Exertional Heat Stroke during a Cool Weather Marathon: A Case Study

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ABSTRACT

ROBERTS W. O. Exertional Heat Stroke during a Cool Weather Marathon: A Case Study. Med. Sci. Sports Exerc., Vol. 38, No. 7, pp. 1197–1203, 2006. A well-trained male runner in his late 30s collapsed 10 m before the finish line, nearly completing the 42.1-km marathon course in 3 h, 15 min. He was responsive to pain, agitated, diaphoretic, and unable to walk. The race start temperature was 6°C (43°F) with relative humidity of 99% and the 3-h temperature was 9.5°C (49°F) with a 62% relative humidity. Approximately 27 min after his collapse, his rectal temperature in the emergency department was 40.7°C (105.3°F), and his failing respiratory status required intubation. His initial Glasgow coma score was 6–7 of 15. His renal output was minimal until he was cooled and given a large fluid flush. His initial echocardiogram showed a “stunned” myocardium with an ejection fraction of 35%. He had a viral syndrome the week prior to the race and was paced by a “fresh” runner the last 16 km of the race. He left the hospital in 5 d and has now returned to running without problems, although several months passed before he felt well while exercising. Exertional heat stroke can occur in cool conditions, and rectal temperature should be checked in all collapsed runners who do not progress with rapid recovery of vital signs and cognitive function. Runners should be instructed not to compete when ill and should not use nonparticipant pacers during the runs. Key Words: COOLING PROTOCOLS, TEMPERATURE MEASUREMENT, ROAD RACE MEDICAL INJURY, CARDIOVASCULAR CHANGES, RENAL CHANGES

Exertional heat stroke (EHS) occurs when an athlete produces more metabolic heat than can be dissipated into the environment, resulting in heat storage and core body temperature elevation. The risk of developing EHS is highest during intense or fast-paced activity in hot, humid weather, but it also can occur in conditions that are generally considered safe for running competitions. EHS is an infrequent problem in marathon racing, but it does occur and can be fatal if it is not recognized and treated promptly. It is one potentially fatal cause of runner collapse during or immediately after competitions and must be ruled out early in the decision process to avoid the complications of delayed diagnosis and prolonged elevation of core body temperature. It is difficult to design studies to evaluate the natural history of EHS because the body temperatures are well above the safety levels that an investigational review board would allow in human subject studies. Much of what we know about the field care of EHS comes from analysis of onsite care and individual case review. Collecting and sharing the relevant clinical information can advance the prehospital and hospital emergency care of EHS, and randomized studies of cooling versus observation care are unlikely because treatment delays increase morbidity and mortality for the athletes. The care of EHS has been advanced by the treatment experiences at onsite “heat labs” such as military training facilities, football practices, and road races.

The following case of EHS occurred during a cool fall marathon. It is significant because the runner had severe cardiovascular, renal, and other organs system changes that fully recovered, and the environmental conditions were well below the usual temperature and relative humidity ranges associated with life-threatening EHS. The patient has given permission for this case to be published and used for educational purposes.

CASE PRESENTATION

The marathon race started at 0800 h in cool conditions with a 6.1°C (43°F) ambient temperature and a 99% relative humidity. At 1100 h the ambient temperature was 9.4°C
from the initial temperature measurement, which was taken approximately 37 deg from the time of collapse.

The area under the curve above 40°C with cooling initiated at 1145 h.

He was transferred to a nearby level 1 emergency hospital. During his initial evaluation. He was drooling and the medical team was concerned about his airway and possible aspiration. He had purposeless leg movement and overall clinical course, was transferred to a nearby level 1 emergency hospital.

On arrival in the emergency department, the runner was pale, diaphoretic, not handling his oral secretions, and he had an initial Glasgow coma score of 6–7. His blood pressure was 124/42, pulse was in a regular rhythm at 144 bpm, and respiratory rate was 20 breaths per minute, and rectal temperature was 37.8°C at 19 min, 38.2°C at 31 min, and 36.8°C (98.4°F) at 45 min from the initial temperature measurement, which was taken about 27 min after his collapse. The body cooling curve is depicted in Figure 1. The temperatures measured by an external (shell) thermometer were 37.7, 38.8, 38.7, and 38°C at the same times as the rectal temperature listed above. His heart rate dropped into the 80s after he was successfully cooled.

He had bilateral movement of the extremities interpreted as seizure activity that was suppressed with 6 mg of lorazepam, although this may have been “phantom running” motion that is often seen in hyperthermic and hypotenemetic encephalopathy. His admission blood work and follow up values in Table 1 demonstrate heat-induced changes and damage to the cardiac, renal, hepatic, and anticoagulation systems that corrected during the admission, but continued to worsen after body cooling was completed. His initial hemoglobin and hematocrit levels of 15.1 g·dL⁻¹ and 45.8%, respectively, along with serum sodium and BUN levels, did not suggest severe dehydration. An initial portable CXR showed his endotracheal tube in good position and no lung abnormalities. His EKG showed sinus tachycardia with an incomplete RBBB (Figure 2) and could be consistent with patterns found normally in an endurance-trained athlete. An echocardiogram done shortly after admission revealed a “stunned myocardium” with global left and right ventricular hypokinesis and a depressed ejection fraction (EF) of 35%. He was aggressively hydrated with 6 L of fluid in the first 3 h of resuscitation in response to suspected dehydration and to protect the vital organs from shock. He had only 4–5 mL of bloody urine output in the first 1–2 h of admission, and then he began a massive diuresis as his direct renal heat injury and presumed dehydration resolved. Dialysis was being considered at the time his renal function resumed. On admission his creatine kinase (CK) was 890 U·L⁻¹, which clinically defines rhabdomyolysis but is an average level for marathon finishers. His CK peaked at 37,000 on

<table>
<thead>
<tr>
<th>Day</th>
<th>Na</th>
<th>K</th>
<th>CK</th>
<th>Troponin</th>
<th>AST/ALT</th>
<th>PT</th>
<th>aPTT</th>
<th>Fibrinogen</th>
<th>BUN</th>
<th>Cr</th>
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<tr>
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<td>mmol L⁻¹</td>
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<td>3.5–5.3</td>
<td>&lt; 135</td>
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<td>&lt; 45–55</td>
<td>11.1–13.2</td>
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<td>171–375</td>
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<td>143</td>
<td>5.3</td>
<td>849</td>
<td>1.49</td>
<td>54/23</td>
<td>14</td>
<td>20.2</td>
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<td>ICU</td>
<td>143</td>
<td>3.3</td>
<td>7485</td>
<td>7.2</td>
<td>15</td>
<td>15</td>
<td>20.7</td>
<td>132</td>
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<td>Day 1</td>
<td>143</td>
<td>3.0</td>
<td>37162</td>
<td>9.78</td>
<td>105/502</td>
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<td>35.9</td>
<td>120</td>
<td>15</td>
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<td>135</td>
<td>3.4</td>
<td>20649</td>
<td>0.92</td>
<td>6</td>
<td>21.3</td>
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<td>9448</td>
<td>1.45</td>
<td>5</td>
<td>14.5</td>
<td>258</td>
<td>5</td>
<td>1.0</td>
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<td>Day 4</td>
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<td>5382</td>
<td>13.5</td>
<td>373</td>
<td>8</td>
<td>1.0</td>
<td>80</td>
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day 1 of admission. His urine was alkalinized to protect from myoglobin precipitation in the kidney, and each hour he was given approximately 500 mL of fluid alkalinized with sodium bicarbonate. His urine pH levels were 5.5 in the ER, 5.5 in the ICU, and 9 on day 1, with the blood gases reflecting the acid–base changes in Table 2.

At about 1330 h, he began to seem more oriented and alert to the nurses. His head CT scan was normal. His body

<table>
<thead>
<tr>
<th>pH</th>
<th>pCO₂</th>
<th>pO₂</th>
<th>HCO₃⁻</th>
<th>Base Excess</th>
<th>Base Deficit</th>
<th>O₂ Content</th>
<th>O₂ Sat</th>
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<td>22–26</td>
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<td>450</td>
<td>16.6</td>
<td>5.4</td>
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<tr>
<td>ICU</td>
<td>7.33</td>
<td>39</td>
<td>183</td>
<td>20</td>
<td>5.4</td>
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<td>147</td>
<td>28.5</td>
<td>5.4</td>
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temperature rose to the 38–38.5°C range at 1400 h and remained elevated until the next morning. At 1600 h the nurses charted that he was alert and oriented x 3. From his recollection, he “woke up” about at 2000 h on the evening of his collapse, still intubated. He was extubated at 1100 h on day 1 of his admission. He has no memory of the event from just before his collapse until he “woke up.” On day 2, a repeat echocardiogram showed an EF of 50% and normal LV and RV function demonstrating a reversal of his heat-induced cardiac depression. He was discharged home on day 5.

In the days following his collapse, additional history was obtained from the runner and his family. He was 71 inches tall and an experienced, well-trained runner with seven previous marathon finishes and a personal best of 3 h, 17 min. His training regimen for this race was similar to his previous marathons, and he did a 2-wk taper with more rest than usual because of a respiratory infection. He had completed “hotter” marathons in similar time to this effort. He related that he was a “heavy sweater” in humid conditions. He ingested fluids during the race by alternating water and sports drinks at each fluid station along the course at a volume that should have kept him reasonably hydrated. The fluid stations are located at 2.5 miles, 5 miles, and then every odd mile to the finish along the course. He did not feel any more dehydrated “than usual” during the race. There is no family history of malignant hyperthermia, although his father “has never done well in the heat”; neither the runner nor any of his relatives have had problems with general anesthetics. He had been ill with a sore throat and general malaise the week preceding the race and was still recovering on race day. He used no medications for his viral syndrome, but did take extra vitamin C tablets and an herbal remedy that did not contain ephedra. He was trying to qualify for the Boston Marathon and required a finish time of 3 h and 15 min or faster for his age group. He maintained the pace through the first 26 km to complete the marathon in the required time. He recalls that he felt hot beginning at 10 km into the race and he was sponging his head to cool himself though most of the race. With about 8 km left in the race he remembers feeling very hot, but he did not feel that he was in danger. To increase his chances of achieving the qualifying standard, he was paced by a friend for the last 16 km of the race, although his friend had difficulty keeping up with him and mostly functioned to encourage him to continue the pace. About 1200 m before the finish line, he began to hyperventilate, and he walked for a minute or two to catch his breath. He was able to resume running and remembers cresting a hill about 800 m from the finish line; at the same time, his heart monitor showed an increase in rate from the about 140 to about 170. He did not note any other problems prior to cresting the hill, and the last thing he remembers is picking up the pace for the downhill run into the finish area. His pacing friend did not notice anything unusual other than a pale appearance; he related that the runner’s legs “locked up” near the finish and that he collapsed to the ground before crossing the finish line.

The runner relates that his recovery from the EHS episode was slow and that he felt ill for 2 months after the incident. He found that he could not eat sweets without feeling ill for several months. A follow-up exercise stress echocardiogram at 2 months postincident was normal, with a normal EF and no signs of ischemic heart disease. He resumed light running after the normal echocardiogram. His muscles ached with any activity for 3 months; in particular, his hamstring muscles were painful and he had soreness and burning in all his running muscles. He did not have a formal return to activity program; he just started running about 3 miles every couple of days and advanced as tolerated. By 6 months postincident, he was running regularly, but he found it difficult to push his training. At 10 months he was able to run a strenuous and hilly 5-mile race in hot conditions, and he felt fine at the end of the race. He was considering training for his next marathon 14 months postincident.

**DISCUSSION**

EHS is a true medical emergency that requires immediate cooling for the best outcomes (6). The usual mechanism for developing EHS is excessive endogenous heat production combined with environmentally impaired heat dissipation, but as this case demonstrates, other factors may be involved (12,24). EHS in road racing is usually associated with shorter fast-paced events like the Falmouth (MA) Road Race (FRR), which is run along 11.5 km of the south Cape Cod coast in mid-August. The FRR medical team treats 1–2 EHS cases per 1000 entrants each year (2,8). In hot and humid conditions like FRR, ice-water tub immersion is probably more effective for rapidly decreasing core temperature. In contrast, EHS is comparatively rare at the Twin Cities Marathon, with 1–2 EHS cases per 10,000 finishers (26). Although fast-paced running or other intense activity in hot humid environments pose the greatest risk, EHS can occur in surprisingly cool conditions, as demonstrated by this case (24). In a study of the minimum temperatures that result in death, the minimum mortality bands occur at higher temperatures as the mean summer temperature rises and the mortality incidence is greater at a lower temperature range in the upper latitudes compared with more equatorial latitudes, implying acclimatization from living in conditions of the area (19). A sudden increase in ambient temperature on race day can increase the risk of EHS.

In addition to physiologic adaptation to heat stress, athletes have behavioral adaptations to protect them. Athletes generally stop exercising when heat stress is perceived during activity, but during competition may ignore these normal behavioral responses. The central control model of heat illness suggests that a central governor of activity should prevent an exercising person from developing EHS by causing the athlete with hyperthermia affecting the brain to “fall out” of activity (23,33). Highly motivated athletes, like this runner, who was paced by a fresh runner for the last 16 km of his run, may be able to override the protective function of the central governor, thereby allowing higher levels of core organ overheating and eventual collapse with true EHS.
The symptoms of EHS are not specific or sensitive and include fatigue, impaired judgment, weakness, flushing, chills, hyperventilation, dizziness, and collapse (13). EHS is more obvious when the athlete collapses to the ground, but even then EHS can be missed if there is not a strong index of suspicion. The diagnostic criteria for EHS are a rectal (core) temperature > 40°C combined with CNS or other organ dysfunction (6,28). EHS must be considered in the basic collapsed runner differential diagnosis that also includes cardiac arrest, exertional hyponatremia, hypoglycemia, and exercise-associated collapse (EAC) (26–28). In this case, the medical team ruled out cardiac arrest and exertional hyponatremia. The rectal temperature could not be safely measured, and EHS had to be ruled out. With the poor airway control, transfer to an emergency facility was considered imperative for the runner’s safety. If not heat stroke, then other catastrophic events could be ruled out in the emergency facility.

Recognition and cooling are the key factors to EHS survival, and the prognosis is good with early recognition and rapid cooling, whereas the prognosis is less favorable with delayed recognition and slow cooling times (8,10,28). Elevated cell temperature associated with EHS causes accelerated cell damage and cell metabolism (18). Medical providers must think “heat attack” with the response time of “heat attack” to reduce morbidity and mortality in athletes stricken with EHS. For athletes who are stable from a cardiorespiratory perspective, it is best to treat with onsite cooling using ice-water tub immersion or cooling techniques with similar cooling rates, and then transfer to an emergency facility for ongoing medical issues as rapid cooling is critical to a favorable outcome (8,10,28). The overall risk of EHS is related to the area under the cooling curve in degree-minutes rather than the absolute core temperature peak (Figure 1). If one assumes a spontaneous cooling rate of 0.03–0.05°C in 9°C ambient temperature for a thin male, this runner’s collapse temperature would have been about 0.8–1.4°C higher than his initial temperature in the emergency department, or 41.5–42.1°C. Based on this assumption, this patient had approximately 37 deg·min above 40°C after his collapse. Somewhere in the range of 60 deg(C)-min above 40°C markedly increases the risk of death based on fatal EHS case reviews. It is likely that his body temperature had risen above critical levels when he began hyperventilating and walked to catch his breath near the end of the race. That would add additional degree-minutes to his profile.

The diagnosis of EHS is dependent upon a reliable estimate of core body temperature. Rectal temperature is the only accessible and accurate field site for core temperature estimates (3,7,11,25), and the diagnostic criteria and treatment protocols for EHS are based on rectal temperature. The core-shell temperatures can differ by 5°C in EHS cases (25). The underlying problem in EHS is core heat accumulation and organ tissue destruction, which does not require that the cooling functions at the shell level be dysfunctional, just not effective for removing the body heat load. Body temperature measurements at convenient sites like the mouth, nasopharynx, aural canal, facial skin, and axilla are influenced by the shell temperature and do not accurately measure core temperature in athletes (3,7,11,25), and this case demonstrates that shell temperatures are spuriously low, potentially directing care away from the underlying pathology. There are two confounding situations that often delay the temperature measurement and, in turn, the diagnosis in athletes. The first is an “interlude” or lucid interval of seemingly normal behavior, but different for the individual, which gives medical providers a false sense of security; the second is assuming that sweaty, cool skin reflects a cool core temperature (9). In this case, excessive leg movement and low index of suspicion delayed the temperature measure and diagnosis until the athlete was in the emergency room.

The field treatment of EHS is immediate cooling (8,10,28). Immersion conduction techniques placing the trunk and extremities in tubs filled with ice and water induce cooling rates averaging 8°C·h⁻¹ (17°F) and have the advantage of hydrostatic pressure that improves blood pressure and cardiac output (2,8,10,32). Ice packs to neck, axilla, and groin cool at a rate that is half that of immersion technique (2). Recent experience combining ice packs to the neck, axilla, and groin with rapidly rotating ice-water–soaked towels to the extremities, trunk, and head has produced cooling rates in the 7°C·h⁻¹ range has been used to successfully treat EHS at the Twin Cities, Chicago, and Marine Corps Marathons. The usual endpoint for cooling therapy is 39°C (102°F) to prevent overcooling (8,10,28), but there may be some advantage to overcooler as is now recommended in acute myocardial infarction. Dehydration is more of an issue in longer-duration races, but need not be present to develop EHS (24,29,30). Cooling promotes extracellular fluid return to intravascular space, and careful fluid replacement should be utilized until the athlete is cooled and hydration status can be better judged or oral replacement can be accomplished (30). Diazepam, midazolam, or magnesium sulfate can be used to control shivering, seizures, or persistent muscle spasms.

In the cool, relatively dry air on the usual Twin Cities Marathon race day, hyperthermic casualties (T_rectal > 40°C) usually cool quickly with ice packs to high–heat-loss areas of the neck, axilla, and groin (16), but the addition of rotating ice-water towels to the extremities, trunk, extremities, and head improves the cooling rate and drops rectal temperatures to < 39°C (102°F) within 20–30 min. Of the 20+ runners with rectal temperatures > 42°C (106°F) who collapsed, only four have been transferred to the emergency room from the medical tent. Of these runners who met criteria for EHS, only two, including this case, have required hospital admission.

Runners with the diagnosis of EHS who recover rapidly (return to “normal” in 30–60 min) and are clinically stable and normothermic are often released from the medical area (2,8,26,28). The EHS runners who “wake up” quickly with body cooling seem to require little more than fluid replacement for treatment after the cooling therapy is completed. These recovered casualties should be given instructions for fluid replacement and should be advised to seek evaluation.
if they experience a change in status. It may be prudent to have them follow up with their primary physician the next day for liver function and metabolic testing (i.e., AST, ALT, CK, BUN, creatinine, Na, K, Cl, Glc). Any runner who is not responding to treatment or who has any complicating factors, like the runner in this case, should be transferred to an emergency facility (28).

The underlying question in this case is, “Why on a cool, clear morning would a runner wearing a singlet and shorts experience a near-fatal heat stroke?” The environment was nearly perfect for marathon racing, even with the radiant heat load (1,21). The calculated wet-bulb globe temperature was 7.9°C (46.3°F) at the time of this runner’s collapse, with very low risk for EHS (1). The low ambient temperature and 60% relative humidity should have maximized heat loss through evaporation, convection, and conduction. With ambient conditions so conducive to heat loss, EHS was not an expected outcome, and other nonthermal considerations in temperature regulation including hydration status, recent viral illness, and utilizing a race pacer most likely played a role. His viral illness in the week prior to the race may have been the factor that altered his body heat loss. He had used no cold remedies containing ephedrine. He was well trained, and the day was not “hot” compared with the previous 7 d with similar ambient temperatures. The other factor that weighs on this case is the use of a pacier for the last 16 km, which may have allowed the runner to overcome his “central governor” and push himself into the EHS range (23,33).

This case also demonstrates that EHS organ system pathophysiology is reversible when cooling is accomplished in a reasonable time frame. As heat accumulates in cells, the organs heated above critical levels begin to fail due to direct heat injury (5,14–16,18,20,31). This runner had central nervous system failure with loss of neural control, absent cognitive function, and possible seizure activity after his collapse. On initial echocardiography he demonstrated cardiovascular fatigue and decreased pump efficiency shortly after his collapse that returned to normal on retesting. Although his CK levels show work- and heat-related muscle damage, the elevated Troponin I levels suggest that there was generalized cardiac muscle damage in addition to skeletal muscle damage, although there are no focal ischemic findings on his EKG tracings. It is unlikely that his initial EF of 35% would sustain over 3 h of running at his race pace. He had direct heat-induced renal filter failure that reversed after cooling. His muscle cell walls failed, but the cell content leak did not cause renal failure because he was hydrated to maintain brisk renal blood flow as part of his in-hospital treatment. His legs were restless and “stiff” after his collapse, and perhaps he should be tested for RYRI gene abnormalities associated with malignant hyperthermia (4,22). His shocklike appearance may have been due to gut cell failure and vascular contamination leaks or the low-output cardiovascular failure. These changes reflect the pathophysiology of EHS with partial or total system failures, loss of thermoregulatory control, and cardiac pump failure that accompanies rising core temperatures.

The learning points from this case are important for both runners and medical providers. EHS can occur in normally safe running conditions, so medical teams should always consider EHS in the differential of a collapsed athlete. This means checking rectal temperatures in athletes who collapse even in cooler conditions. Runners should not run hard or not run at all when ill. Outside pacing is a practice that should be abandoned because it changes the “behavioral” adaptations for runners who would most likely slow the pace when feeling ill. Although dehydration is a risk factor for heat stroke in longer distance races like the marathon compared with 10- to 15-km races, it may not have been a primary factor in this collapse based on the patient’s history of fluid intake. As demonstrated in this case, rapid intervention is life saving, and organ changes associated with EHS can be reversible if casualties are cooled quickly. It may be prudent to start cooling empirically when a rectal temperature cannot be obtained, and there may be some rationale in runners who have more prolonged temperature elevations, shock, and do not “wake up” during cooling to drop body temperatures below the normal body temperature, as is now recommended in myocardial infarction. Runners may benefit from running with a buddy to watch each other for changes of EHS.

REFERENCES


