Heat-Related Illness

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4.1 Introductory Comments

History vividly records the inaugural women's Olympic marathon in Los Angeles 1984, an event celebrated for the victory of the hometown favourite Joan Benoit. But for many, the enduring memory of that hot July day will be of Swiss athlete, Gabriela Andersen-Schiess, dragging her pathetic contorted frame around the final 400-metre lap of the Los Angeles Coliseum [1]. Hyperthermic, dehydrated and hemi-paretic, her extreme distress drew disgust and dismay from helpless onlookers. And in the cauldron of the Coliseum, this hapless, ataxic athlete crossed the finish line to collapse into the arms of waiting medics, a vision indelibly etched in the memories of spectators and television viewers alike. Fast-forward 13 years to 1997 when, at the World Ironman Championships, the punishing Hawaiian heat took its toll on two accomplished triathletes, Sian Welch and Wendy Ingraham. Again, TV viewers joined onlookers, to witness the bizarre end of an epic event. For the final 200 m, two incapacitated, heat-stressed athletes, incapable of remaining erect were humiliated into an infantile crawl across the finish line to be gobbled up by concerned medical staff [2]. And at the 2016 International Triathlon final event in the heat of Cozumel, Mexico, a further arresting incident took place, this time the collapse of Olympian Jonny Brownlee with the World title and the finish literally in his sight. Neurologically impaired to a point of obvious disorientation, Brownlee was presumably hyperthermic, as well as energy and electrolyte depleted. His limp frame was dragged bodily across the line by fellow competitor Alistair, coincidentally his older sibling and the reigning Olympic champion [2]. Although his recovery was uncomplicated he was clearly affected by the combination of prolonged physical exertion and environmental stressors.

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The common thread connecting these examples is the phenomenon of exercise-associated collapse. However, in each case the high ambient temperature and the obvious signs of neurological impairment highlight the potential for far more sinister clinical outcomes. Those responsible for the provision of on-site medical care to endurance athletes must remain vigilant to the possibility of exertional heat stress and its consequential challenge to thermoregulatory mechanisms. Further, such well-supported, elite events represent only a fraction of the cases of exertional collapse in endurance athletes including many unheralded, unreported, mass participation events involving a cohort of social, club-level athletes, the so-called "weekend warriors". Collectively however, these instances underscore morbidity in contemporary endurance sport that cannot be ignored, placing heat-related illness in alarming perspective. Throughout this chapter, the terms heat "illness" and "injury" are interchangeable, denoting the consequences of prolonged exertional heat stress.

4.2 Exertional Heat Stress: What Have We Learned?

The past three decades have witnessed an explosion of interest in endurance sport, precipitating advanced coaching and a parallel scientific focus on the stresses of prolonged physical exertion [3–9]. Yet the cases referred to previously beg the fundamental question of heat injury prevention, an approach that appears to challenge the prevailing default stance of active resuscitation. Given the timing, duration and energy requirements of contemporary endurance events, heat-related injury with attendant serious health implications will always be a major concern. Traditionally, events such as triathlons or multi-sport variations are held in spring and summer, exceed one-hour duration and engage athletes in a combination of land-based and aquatic disciplines. They invariably challenge the regulation of body temperature in circumstances where too frequently, the sum of energy production and environmental stressors exceeds the body's innate capacity to dissipate heat. Several international sporting federations including Triathlon (ITU), Swimming (FINA), volleyball (FIVB) and Athletics (IAAF) have made significant research investments leading to a greater understanding of the pathophysiology of exertional heat illness [10–12]. In turn, this has spawned an extensive body of literature on thermal regulation in athletes, enhancing knowledge of its genesis and systemic implications [6–8, 13–16]. Informed by this research, sports physicians are becoming more adept at recognising signs of impending heat illness, and coaches are implementing strategies to adapt athletes to the impact of heat stress while event organisers seek scientific and clinical input into planning major races. FINA has applied research-informed evidence to regulate temperature limits for the 10 km marathon swim, popularised since its addition to the Olympic programme in Beijing 2008. Similarly, water temperature guidelines in triathlon competition with implications for wetsuit use have also been adopted by the ITU and FINA [10, 11]. Both the ITU and the IAAF have introduced temperature restrictions on land-based endurance events, governed by wet globe bulb temperature (WGBT) monitoring. The WGBT device measures ambient temperature, relative humidity, wind, and solar radiation to determine safe

conditions for prolonged exercise [12]. The FIVB medical rules have been influenced by recent research into the prevailing centre court temperatures at World Tour and World Beach Volleyball Championships (2009–2011) where WGBT recordings were matched against instances of medical forfeiture by athletes. These data have informed subsequent guidelines for minimising heat stress injury in the professional beach volleyball community [14]. In preparation for the 2020 Olympics and Paralympic Games in Tokyo, organisers are already expressing concern over the expected temperatures that will prevail during the period of the Games [17]. WGBT indices are being applied to the schedule of Games training and competition programme in an effort to minimise the potential for heat stress injuries. There has also been a call by the American College of Sports Medicine (ACSM) for the Games Organising Committee to limit competition times to early morning or evening in an attempt to minimise the effects of solar radiation on potentially affected outdoor disciplines [18]. While proactivity by responsible governing bodies is acknowledged, the literature and public media reports would suggest that some experienced, well-trained endurance athletes still remain susceptible to severe heat-related injury.

Research has shown that for a number of reasons including individual variability, acclimatisation, training and nutrition, not every athlete with a rectal temperature of 40 °C will advance to catastrophic end-stage exertional heat stroke with multi-organ failure [3, 6–9]. Athletes including marathoners, soccer players, open water swimmers and American football players have been reported with sustained rectal temperatures above a 40 °C threshold without any serious medical consequence [7]. In a similar vein, the interplay between the concepts of hyperthermia, exhaustion and dehydration is important to recognise. While the concept of exhaustion as the inability to sustain physical output is a well-reasoned physiological response, the time to complete exhaustion is also shown to diminish with rising ambient temperatures above 20 °C [6, 7, 16].

This chapter addresses the basic understanding of thermal regulation, considers the spectrum of heat-related injuries and suggests strategies to prevent heat illness, informed by recent research and clinical experience.

4.3 Thermoregulation in Humans

Thermal regulation is the ability of the body to maintain a core temperature of 37 °C to within a relatively narrow range (33.2–38.2 °C), a fundamental homeostatic mechanism acknowledged by observational studies and reliant upon several physiological factors [4, 5, 7]. Normal core body temperature rises rapidly in response to thermal challenge and in the context of endurance sport this is the product of the energy generated from sustained physical activity plus recognised environmental influences. These factors, including ambient temperature, humidity and solar radiation are beyond the control of the athlete and remain the responsibility of event organisers. As already referenced, these external influences have been recognised by the medical advisory committees of the ITU, FINA, FIVB and the IAAF [10, 11, 14]. Their interest in preserving the safety of athletes is embedded in sport-specific rules and event regulations. In addition, the International Olympic Committee has

published a position statement acknowledging a number of environmental influences including temperature, that challenge the contemporary elite athlete in a variety of sporting disciplines [15].

As mammals we possess an innate propensity for homeostasis, with four recognised pathways for the transfer of body heat. These are via the primary mechanisms of convection, conduction, radiation and evaporation. Collectively, these are essential to the regulation of core body temperature and critical to homeostasis, with fluctuations beyond a couple of degrees posing a significant challenge to thermoregulation [19]. Heat loss mechanisms, triggered in the anterior hypothalamus, reflect the temperature differential between skin surface and the environment. As body temperature increases, active sympathetic cutaneous vasodilation increases blood flow in the skin and this initiates sweating through eccrine gland activity. If skin temperature exceeds that of the surroundings, heat loss occurs through radiation and conduction. But, if the ambient temperature exceeds that of the skin, the body gains heat that is dissipated through the evaporation of sweat. Consequently, physical activity in high ambient temperatures depends upon adequate evaporation and where this is impeded, the consequential rise in core body temperature may precipitate severe heat injury [6, 7, 9]. A well-hydrated athlete will have the capacity for adequate sweat production. And adequate fluid intake is quite obviously an influence over which the athlete has control, enhanced by the provision of sufficient food and drink stations around any given course. Ambient factors such as temperature and relative humidity remain an issue for race organisers charged with venue selection and seasonal timing. As already described, when the surrounding temperature is higher than that of the skin, any impedance to evaporation, the primary means of heat dissipation, will cause the internal body temperature to rise. Prolonged physical exertion in excessive humidity impedes sweat evaporation, a mechanism essential for the maintenance of thermal neutrality [3, 6, 19].

In summary therefore, under normal circumstances, body heat generated from physical activity is quickly dissipated by the adaptive "cooling" mechanisms of radiation and convective loss through the skin, evaporation of sweat and via small amounts of evaporation through respiratory loss. These mechanisms diminish the potential hazards of exertional heat injuries, the most serious of which is heat stroke, commonly associated with a body core temperature exceeding 40 °C. While some authorities quibble over the nomenclature, there is an agreed "spectrum" of exertional heat injuries reported in the sports medicine literature, notably in events of high intensity or long duration [20]. Clinicians agree that heat illness, unrecognised and untreated, may present quite innocently with initial signs of oedema, heat rash, cramps and syncope, and progress to heat exhaustion or heat stroke representing the most severe, life-threatening end of this continuum [6, 8, 9, 14–16].

4.4 Pathophysiological Factors in Heat Injury

Our initial understanding of heat injury and its serious clinical sequelae has its genesis in the general population during times of excessively high ambient temperatures. Heat stroke with attendant life-threatening consequences is considered a

potential outcome from exposure to a high environmental temperature or prolonged strenuous exercise. A universally accepted definition includes a core body temperature above 40 °C, the absence of sweating and accompanying signs of central nervous system dysfunction [21]. These signs may include confusion, disorientation, ataxia, delirium, convulsions or coma. From a pathophysiological perspective, it is also generally agreed that unremitting heat stroke is essentially a form of hyperthermia that progresses to multi-organ failure as the result of a systemic inflammatory response. Cases of non-exertional heat stroke, involving individuals with existing comorbidities such as aging, obesity, diabetes, renal impairment and heart disease, demand acute medical intervention and have been associated with high rates of morbidity and mortality, widely reported in North American and European data [22, 23]. However, the extrapolation of the pathophysiology of non-exertional heat stroke has informed the contemporary understanding of exertional heat stress in sport, with sustained strenuous physical activity the obvious additional stressor to thermoregulation [21].

As already discussed, there is general acceptance of a spectrum of heat-related injury. At points along this continuum an athlete may display signs of poor adaptation to the escalating consequences of increasing body temperature. However, where there is little or no challenge to the homeostatic mechanism of thermoregulation, the adapted athlete will simply experience "normal" energy depletion and ultimate fatigue [6]. This may or may not result in what is commonly seen as exertional collapse, a relatively benign condition that responds to conservative management. However, sustained, increasing body temperature will stimulate cutaneous vasodilatation, thereby diverting blood from central viscera to the skin where sweating is initiated as a primary means of heat dissipation. This shunt of blood from the central circulation to the periphery challenges major organs and precipitates a relative reduction in intravascular volume that may lead to hypotension, associated with the phenomenon of "heat syncope". Meanwhile, sweating-induced losses exacerbate dehydration and sodium depletion, linked with heat exhaustion and generally considered as contributory to heat-related muscle cramping. Any further loss of salt and water further impairs normal thermoregulatory mechanisms and together with the reduction in visceral perfusion may precipitate cellular damage and ultimate organ failure if the challenge to core body temperature continues unchecked [7, 17, 19, 21].

4.5 Pathogenesis of Severe Heat Injury

The underlying, complex cascade of events in exertional heat stroke (EHS) commences at a cellular level where the membranes of heat-stressed cells become damaged, their energy systems disrupted and cell membrane permeability becomes altered. These changes precipitate the leakage of intracellular endotoxins into the systemic circulation that ultimately give rise to a well-documented syndrome of multi-organ damage and failure [24, 25].

A process labelled the "systemic inflammatory response syndrome" is also described, as the consequence of endotoxin leakage through the intestinal mucosa

accompanied by interleukins (IL)-1 and (IL)-6 from skeletal muscle. These proteins enter the systemic circulation to activate leukocyte production causing diffuse inflammatory effects that damage vascular endothelium with a consequential risk for microthrombotic events [25]. Platelet production is suppressed by heat, there is enhanced fibrin formation and ultimately a diffuse coagulopathy manifests as disseminated intravascular coagulation (DIC). Further complicating gut cell membrane damage is the leakage of intestinal gram-negative bacterial fragments into the systemic circulation that are responsible for the ominous consequence of endotoxic shock. These effects are enhanced in the presence of dehydration with overwhelming clinical consequences for any vulnerable athlete [26–28].

In addition to these sequelae, it is also well established that the integrity of skeletal muscle is seriously challenged by temperatures exceeding 40 °C [29]. Sustained heat insult gives rise to structural changes at a cellular level resulting in diffuse muscle fibre destruction. This pathophysiological process causes a clinical condition known as rhabdomyolysis [28, 29]. Muscle enzymes, in particular creatine kinase (CK), leak into the circulation from damaged cells, with CK levels more than a thousand times normal deemed to be an accurate marker of rhabdomyolysis. Free myoglobin fragments, another product of muscle damage, give rise to dark-stained urine, the typical discolouration of frank myoglobinuria. Exercise-induced rhabdomyolysis may advance to produce severe renal insult through tubular toxicity and obstruction. Intracellular potassium, also released into the systemic circulation from damaged cells gives rise to hyperkalemia, a recognised precipitant of fatal cardiac arrhythmia [28]. Collectively, these potential life-threatening consequences underscore the seriousness of prolonged physical exertion in the heat [30].

4.6 Exertional Heat Illness: A Continuum of Disorder

There is an acknowledged potential for heat illness in any situation involving sustained, intense physical activity, particularly in hot-humid conditions. Exertional heat illness, affecting athletes during high-intensity or long-duration exercise may result in withdrawal from an event or collapse during or soon after its conclusion. Although a wide variation in individual athlete response to heat stress is acknowledged [6], these illnesses include exercise-associated muscle cramping, heat exhaustion or exertional heatstroke. Athletes who are dehydrated, not well acclimatised, who use certain medications, or have recently been unwell, are at greater risk and more vulnerable to thermal challenge. A constellation of signs and symptoms associated with exertional heat illness exist along a recognised continuum of disorder. Early recognition and rapid cooling can reduce the morbidity and mortality associated with these clinical challenges that in many cases are subtle and easy to miss. Therefore, coaches, medical personnel, and race officials must be vigilant and monitor at-risk athletes closely. Heat exhaustion and exercise-related muscle cramps are not typically linked with excessive hyperthermia, but result from fatigue, dehydration and/or electrolyte depletion. But where central mechanisms of heat regulation fail and normal heat dissipation

processes fail, unidentified athletes may succumb to serious outcomes. The following is a brief summary of common exertional heat injuries.

1. Heat cramps:

   Painful involuntary episodes of muscle spam associated with prolonged exercise in hot conditions have given rise to the term "heat cramps". There is also a significant body of research interest in the same condition for which the term Exercise-Associated Muscle Cramps (EAMC) has been coined. This form of cramping in athletes occurs in the muscles of the calves, but may also affect the arms and abdomen. There is scientific debate between two possible aetiologies for EAMC. The first implicates prolonged sweating with attendant electrolyte loss, particularly of sodium, as a precipitant of heat cramps, while the alternative hypothesis is that of "altered neuromuscular control". Contemporary debate would favour the latter as the most valid pathophysiological mechanism [31]. Combined eccentric-concentric, repetitive muscle contraction through a short range of motion during the foot plant and "toe-off" phases of running would suggest the propensity for common cramping of the gastrocnemii (calf muscles). Similarly, a prolonged erect running posture engaging static contraction of the anterior abdominal wall, with rigid, adducted arms and flexed elbows provides additional logic for cramps of these regions. It is also proposed that the spontaneous discharge of hyper-excitable axon terminals results in muscle fasciculation that may progress to debilitating muscle cramps [32]. In light of the informed debate over the pathophysiological basis of heat cramps it remains generally agreed that rest, passive stretching and oral fluid replacement to replenish sodium losses are accepted measures in the management of heat cramps. Maintaining regular fluid intake during the event is still sound advice to all endurance athletes. Dehydration in itself represents an issue for all athletes but particularly those who are sweating profusely during events in hot climates. Setting aside the electrolytes lost in sweat, once fluid depleted, an athlete will no longer have the capacity to initiate sweating and thereby fails to engage a key thermoregulatory mechanism. While heat cramps represent the mild end of the spectrum of heat injuries, this phenomenon, unchecked, may be the precursor to more sinister injury associated with prolonged physical activity.
2. Heat exhaustion:

   Heat exhaustion from prolonged physical exertion in high temperatures frequently co-exists with high humidity. Athletes suffering from full-blown heat exhaustion invariably look distressed but as with all forms of heat injury, signs and symptoms appear on a spectrum from mild to moderate to severe. The early signs of impending heat exhaustion may be those of severe heat cramping associated with profuse sweating and tachycardia [3]. An unidentified athlete may progress to becoming confused and disorientated, representing early ominous central nervous system consequences of raising body core temperature. Uncharacteristic behaviours, worsening confusion, unsteady gait and inappropriate response to instructions may also be subtle signs of progressive heat stress implicating the brain [6]. Heat exhaustion is frequently accompanied by symp-

toms of dizziness, nausea, and fatigue and signs such as vomiting and mild temperature elevation. Athletes may have complained earlier of muscle cramping and on examination are found to have a rapid thready pulse and cold clammy skin in the presence of excessive sweating and dizziness. There is often an associated feeling of fatigue and impending collapse suggesting that these athletes deserve close monitoring by race officials. Unheeded, and untreated, the gain in body heat may completely overwhelm thermoregulatory mechanisms and progress to severe heat stroke. That said however, heat exhaustion, detected early, generally resolves with symptomatic care and oral hydration [3, 6, 8, 11].

3. Exertional heat stroke:

As already emphasised, heat exhaustion and heat stroke may occur along a recognised continuum of heat-related injury, but by definition, heat stroke always implies a core body temperature above 40 °C. Exertional heat stroke (EHS) is the most severe form of heat injury, whereby sustained, excessive body heat results in a syndrome of central nervous system dysfunction and a systemic inflammatory response [21, 33]. The mechanisms by which an athlete with exertional hyperthermia proceeds to a state of collapse is characterised by a cascade of disturbances of the nervous, cardiovascular, haematological and renal systems. It represents a life-threatening injury resulting from the progression of non-specific symptoms including headache, nausea and general malaise to severe systemic effects typified by the absence of sweating (anhidrosis), hepatic failure, disseminated intravascular coagulation and rhabdomyolysis. The progressive neurological signs of confusion, delirium and coma are frequently associated with arrhythmia and a potentially fatal outcome [3, 6, 8]. The effective management of heat stroke demands prompt recognition, immediate withdrawal from the event, protection from the prevailing elements, aggressive whole body cooling and appropriate fluid and electrolyte repletion [6, 7].

4.7 Susceptibility to Exertional Heat Injury

A significant body of experience derived from military reports and associated clinical data has informed the understanding of EHI and individual susceptibility. These reports are rich in details of clinical presentation, and corroborate the pathophysiological basis for heat injury. Factors relating to the individual susceptibility to heat injury may be classified as either extrinsic or intrinsic. The former includes environmental factors such as ambient temperature, humidity and solar radiation [34–37]. And in multisport events this extends to water temperature. As already alluded to, the control of these factors resides with international federations and race organisers who bear the responsibility for venue selection and the seasonal timing of major events [32, 38].

However, it is also understood that individual athletes preparing for endurance events should be obviously well trained, habituated (acclimatised) to exercising in heat, pre-hydrated, free from the effects of recent systemic illnesses and not taking medications known to increase susceptibility to heat stress. Drugs predisposing to

heat stroke include stimulants, antihistamines, anticholinergics and phenothiazines. The use of sunblock and the choice of appropriate clothing (texture and colour) are additional intrinsic factors that remain the responsibility of athlete. The education of coaches and athletes in areas of hydration, acclimatisation and freedom from intercurrent illness are important measures to minimise the risk of exertional heat stress. Heat illness and debilitating consequences are most commonly recognised by race officials whose responsibility it is to alert appropriate medical support. This acknowledges pre-race planning to include the provision of adequate medical staff with tents, awnings or suitable shelters for affected athletes.

4.8 Concluding Comments

Illnesses associated with sustained physical activity in hot humid conditions are widely reported. These have become a contemporary challenge for all endurance athletes. Well-adapted, pre-hydrated athletes are less likely to succumb to heat stress, however there are documented instances of heat-related injury at the highest competitive levels. When the normal mechanisms of heat regulation cannot cope with the imbalance of heat production and heat dissipation, there are recognisable clinical signs of distress that must be acknowledged. It behoves all organisers of endurance events to take into consideration prevailing environmental conditions, using instruments such as WGBT indices. They must also make adequate provision for the medical care of athletes and through vigilance, intervene appropriately where clinical signs indicate.

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