

Exertional heat stroke: life-saving recognition and onsite treatment in athletic settings

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ABSTRACT

The prognosis for exertional heat stroke depends upon the product of length of time the core temperature is elevated and the degree of elevation. The athlete with EHS who is discovered promptly and efficiently cooled will most likely survive the episode with little if any residual. In contrast, the athlete who has delayed presentation for treatment, especially if the area under to cooling curve is > 60 degree-minutes (centigrade), will have a complicated and often fatal course. Conductive cooling methods with ice or cool water immersion or rotating ice water cooled towels provide rapid and consistent whole body temperature reduction that is both organ and life saving. Recognition depends on a high index of suspicion on the part of athletes, coaches, and onsite medical personnel. In high risk conditions, athletes should “buddy up” to watch for subtle changes that can signal the onset of EHS.

Exertional heat stroke (EHS) is a risk for athletes who train or compete in hot humid conditions, especially when the heat and humidity are unexpectedly high or above the athlete's usual training and living conditions⁽¹⁻³⁾. Exertional heat stroke occurs in runners competing in 5 K through the 42 K distances, in football, both American rules and soccer, and in most other high intensity or longer duration athletic activities⁽⁴⁻⁸⁾. There have been exertional heat stroke deaths in all these activities, especially when the condition is not promptly recognized and treated. The paradox of heat stroke is that in hazardous heat conditions not all athletes seem to be the same risk and when heat stroke occurs in an athlete there are usually many others at the same site who are not affected. In contrast, there are athletes who develop exertional heat stroke in conditions that should be ideal for prolonged, high intensity exercise⁽⁹⁾.

Exertional heat stroke is defined by a rectal temperature above 40°C associated with abnormal organ system function induced by tissue hyperthermia⁽¹⁾. In athletes this is most readily seen as central nervous system (CNS) dysfunction that manifests with confusion, loss of neuromuscular control, or frank collapse^(1,10,11). Some athletes demonstrate very subtle CNS changes in personality or coordination that are difficult to detect by those who are not familiar with the athlete, which leads to missed or delayed recognition. When the core body temperature reaches critical levels, body organ systems begin to fail. The multisystem organ failure associated with exertional heat stroke is reversible as long as the temperature elevation duration is “short.” It appears from the review of successfully treated and fatal EHS cases that athletes with rectal temperatures above 41°C who are identified early and cooled rapidly, recover with little if any residual, whereas athletes who are “missed,” especially if the rectal temperature is above 42°C, and

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cooled later usually die⁽¹¹⁻¹³⁾. The critical element is not the peak of the rectal temperature, but rather the area under the cooling curve above 40°C in degree minutes (figure 1). Athletes seem to tolerate up to 60 degree (C)-minutes without difficulty, but fall victim to the cascade of fatal system failures somewhere over 60 degree (C)-minutes. Acute heat induced heart and renal failure are reversible with early cooling. Once the clotting systems begin to fail, the chances of survival seem to plummet. These are not “proven” rules, but seem to be valid when outcome data are retrospectively reviewed.

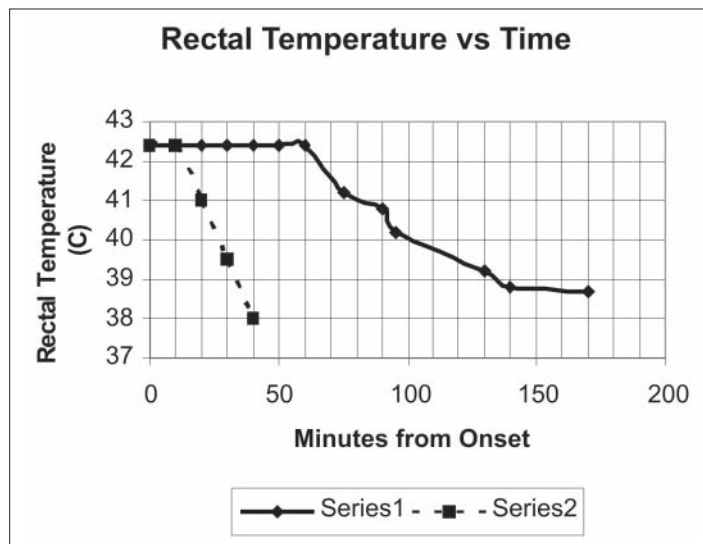


Fig. 1 – Composite EHS treatment cooling curves for runners identified at the finish line who all lived (Series 2) and football players who were not immediately identified who all died (Series 1). The area under the cooling curve for series 1 is approximately 50 degree-minutes and for series 2 is approximately 200 degree-minutes. Series 1 athletes were cooled with fans and water sponging in emergency rooms and series 2 athletes were cooled in medical tents with tub immersion.

While hot humid environments and dehydration are often considered the primary antecedent conditions for exertional heat stroke, there are other factors that seem to play a critical role in the evolution of exertional heat stroke in a given athlete⁽¹⁴⁻¹⁷⁾. Dehydration to a clinically significant level of greater than 5-10% of body water loss will certainly decrease the ability to produce sweat, decrease the cardiac output for body systems support, and decrease the critical cardiovascular transport of heat from the core to the body surface. This in turn accelerates the core temperature elevation. However, heat stroke occurs in athletes who have adequate body water, particularly in shorter road races^(12,13). Individual factors like acclimatization to heat, recent viral illness, medications, and vasoactive supplement use seem to play a role in many cas-

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es⁽¹⁸⁻²¹⁾. The individual drive to excel or perform at maximal levels despite the heat or preexisting risk factors seems to be a factor in many cases. For example, three marathon runners with exertional heat stroke in relatively safe conditions with WBGT < 14°C trying to make personal goals of qualifying for the Boston marathon, finishing in < 4 hours, and finishing in < 5 hr, 20 min all collapsed with exertional heat stroke; two before the finish line and one immediately after the finish line, all at or near their intended pace. (Unpublished data, W Roberts) One had been ill with a viral infection in the week prior; the others were well with no identifiable risk factors. In a review of several American rules football deaths, one of the common factors shared by these young athletes was an unbridled will to achieve and excel no matter what the conditions. Add to this innate drive, heat trapping protective equipment and some athletes end up with dramatically elevated core temperatures that destroy tissues and disrupt organ systems⁽²²⁾. What triggers the loss of heat dissipation in these athletes and what allows them to continue activity despite a critically rising core temperature remain unanswered questions.

RECOGNIZING HEAT EXERTIONAL HEAT STROKE

When an athlete collapses in hot humid conditions, the set of potential conditions that cause the collapse is relatively narrow and includes exertional heat stroke. A rectal temperature measurement can rule out exertional heat stroke quickly and is the simplest means of detecting EHS^(8,11,13,23). The more convenient body temperature measurements of skin, aural canal, oral subglottic, and axillary regions are not accurate or precise measures of core body temperature in athletes who are often actively cooling at the shell level^(8,10,23,24). The athletes who actually collapse to the ground are the easiest to detect, and a rectal temperature should be measured as a routine vital sign in collapsed athletes, even in "safe" temperature and humidity ranges.

The greater challenge in recognizing exertional heat stroke occurs when athletes are able to keep their feet under them with core temperatures that are dangerously elevated. It is these cases where the diagnosis of EHS is often delayed or missed, and the treatment delays lead to adverse outcomes. In the usual scenario, an athlete heats and fatigues to a point that the brain shuts down the musculoskeletal system and the athlete either collapses or discontinues activity⁽²⁵⁾. Some of these athletes with "heat exhaustion" are indistinguishable from exertional heat stroke casualties and a rectal temperature measurement will be required to rule out heat stroke.

The signs and symptoms of EHS, other than the rectal temperature measurement of > 40 EC, are neither sensitive nor specific. Hyperthermia can range from range 40 to 45 and hyperthermia in the range of 40-42 EC without heat stroke has been documented. Symptoms of EHS include fatigue, impaired judgment, weakness, flushing, chills, dizziness, and change in personality (often subtle). Signs include hyperventilation (think of panting and Aprimal cooling@ like a dog), hypotension, tachycardia (elevated heart rates over 100 and often over 150 when the heart is failing), CNS depression, skin color changes consistent with shock, and occasionally seizure. CNS depression is the most important field marker of heat stroke. The changes may vary from subtle personality changes to bizarre behavior. This may be the first and only sign of EHS until the athlete collapses and is unable to get up. Athletes often have no memory of event from time of critical brain temperature elevation. It is not unusual for road racers to stop their watches to record the race time and not remember the act. In contact sports this memory loss can be mistaken for head injury. Athletes with EHS are often described as having a blank stare, or as the saying goes, "lights on, no one home." In quadruped mammals, the loss of hind limb function is common as the body overheats. This translates to lower limb dysfunction and collapse in humans. Left un-

treated, athletes will linger in mild delirium and eventually drift into stupor or coma. The mild delirium can persist for an hour or more, but the heat induced organ damage only intensifies during this time and the chances for survival plunge.

The textbook description of skin color and appearance in heat stroke is dry, pink or red, and hot. In EHS, this is a late finding and clinging to the classic description of the skin is a common cause for late recognition of heat stroke. The most common skin findings in exertional heat stroke are similar to shock with circulatory collapse. The skin is usually sweaty and often cool as the body is still actively attempting to cool. Once sweating has stopped, the skin becomes pink in color and hot to the touch; this is the common finding in severely dehydrated athletes and athletes who have prolonged high brain temperatures that induce hypothalamic failure. Autonomic nervous system dysfunction occurs with hypothalamic failure and is accompanied by miosis (pin point pupils), decreased pain response, and anhidrosis. If you screen for exertional heat stroke with dry skin you will miss or delay the diagnosis. Seizure is not common in exertional heat stroke, until the brain has been "cooked" for a considerable length of time and is rare in athletes who are diagnosed and treated promptly. The phenomenon of phantom running where the athlete continues to make a running motion with the arms and legs can be confused with seizure and is an occasional finding in exertional heat stroke.

The laboratory and diagnostic imaging changes that occur in exertional heat stroke depend upon the duration of critical hyperthermia levels. In the field, blood studies are used to assess for hyponatremia (serum Na⁺), hypoglycemia, hydration estimate (BUN, hct), pulmonary status (% O₂ saturation), and renal status (BUN, hct, K⁺). The field diagnosis of EHS does not require laboratory studies. In the hospital setting, the hepatic, renal, muscle, hematological, and coagulation systems should be assessed serially to determine the cares needed for survival. Rhabdomyolysis is common in all road racers, especially at the longer distances. In EHS, the risk of symptomatic rhabdomyolysis rises dramatically if the renal blood flow is not sustained and myoglobin precipitates in the kidneys⁽²⁶⁾. Athletes who are not recognized and treated within the "golden hour" of degree minutes will need careful attention to fluid status to protect the kidneys from the effects of rhabdomyolysis and the other organs from shock^(11,27-29). The heart is heat sensitive and echocardiograms following heat stroke often show high output failure patterns with markedly decreased ejection fraction⁽¹⁷⁾. This cardiac muscle depression can be completely reversed with rapid cooling. Renal function can also shut down as a result of the heat insult, but can return to normal if cooling occurs before there is permanent cell death and change in the renal structure.

TREATING EXERTIONAL HEAT STROKE

Once hyperthermia with associated CNS changes has been identified in an athlete, the treatment is immediate whole body cooling^(11-13,30-33). The most rapid form of body cooling is ice water tub immersion with cooling rate that can exceed 8°C per hour⁽¹²⁾. When tubs are not available or when the athlete is not able to be immersed in a tub for body size or medical complications, rapidly rotating ice water towels or sheets is nearly as effective with cooling rates in the 7°C per hour range. (Personal observation, W Roberts, MD) The ice water towels can be augmented with ice packs over the major heat loss areas in the groin, neck, and axilla⁽³⁴⁾. Both the immersion and the ice towel techniques are conductive heat loss measures. The heat exchange is rapid and consistent. Fan and mist or evaporative cooling may be effective in some situations, but the cooling rates in actual heat stroke patients have not compared well (approximately half) to immersion and rotating towels (see figure 1), especially if used in humid conditions⁽¹²⁾. The textbooks still refer to the fan and mist technique as the meth-

od of choice, but in the field it is not very effective. Fans may be used to augment the ice towel or immersion techniques.

The immersion and rotating towel methods are pictured in figures 2 and 3. Tub immersion requires a shallow tub half filled with a mixture of ice and water. The athlete is placed in the tub and the head is supported to keep the mouth and nose above water. Body parts that are not immersed in the tub can be sponged with the ice water or covered with ice water towels. When a tub is not available, the ice water is mixed in a container, like a large cooler, and hand towels or sheets are soaked, wrung out, and placed on the athlete. Two providers working with 4-6 towels each can soak and exchange the towels rapidly and produce swift drops in core temperature.



Fig. 2 – Immersion in an tub of ice water for EHS treatment

(Photo courtesy of William O Roberts MD)



Fig. 3 – Cooling with rotating ice water soaked towels

(Photo courtesy of William O Roberts MD)

In very large athletes, an internal form of conductive heat exchange like gastric lavage may be needed to bring the temperature down, however the towel method should be effective if the cardiac output is maintained⁽²⁹⁾. Cardiopulmonary bypass appara-

tus and gastric or peritoneal lavage could also be attempted to remove body heat (as is done in severe hypothermia to add body heat), but the delays to implement these more invasive procedures could do more harm than good.

The prognosis for EHS casualties is based on the duration of hyperthermia and the response to cooling. Patients who “wake up” during the cooling treatment usually do well⁽²⁷⁾. Casualties who remain comatose, stuporous, or inappropriate in their verbal and physical responses often have bad outcomes. Organ system support is needed when there is delay in recognition and treatment or the patient does not “wake up,” because the tissue damage will suppress normal function. Exertional heat stroke casualties may require intubation for airway management if in stupor or coma to protect the lungs from aspiration. The tachycardia associated with EHS will usually relent as the body temperature is decreased and the heat induced pump failure is restored⁽³⁵⁾. More than one athlete has succumbed to elevated body temperature while the medical staff concentrated on cardiac abnormalities that were heat related. The overheated kidney will often fail to produce urine and renal support with dialysis may be necessary. If the kidney tissue is cooled to normal range “in time,” the renal function can return to normal almost as if turned on by a switch. Respiratory gas exchange is also tissue temperature dependent, but pulmonary function may also be affected by tissue edema that can result from vigorous fluid resuscitation. Organ preservation in shock situations is augmented by vascular flow and normal saline fluid resuscitation is critical to tissue survival⁽²⁸⁾. In EHS patients who do not respond to cooling, monitoring central venous pressures will improve the safety profile during large volume fluid administration^(17,28).

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