of particular vulnerability, and one where the adaptation of cooling to available resources is most consequential. The evidence base for EHS is heavily skewed toward high-resource settings — a systematic review found that the great majority of exertional-heat-illness studies originate

from the United States — and preparedness in many low- and middle-income settings remains underdeveloped, with rectal thermometry often unavailable and structured emergency response limited [13]. The reported community case from Vietnam exemplifies both the risk and the solution: on-site staff initiated cooling with ice packs and cold towels and infused fluids before transfer, and the patient, despite severe multi-organ dysfunction, recovered fully, illustrating that practical, low-cost measures — tarp-assisted immersion with locally available cold water, or large-volume ice packs where immersion tanks are not feasible — can be lifesaving even without sophisticated infrastructure [13]. The consistent recommendation is that simple, locally available cooling capacity, combined with trained staff and clear transfer protocols, should be integrated into the planning of community sporting events, particularly as endurance events grow in popularity in these regions [13].

9.8. When to stop cooling

A final practical parameter of any cooling protocol is the endpoint. Because aggressive cooling, if continued unchecked, risks overshooting into hypothermia, cooling should be discontinued once core temperature, measured by a rectal probe, reaches approximately 38.9 °C (102 °F), a target that has been used safely in large series and that leaves a margin above normothermia [2,9]. Continuous monitoring of rectal temperature during cooling is therefore recommended so that the endpoint can be judged accurately [9]. Where rectal temperature is unavailable, protocols guided by immersion duration have been derived: immersion in water at or below 9 °C for eleven to twelve minutes, or in 10–26 °C water for eighteen to nineteen minutes, has been shown to reduce core temperature safely within therapeutic targets [11]. The definition of a clear cooling endpoint completes the protocol and guards against the iatrogenic hazard of over-cooling, while ensuring that the patient is brought reliably out of the dangerous hyperthermic range [2,9,11].

10. The Gap Between Evidence and Practice

A distinctive and sobering feature of the EHS literature is the breadth of the gap between what the evidence establishes and what is actually done, particularly in the prehospital phase, and this gap is arguably the principal remediable cause of preventable death and complication. The evidence for rapid, adequate cooling is unusually strong and quantitatively precise, yet surveys of the systems responsible for delivering it reveal pervasive shortcomings. A review of EMS protocols across all fifty United States and the District of Columbia found that only about 18 % explicitly recommended CWI and only about 22 % specifically permitted cooling to be completed before transport; more alarmingly, roughly 12 % of protocols instructed providers with some variation of the phrase "do not delay transport to cool the patient" — the exact inversion of the "cool first, transport second" principle — and when protocols that did not address CWI at all were included, CWI was not correctly recommended in nearly four in five protocols [12]. This means that a majority of prehospital systems either fail to endorse or actively discourage the single intervention most strongly associated with survival [12].

The problem extends from protocols to knowledge and equipment. In the survey of EMS providers, only about 9 % had read the relevant consensus statement on prehospital care of EHS, the average score on a knowledge assessment was approximately 37 %, and while most

providers rated themselves comfortable recognising and managing EHS, this confidence was not matched by competence [10]. Access to the correct tools was similarly deficient: only about 10 % of providers had a rectal thermometer available, and the most common cooling supplies were chemical cold packs and air conditioning — modalities that, as shown above, cannot achieve adequate cooling rates [10]. The consequence is a system poorly configured to recognise EHS (through the absence of valid temperature measurement) and poorly configured to treat it (through the absence of adequate cooling capacity and the presence of counterproductive transport-first protocols). Closing this gap requires updated, evidence-based protocols that explicitly recommend CWI and permit on-site cooling, the provision of rectal thermometers and immersion capability, and continuing education across all provider levels, together with collaboration between sports-medicine and emergency-medicine communities so that a patient in whom cooling has been correctly initiated on site is not removed from that cooling by responders following an outdated protocol [10,12]. The recurrence of this evidence–practice gap across EMS, occupational and community settings suggests that the frontier of EHS care lies less in new science than in the implementation of what is already firmly established [10,12,34].

The patient-level consequences of inconsistent management have been documented directly. In a combined survey and medical-record review of sixty athletes and military personnel who had suffered EHS or exertional rhabdomyolysis, prehospital management was found to be inconsistent and, in the majority of cases, not conducted according to available guidelines; cooling methods used on site included ice packs, running water and ice baths, but nearly a quarter of participants reported not being cooled on site at all [19]. This real-world variability, occurring in a high-income country with a developed health system, underscores that the failure to translate the cooling evidence into practice is not confined to under-resourced settings but is a general phenomenon requiring systematic correction [19].

11. Prevention and Heat Mitigation

Although recognition and cooling determine the outcome of an established EHS, the most effective strategy of all is to prevent the condition from developing, and the literature describes a coherent set of mitigation measures directed at the modifiable determinants of risk [1]. These measures operate at the level of the individual, the event and the organisation, and they are relevant to the present review because they define the context within which recognition and cooling occur and because their partial and inconsistent adoption mirrors the implementation problems seen in treatment.

Heat acclimatisation and acclimation are the most important individual countermeasures. Repeated exposure to exercise in the heat, whether in a natural (acclimatisation) or artificial (acclimation) environment, produces adaptations that reduce physiological strain and the susceptibility to heat illness: enhanced sweating and skin-blood-flow responses, expansion of plasma volume, improved cardiovascular stability and acquired cellular thermotolerance, together lowering resting and exercising core temperature for a given load [1,15]. Detailed heat-exchange measurements have shown that a period of heat acclimation following summer acclimatisation shifts heat dissipation toward the evaporative avenue, increasing evaporative heat loss and thereby lowering internal temperature, and that the induction of these adaptations

depends on achieving sufficient hyperthermia and sweating during the acclimation sessions [15]. The optimal acclimatisation period is generally two weeks, and its benefits are substantial; yet uptake is often poor. Among ultra-endurance runners preparing for a tropical event, only about a quarter of those living in temperate climates had trained in the heat beforehand, and the average preparation fell short of the recommended duration [35]. This under-use of acclimatisation, attributed in part to a lack of awareness of the risk and to the practical constraints faced by amateur athletes, represents a missed preventive opportunity, particularly given evidence that residing in a hot climate confers little protection unless the individual has actually been exposed to heat in the preceding weeks — that is, unless seasonal acclimatisation has occurred [35].

The mechanistic basis of these adaptations reinforces their preventive value and their relevance to recognition. Partitional calorimetry in trained endurance athletes has shown that heat acclimation increases maximal skin wettedness and evaporative heat loss and shifts a greater proportion of total heat dissipation onto the evaporative avenue, so that the acclimated athlete achieves a lower internal temperature for the same metabolic heat production [15]. Notably, in trained individuals the reduction in core temperature after acclimation is driven principally by enhanced evaporative heat loss rather than by a reduction in metabolic heat production, a pattern that differs from that observed in untrained individuals and that highlights the population-specific nature of heat adaptation [15]. The practical corollary is that the induction of acclimation requires the deliberate achievement of hyperthermia and copious sweating during training sessions, and that its protective effect operates by widening the margin between heat production and the environment's evaporative capacity — the very margin whose collapse produces uncompensable heat stress and EHS [15]. Understood in these terms, acclimation is not a vague conditioning benefit but a specific physiological intervention that directly addresses the heat-balance failure at the root of the syndrome, and its systematic neglect among recreational and amateur athletes is therefore a substantive, not merely a theoretical, gap in prevention [15,35].

A distinct and less familiar avenue of prevention derives from the gastrointestinal paradigm of EHS. Because sustained exertional-heat strain damages the intestinal barrier and permits the translocation of microbial products that drive systemic inflammation, measures that preserve gut-barrier integrity have been proposed as nutritional countermeasures against EHS, complementing the conventional thermoregulatory approach of cooling and acclimation [3]. Although consensus documents on this strategy are not yet available and the direct human evidence remains limited by the ethical impossibility of inducing EHS experimentally, the rationale is coherent and the barrier can be assessed non-invasively, which supports its investigation as an adjunctive preventive target [3]. This gastrointestinal dimension intersects with hydration, since dehydration compounds splanchnic hypoperfusion and barrier injury, and with the wider nutritional management of endurance exercise. In ultra-endurance running, hydration practices are highly variable and are complicated by the competing hazards of under-hydration, which raises the risk of heat illness and of the acute kidney injury that frequently accompanies prolonged exertion, and over-hydration, which risks exercise-associated hyponatraemia; the balance of recent guidance favours individualised hydration governed substantially by thirst rather than rigid volume targets, together with judicious sodium and

carbohydrate intake [20,35]. The convergence of the thermoregulatory, gastrointestinal and hydration strands illustrates that prevention, like recognition and cooling, is most effective when it is individualised and mechanistically informed, and that the reduction of EHS risk is served not by any single measure but by the coherent application of acclimation, hydration, cooling and organisational policy tailored to the athlete and the event [3,20,35].

Adequate hydration is a second pillar of individual prevention, though its role is more nuanced than sometimes assumed. Severe dehydration impairs thermoregulation by reducing the sensitivity of the sweating and skin-blood-flow responses, and euhydration before exercise with replacement of a portion of sweat losses during exercise is recommended to mitigate the compounding effects of hyperthermia and dehydration [1]. However, the hydration question is complicated by the opposite hazard of over-drinking, which can precipitate exercise-associated hyponatraemia, and the appropriate strategy — drinking to thirst versus a programmed schedule — remains debated and depends on the duration and intensity of the activity [35]. Among ultra-endurance runners, hydration practices were found to be highly variable, and recent guidance cautions against both under-hydration, which raises the risk of heat illness and renal injury, and over-hydration, which risks hyponatraemia [20,35]. The practical implication for prevention is individualisation rather than a single universal fluid prescription, and the recognition that hydration is one contributor among several rather than a panacea [1,35].

Cooling can also be applied preventively, before (pre-cooling) or during (per-cooling) exercise, to increase heat-storage capacity and blunt the rise in core temperature. Internal pre-cooling by the ingestion of cold fluids or crushed ice has been shown to lower pre-exercise core temperature and to extend the time to reach a critical temperature: ingestion of crushed ice before and during cycling in the heat reduced rectal temperature by around 0.3 °C relative to cold water and prolonged endurance capacity, an effect attributed to the additional heat absorbed by the phase change of ice to water [36]. Ingestion of ice slurries similarly lowers core and tympanic temperature and can be combined with carbohydrate to maintain substrate availability [37], and pre-exercise ice-slurry ingestion has been shown to mitigate the hyperthermia-induced hyperventilation and cerebral hypoperfusion that accompany exercise in the heat, primarily by lowering core temperature [38]. External pre-cooling and per-cooling strategies — cooling vests, cold towels, water sprays and immersion — are widely used by athletes; a survey of Paralympic athletes at Tokyo 2020 found that cooling was employed by roughly three-quarters of respondents, most commonly cold towels and packs, beverages and water sprays, with most combining pre- and per-cooling [25]. The evidence from the Thermo Tokyo study that the physiological response to heat is highly individual reinforces the conclusion, common to acclimatisation, hydration and cooling, that heat-mitigation strategies should be tailored to the individual athlete's thermoregulatory response rather than applied uniformly [23].

At the level of the event and the organisation, environmental monitoring and activity-modification policies are the principal tools. The WBGT index is the most widely used measure of environmental heat-stress risk and underlies the heat policies of major sporting and military organisations; critical WBGT thresholds can be adjusted for clothing, activity level and acclimatisation status, and they guide decisions to modify, postpone or cancel activity [1]. More

sophisticated, sport-specific policies have been developed that evaluate the evaporative capacity of the environment relative to the evaporative heat-loss requirement of the participant, accounting for equipment, activity level and morphology, and that trigger graded interventions such as more frequent breaks and, ultimately, suspension of play [1]. Work-to-rest ratios, scheduling of activity during the coolest parts of the day, modification of uniforms and equipment to improve heat loss, and the provision of shade, fluid and cooling facilities are further organisational measures [1,24]. In youth football, disproportionate work-to-rest ratios during practice and the retention of full uniforms in high-WBGT conditions were identified as modifiable extrinsic risk factors, and the provision of adequate rest, gradual uniform integration and medical coverage were recommended [24]. The success of comprehensive organisational countermeasures is illustrated by the Tokyo 2020 Paralympic Games, at which shading, air-conditioned rooms, fluid and ice supply, early-morning scheduling of endurance events and the adoption of WBGT-based cancellation limits contributed, alongside individual strategies, to the absence of medically diagnosed heat illness despite extreme conditions [25]. Prevention, like treatment, therefore depends on the coordinated application of individual and organisational measures, and its incomplete adoption represents a further avenue for reducing the burden of EHS [1,24,25].

It is worth noting, finally, that the therapeutic use of cold and heat exposure extends beyond the emergency treatment of EHS into the broader domains of recovery, adaptation and health. Cold-water immersion and other cold-exposure modalities are widely used by athletes to accelerate recovery and reduce muscle soreness after exertion, and their physiological effects — vasomotor, cardiovascular and metabolic — overlap with the mechanisms exploited in emergency cooling [17,18]. Passive heat exposure such as sauna bathing, conversely, produces controlled hyperthermia and cardiovascular adaptation and has been studied for its health effects [18]. While these applications lie outside the acute management of EHS, they reinforce the central physiological insight of this review: that the deliberate manipulation of body temperature, understood through the physics of heat transfer, is a powerful tool that can be directed either to adaptation and recovery or, in the emergency, to the reversal of life-threatening hyperthermia [16–18].

12. Return to Activity, Heat Tolerance and Recurrence

The management of EHS does not end with successful cooling; the decision about when and whether the survivor may safely return to strenuous activity is a distinct clinical challenge, and one on which the evidence is less developed than for the acute phase. The concern is twofold: that a residual, possibly persistent, impairment of thermoregulation — heat intolerance — may render the individual vulnerable to a further episode, and that premature return before organ recovery is complete may itself be hazardous [19,27]. The available guidance emphasises confirmation of systemic recovery, including normalisation of core temperature, laboratory parameters and organ function, followed by a graduated reintroduction of exercise and heat exposure [8]. Structured serial assessment of biomarkers, electrocardiography and echocardiography provides a pragmatic basis for judging cardiac and metabolic recovery, particularly where advanced imaging is unavailable, and clearance for gradual return to exercise

is appropriate once these have normalised, as in the community case in which all markers returned to normal by one month [13].

The most developed objective tool for assessing residual heat vulnerability is the heat tolerance test, used chiefly in military settings as part of the return-to-duty process. In the Israel Defense Forces protocol, EHS patients undergo a standardised heat tolerance test six to eight weeks after the event, consisting of a two-hour treadmill walk in a climatic chamber at controlled temperature and humidity, during which heat intolerance is diagnosed if rectal temperature rises above 38.5 °C, heart rate exceeds 150 beats per minute, or either fails to plateau [39]. Individuals classified as heat intolerant are prohibited from intense activity until a repeat test, and the two case studies reported by Ketko and colleagues illustrate both the value and the complexity of the approach: one patient, initially heat intolerant, demonstrated a progressive recovery of thermoregulation and was classified as heat tolerant only eight months after collapse, whereas the other remained heat intolerant on repeated testing eleven months after the event, a distinction the authors interpreted as separating a transient, acquired impairment from a possibly congenital one [39]. These cases underscore that recovery of thermoregulatory function is variable and may take many months, that at least two tests, and sometimes a third, may be required before a definitive judgement, and that the heat tolerance test is an effective and sensitive screening tool for both temporary and permanent heat intolerance [39]. The recommendation that emerges is to perform a heat tolerance test after EHS and, in the case of a positive result, to repeat it periodically for at least a year before permitting unrestricted return to intense activity [39].

The recurrence data reinforce the importance of caution in this period. As noted earlier, analysis of a warm-weather road race demonstrated that survivors of EHS carried a significantly elevated risk of a second episode, concentrated in the first two years after the index event, with more than half of recurrent cases occurring within two years and the majority within four [27]. This temporal pattern aligns with the biochemical and physiological evidence of a prolonged recovery, and it argues for heightened surveillance, more conservative activity progression and closer attention to modifiable risk factors during this window [27]. It also intersects with the debate about whether recurrence reflects a persistent acquired impairment or an underlying, possibly genetic, susceptibility — a question that remains unresolved but that has practical significance, because an individual with a fixed predisposition may require permanent restriction whereas one with a transient impairment may recover fully [27,39]. The convergence of the heat-tolerance-test and recurrence literatures is that return-to-activity decisions after EHS should be individualised, evidence-informed and cautious, and should not rely on the resolution of acute symptoms alone [27,39].

An important gap, particularly relevant to athletic as opposed to military populations, is the absence of a validated, sport-specific protocol for neurological clearance after EHS. Current guidelines emphasise systemic recovery but rarely incorporate targeted neurological assessment, even though subtle neurocognitive or cerebellar deficits may persist after systemic parameters have normalised and may compromise both performance and safety on return to competition [8]. Drawing on the analogy with the graduated return-to-play protocols developed for sport-related concussion, a staged neurological return-to-sport pathway after EHS has been

proposed, progressing from rest and symptom resolution through light aerobic exercise, sport-specific training in thermoneutral conditions, controlled heat exposure and finally full medical and neuropsychological clearance [8]. Until such protocols are validated, the prudent approach is to extend the caution applied to systemic recovery to the neurological domain, and to base clearance on comprehensive evaluation rather than on the normalisation of temperature and laboratory values alone [8].

13. Long-Term Sequelae and Follow-Up

Although EHS is potentially fully reversible when treated promptly, a substantial minority of survivors experience long-term sequelae, and the recognition that the disease does not always end with hospital discharge has important implications for follow-up care. The physical sequelae reflect the multi-organ nature of the acute injury: survivors may develop heat intolerance, chronic kidney disease, cardiovascular disease and persistent muscular symptoms, and the long-term data, though limited, indicate that these are not negligible [3,19]. In the combined survey and record review of EHS and exertional rhabdomyolysis survivors, a large proportion reported persistent symptoms at six and twelve months, including a subjective feeling of being overheated more easily, muscular symptoms at rest and during exercise, and neurological complaints such as slower thought processes and impaired memory and balance [19]. The renal dimension is underlined by evidence linking prior heat injury to an elevated long-term risk of chronic kidney disease, and by the recognition that acute kidney injury sustained during exertion may not always be transient [3,20].

The neurological sequelae warrant particular attention because they are the most disabling and because they connect directly to the pathophysiology of selective CNS vulnerability described earlier. A dedicated narrative review of long-term neurological outcomes after EHS in athletes found that, while many acute manifestations improve with rapid cooling, a significant minority of survivors develop persistent deficits, of which cerebellar dysfunction — ataxia, dysarthria and nystagmus — is the most common and often the most disabling, reflecting the selective degeneration of cerebellar Purkinje cells [8]. Permanent neurological deficits have been documented in a meaningful proportion of survivors with known outcomes, and magnetic resonance imaging may demonstrate cerebellar atrophy weeks to months after the event, even when early imaging appeared normal [8]. Crucially, the speed of cooling is identified as the single most important modifiable factor influencing neurological prognosis: rapid cooling, particularly by cold-water immersion, is associated with a lower incidence of permanent deficits, and even short delays can result in irreversible Purkinje-cell damage [8]. This finding closes the conceptual loop of the present review, demonstrating that the same rapid cooling that determines survival also determines the preservation of long-term brain function, and that recognition and cooling therefore protect not only life but neurological integrity [8].

The mental-health and quality-of-life dimensions of recovery have been comparatively neglected, yet the evidence suggests they are significant. Validated questionnaires administered to EHS and exertional-rhabdomyolysis survivors indicated problematic fatigue in nearly a third and mood or anxiety disorder in a smaller but meaningful proportion, and residual symptoms such as fatigue, heat intolerance and mood disturbance have been reported months to years after the incident [8,19]. Beyond the individual sequelae, a striking finding of the survivor cohort

was the perceived inadequacy of follow-up care: the great majority of participants reported that their follow-up had been insufficient and that more frequent and intensive follow-up, particularly guidance on returning to activity, would have benefited their recovery [19]. This deficiency in follow-up mirrors, at the chronic end of the disease course, the same evidence–practice gap seen in the acute prehospital phase, and it points to a systematic under-provision of structured post-event care [19].

The implication for practice is that follow-up after EHS should be proactive, prolonged and multidimensional, extending beyond the initial recovery period to encompass surveillance for renal, cardiovascular, neurological and psychological sequelae, standardised assessment of heat tolerance and, where relevant, neurocognitive function, and clear, individualised guidance on return to activity [8,19]. The comprehensive one-month biomarker and cardiac follow-up reported in the Vietnamese community case, which confirmed complete recovery, exemplifies the value of structured longitudinal monitoring, even in resource-limited settings, and demonstrates that such follow-up is feasible with modest means [13]. The recurring conclusion across the sequelae literature is that EHS should be conceived not as a discrete acute event but as a condition with a potential long tail, and that the quality of recognition and cooling in the acute phase, together with the quality of follow-up thereafter, jointly determine the survivor's long-term outcome [8,13,19].

14. Discussion

The body of evidence synthesised in this review supports a coherent and, in its essentials, unambiguous conclusion: the outcome of exertional heat stroke is determined chiefly by two modifiable factors — the promptness of recognition and the adequacy of cooling — and the science underlying both is stronger and more quantitatively precise than its inconsistent implementation would suggest. Several threads deserve to be drawn together and critically appraised.

The first is the striking coherence between the pathophysiology of EHS and the empirical evidence on cooling. The modern understanding of the condition as a time-dependent, systemic inflammatory and multi-organ syndrome, in which the duration rather than the peak of hyperthermia governs injury, provides the mechanistic rationale for the empirical finding that cooling rate is the decisive therapeutic variable [4,5]. The convergence of independent analyses on the 0.15 °C per minute threshold — below which mortality and complications rise sharply and above which they approach zero — gives clinicians an unusually clear, quantitatively defined target, and it transforms the choice of cooling modality from a matter of preference into a matter of measurable efficacy [4,11]. This is a rare instance in emergency medicine of a treatment target that is both mechanistically justified and empirically validated, and it should anchor all protocols. The corollary is equally clear: modalities that cannot achieve this rate — ice packs to the great vessels, cold intravenous fluids, cooling blankets, single-fan evaporative cooling and chemically activated cooling vests — are not adequate primary treatments, however convenient, and their use as such represents a departure from the evidence [4,33].

The second thread is the comparative ranking of cooling modalities and the practical adaptation of cooling to setting. The evidence establishes a clear hierarchy in which cold-water immersion

of a large body-surface area is the gold standard, with multi-segment immersion, tarp-assisted cooling and ice-sheet or ice-towel methods forming a tier of acceptable field alternatives, and the small-surface-area and air-dependent methods forming an inadequate tier suitable only as adjuncts or bridges [4,28,32,36]. The firefighter-cooling literature is particularly instructive because it dissects the physical determinants of cooling rate — surface area, immersion depth, water temperature and circulation — and demonstrates why hand-and-forearm immersion, despite its physiological plausibility and institutional endorsement, is inadequate, whereas multi-segment immersion is effective [36]. This example is a cautionary tale about the gap between plausible reasoning and measured efficacy, and it reinforces the principle that cooling recommendations must be grounded in cooling-rate data rather than in mechanistic appeal. At the same time, the occupational and community literatures make clear that the "best" modality in a given situation is the most effective one that can actually be applied without delay, so that a portable ice-towel kit applied immediately may serve a collapsed worker better than a superior method that cannot be assembled in time [28]. The reconciliation of these two principles — insist on adequate cooling rate, but adapt to the constraints of the setting — is the practical heart of cooling protocol design.

The third thread is the recurring and troubling gap between evidence and practice, which appears at every stage of the pathway and in every setting. On the recognition side, the reliance on invalid peripheral thermometry and the poor knowledge of best practices among EMS providers undermine the very first step of management, while on the treatment side a majority of prehospital protocols fail to endorse, or actively discourage, on-site cold-water immersion [10,12]. This gap is not confined to under-resourced systems; it has been documented in high-income countries with developed health services, where prehospital management of EHS survivors was inconsistent and frequently non-adherent to guidelines [19]. The implication is sobering but also encouraging: because the science is settled, the principal remaining cause of preventable death and disability in EHS is a failure of implementation, and implementation is, in principle, correctable through updated protocols, provision of appropriate equipment, education and inter-professional collaboration [10,12]. The frontier of progress in EHS care lies less in the discovery of new treatments than in the reliable delivery of what is already known [10,12,34].

The fourth thread concerns the individualisation of risk and response. A consistent finding across the physiological literature is the large inter-individual variation in the thermoregulatory response to a given heat and exercise load, such that the same conditions that are safe for most may be dangerous for a susceptible minority [23]. This heterogeneity has two implications. For prevention, it argues against a purely threshold-based, one-size-fits-all approach and in favour of individualised assessment of heat tolerance and tailored mitigation strategies [23]. For recognition, it means that vigilance cannot be relaxed simply because environmental indices appear tolerable, and that the index of suspicion must remain high whenever a person collapses with neurological dysfunction after exertion in the heat, regardless of the measured conditions [1,23]. The behavioural dimension compounds this: the very motivation that produces athletic and military excellence can override the protective sensory feedback from hyperthermia, and diminished interoceptive awareness may be an intrinsic vulnerability, so that the archetypal EHS victim is the highly motivated individual pushing beyond safe limits [26]. Recognition, in

this sense, begins before collapse, in the anticipation of who is at risk and in what circumstances.

The fifth thread is the extension of the recognition-and-cooling imperative beyond survival to the preservation of long-term function. The demonstration that the speed of cooling is the single most important modifiable determinant not only of survival but of neurological prognosis — because delays permit irreversible cerebellar Purkinje-cell injury — elevates the stakes of prompt cooling still further [8]. It reframes rapid cooling not merely as a life-saving intervention but as a brain-protective one, and it strengthens the case for treating EHS as a time-critical neurological emergency. The long-term sequelae literature, together with the documented inadequacy of follow-up, further indicates that the quality of acute recognition and cooling and the quality of subsequent surveillance jointly shape the survivor's trajectory, and that both ends of the disease course require attention [8,13,19].

Two broader conceptual points frame these threads. The first is that EHS sits at the intersection of physiology, behaviour, environment and organisation, and that its effective management requires integration across these domains; the physiology of thermoregulation, understood as an integrative system linking neural, endocrine, immune and metabolic responses, explains both the systemic reach of the injury and the power of temperature manipulation to reverse it, while the behavioural and organisational context determines whether that physiological knowledge is applied in time [13,40]. The second is that the same physical principle — the transfer of heat across a gradient — underlies both the failure that produces EHS and the treatment that reverses it, so that a clear grasp of heat-transfer physics is the common foundation of prevention, recognition and cooling [14,15]. Understanding EHS as a disorder of heat balance, recognising it through the combination of context, neurological dysfunction and valid core temperature, and treating it by the rapid application of the most effective available cooling, together constitute a coherent and evidence-based approach whose principal challenge is not knowledge but consistent execution.

This review has limitations that qualify its conclusions. As a narrative rather than a systematic review, it did not apply a formal quality-assessment instrument or quantitative pooling, and the selection and weighting of evidence, while guided by the strength of the underlying data, necessarily involved judgement. Much of the highest-quality evidence on cooling derives from case reports and case series rather than randomised trials — an unavoidable consequence of the ethical impossibility of inducing EHS in humans — so that the cooling-rate thresholds, though remarkably consistent, rest on observational data [4]. The evidence base is also geographically skewed toward high-resource settings, limiting its generalisability to the low- and middle-income community events where the burden is growing and preparedness is weakest [13]. Finally, several promising avenues — wearable and predictive technologies, biomarker-guided triage, neurologically informed return-to-sport protocols — remain at an early stage and require validation before they can be recommended for routine use [8,29]. These limitations notwithstanding, the central conclusions of the review — the primacy of recognition and adequate cooling, and the urgency of closing the evidence–practice gap — rest on a broad and internally consistent body of evidence.

15. Conclusions

Exertional heat stroke is a life-threatening but largely survivable emergency whose outcome is decided, more than by any other factor, by how quickly it is recognised and how effectively it is cooled. On the basis of the evidence reviewed, several principal conclusions may be offered.

First, recognition rests on the combination of central nervous system dysfunction and elevated core temperature in the context of exertion in the heat, and it must not be defeated by the limitations of temperature measurement. Rectal thermometry is the reference standard; peripheral methods are unreliable and may falsely exclude the diagnosis; and a core temperature below 40 °C, whether because of rapid post-collapse decline or prior cooling, does not rule out EHS. Where a valid temperature cannot be obtained, collapse with neurological dysfunction after exertion in the heat should be treated as EHS until proven otherwise.

Second, cooling is the definitive treatment, and its adequacy is quantifiable. A cooling rate above 0.15 °C per minute is consistently associated with survival without complications, and cold-water immersion of a large body-surface area is the gold standard, reliably achieving this target. Alternative field methods — multi-segment immersion, tarp-assisted cooling, ice sheets and ice towels — are acceptable when immersion is impractical, whereas ice packs, cold intravenous fluids, cooling blankets, single-fan evaporative cooling and cooling vests are inadequate as primary treatment. The governing operational principle is "cool first, transport second," with cooling continued until core temperature reaches approximately 38.9 °C.

Third, the best cooling modality in any given situation is the most effective one that can be applied without delay, and cooling must therefore be adapted to the setting — from immediate immersion in supervised sport and the military to portable, locally available methods in occupational and community settings, including the low- and middle-income community events where preparedness is weakest.

Fourth, the principal remaining cause of preventable death and disability in EHS is not a deficit of scientific knowledge but a failure of implementation. Reliance on invalid thermometry, poor provider knowledge, inadequate equipment and prehospital protocols that fail to endorse or actively discourage on-site cooling all undermine the delivery of proven care. Closing this evidence–practice gap through updated protocols, appropriate equipment, education and collaboration between the sports-medicine and emergency-medicine communities is the most immediate opportunity to reduce the burden of EHS.

Fifth, the imperative of prompt recognition and cooling extends beyond survival to the preservation of long-term function, because the speed of cooling is the single most important modifiable determinant of neurological outcome. Prevention through heat acclimatisation, individualised hydration, pre- and per-cooling and organisational heat policies, together with cautious, individualised return-to-activity decisions informed by heat-tolerance testing and by the elevated risk of recurrence in the first two years, and proactive long-term follow-up for renal, cardiovascular, neurological and psychological sequelae, complete a comprehensive approach in which recognition and cooling remain the decisive, and eminently modifiable, determinants of outcome.

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