The “Golden Hour” for Heatstroke Treatment

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The “Golden Hour” for Heatstroke Treatment

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Exertional heatstroke is a life-threatening event. It occurs mainly among the young healthy populations of athletes and soldiers. The severity of exertional heatstroke is directly correlated to the area under the temperature duration curve. Therefore, rapid cooling in the field has an enormous effect on prognosis. Four cases of exertional heatstroke are presented which differ in their outcome. We argue that there is a “window time period” within which simple and effective cooling techniques can determine prognosis.

Introduction

Heatstroke is one of the most serious conditions associated with elevated body temperature.1 It occurs when metabolic and environmental accumulated heat exceeds the body’s ability to dissipate it. The elevated body core temperature becomes a noxious agent, causing damage to the body’s tissues and resulting in a characteristic multorgan clinical and pathological syndrome, which is occasionally fatal.2,3

Heatstroke may appear in one of two forms: classical heatstroke or exertional heatstroke (EHS). The former affects mainly the elderly during severe heat waves.4,5 The latter is a sporadic event occurring among the young healthy population of soldiers, athletes, miners, etc., and is associated with heavy exertions, not necessarily held under heavy heat loads.4,6 Complications of EHS may include hepatic failure, renal failure, disseminated intravascular coagulation, rhabdomyolysis, and the adult respiratory distress syndrome.2,7,8

The severity of EHS can be affected by the duration of unconsciousness (less than 1 hour in mild cases and 3 to 24 hours in severe cases) and by the levels of hepatic and coagulation enzyme disturbances.2,7,8 The severity of EHS has been shown to be directly and closely correlated with the area under the temperature-duration curve.10 Thus, decreasing the length of time at which the body remains above a critical temperature should have a crucial influence on the EHS victim’s outcome and prognosis.5,10

In this article, we present four cases of EHS that differ in their severity and outcome, which we argue was mainly attributed to the different time intervals until proper treatment was initiated. These cases demonstrate the importance of the “golden hour” as a lifesaving time interval in the case of EHS.

Case Reports

Case 1

A 26-year-old previously healthy man participated in a pre-draft military selection program, which took place during moderate heat load (ambient temperature ($T_a$) = 24°C (45.3°F), relative humidity (RH) = 60%). The exercise regimen consisted of short and long running distances with various back loads and other various interval trainings. After 2 hours of high-intensity exercises, the trainee collapsed. His rectal temperature ($T_r$), measured on site with a clinical thermometer, was above 42.5°C (55.6°F) (the end of the thermometer’s scale). Cooling treatment by splashing copious amounts of water on his body was immediately initiated. In the emergency room, 45 minutes later, the patient was psychotic, aggressive, and with a $T_r$ of 40.0°C (54.2°F). Within 1 hour of treatment consisting of continued cooling, intravenous fluids, and intramuscular haloperidol, $T_r$ decreased to 37.5°C (52.8°F). One hour later, the soldier recovered to full consciousness. He was then hospitalized for 3 days, during which time he was free from complaints. Laboratory evidence revealed muscular damage with creatine phosphokinase activity peaking at 6,010 U/L 24 hours from the collapse and an increase in liver enzymes: alanine aminotransferase, 97 U/L; aspartate aminotransferase, 228 U/L; lactate dehydrogenase, 392 U/L; prothrombin time, 74% (normal range, 70%–120%); and partial thromboplastin time, 28 seconds (normal range, 28–40 seconds). There was no other evidence of liver dysfunction. All laboratory results returned to normal values within 1 week after the collapse. A heat tolerance test was performed according to Shapiro et al.11 after 8 weeks and demonstrated normal physiological response to exercise heat stress.

Case 2

An 18-year-old soldier collapsed at the end of a 12-km march (2.5-hour duration) held under moderate heat load ($T_a$ = 26°C (46.4°F), RH = 50%). Rectal temperature measured upon collapse was 41.0°C (54.8°F). The soldier was immediately cooled by extensive amounts of tap water and cold intravenous infusion. On his arrival at the emergency ward 1 hour later, the soldier was fully conscious with $T_r$ = 38.0°C (53.1°F). Laboratory examination the following day revealed muscle damage with creatine phosphokinase activity levels peaking at 44,000 and hepatic elevated enzymes: alanine aminotransferase, 250 U/L; aspartate aminotransferase, 600 U/L; and bilirubin, 1.2 mg/dL. There was no evidence of renal or coagulation dysfunction. The soldier was hospitalized for 5 days with no complaints. Eight days later, all laboratory results returned to normal values. A heat tolerance test,11 performed 4 weeks after the EHS occasion demonstrated a normal physiological response to exercise in a hot climate.

Case 3

An 18-year-old military recruit participated in a 5-km (1-hour) night march, carried out under moderate heat load ($T_a$ = 26°C (46.4°F), RH = 78%). At the end of the march, the soldier collapsed. A few minutes later, at the base clinic, the patient was...
delirious with alternating aggressive behavior and vomited several times. Hyperventilation, aggressive reaction, and history of a previously healthy subject participating in a short march led the physician in charge to misdiagnose the condition as conversion reaction and, therefore, body temperature was not measured. After 3 hours, convulsions accompanied by dark vomiting appeared, and the patient was evacuated to a hospital. On admission to the emergency room 4 hours after the collapse, the patient was comatose, in shock (systolic blood pressure, 60), and had a rectal temperature of 39.6°C (54.0°F), which was the first measurement. Laboratory examination revealed: metabolic acidosis (pH 7.2; bicarbonate, 15); creatinine, 4.4 mg/dL; aspartate aminotransferase, 550 U/L; prothrombin time, 10% of normal; and partial thromboplastin time, 85 seconds. Despite intensive treatment including infusion of 12 L of crystalloid fluid, albumin, blood, fresh frozen plasma, dopamine, and steroids (for blood pressure maintenance), the patient died 24 hours later. Blood cultures taken on admission were sterile. Heatstroke diagnosis was confirmed by autopsy.

Case 4
A 20-year-old infantry soldier participated in a competitive 5-km run with full battle gear during moderate heat load (T° = 28°C [47.7°F], RH = 60%). The run took approximately 25 minutes to complete, after which the soldier collapsed. No cooling measures were taken, and he was evacuated to a nearby infirmary. Upon arrival 15 minutes later, he was unconscious, and T° was 40.0°C (54.2°F). After 3 hours, convulsions accompanied by dark vomiting appeared, and the patient was evacuated to a hospital. On admission to the emergency room 4 hours after the collapse, the patient was still unconscious, and T° was 40.0°C (54.2°F). He was intubated and given intravenous fluids and 1 g of paracetamol by suspension. The patient was then admitted to the intensive care unit. He arrived at the intensive care unit 2 hours after his collapse, while his T° was still 39.6°C (54.0°F). Cooling was initiated by repeated cold water gastric lavage. Three hours after his collapse, the patient’s T° was 38.5°C (53.4°F), and shortly afterward he was extubated. The patient was given an additional 2 g of paracetamol during the first day of hospitalization. On the following day, the soldier’s condition deteriorated with evidence of acute hepatic failure, disseminated intravascular coagulation, and adult respiratory distress syndrome. Despite intensive treatment (preparation for liver transplantation was initiated), the patient died of multiple organ failure on the fifth day after his collapse. Postmortem examination confirmed the diagnosis of EHS.

**Discussion**

EHS is a threat to all of those who engage in vigorous physical exertion. It is of particular concern in military situations where the combination of high-intensity exercise coupled with overmotivation and peer pressure may prove to be detrimental. The damage inflicted by EHS is determined by the degree of hyperthermia and its duration and is a function of the temperature-duration area above a critical temperature. Cooling is a lifesaving treatment for EHS victims. It should, therefore, be initiated immediately upon collapse and only minimally delayed for vital resuscitation measures.

In the present study, four cases of EHS that occurred after relatively short periods of exercise are presented. In cases 1 and 2, effective cooling and using large quantities of tap water were applied shortly after the collapse. Both cases enjoyed favorable prognosis with complete recovery after hospitalization of less than a week. Despite the high initial rectal temperatures (42.5°C [55.6°F] and 41.0°C [54.7°F], respectively), the cases were summarized as mild, as evidenced by the relatively short period of unconsciousness, mild to moderate rhabdomyolysis, and hepatoportal dysfunction. The mild course and the good prognosis were in all probability a direct consequence of the rapid cooling rate of 2.5 to 3.0°C (33.4–33.6°F) during the first hour after collapse.

In cases 3 and 4, cooling treatment was delayed. In case 3 the primary physician misdiagnosed the patient as having a conversion reaction. Thus, body temperature was not even measured, a fact that delayed treatment and evacuation. In case 4, EHS was diagnosed by the primary physician at the site of collapse and also in the emergency room. However, this resulted in only futile actions being taken. Efficient cooling was initiated only 3 hours after the collapse when the patient referred to the intensive care unit. Furthermore, the use of paracetamol in this case was particularly unjust and might even have been harmful.

Various cooling methods, such as alcohol sponge, ice packs, cold or ice water immersion, evaporative cooling using air conditioners, powerful fans, or sophisticated “body cooling unit” equipment have been reviewed in the literature. Nevertheless, the necessity to reduce body temperature as quickly as possible requires simple and effective methods that can be applied at the site of collapse. It has been found that the most practical and efficient cooling methods in the field consist of spraying large quantities of tap water (15–16°C [40.3–40.9°F]) on the skin, providing shade, and finding some means of blowing air on the patient. Tap water is readily available in a field scenario, does not require medical expertise, and can be applied by military commanders or organizers of sports events as well. It does not require sophisticated logistic arrangements or equipment, which are required for other cooling techniques. Cooling by tap water, as opposed to ice water, has a physiological rationale, because it eliminates the hazard of cold-induced vasoconstriction and heat-producing shivering, which reduce cooling effectiveness. The physiological advantage of rapid cooling with tap water was endorsed by the American College of Sports Medicine. The college adopted this method as its “method of choice” and incorporated it in their position stand.

Antipyretics have no role in the treatment of EHS, as they only serve to readjust the body’s temperature set point in cases of disease. In EHS, the body temperature’s set point is not elevated but rather overwhelmed. Moreover, paracetamol may be detrimental to the development of fulminant hepatitis in EHS patients because of previous heat-induced hepatic damage. The misdiagnosis in case 3 and the improper futile cooling treatment in case 4 caused a delay in efficient cooling, which only occurred 3 to 4 hours after the collapse. The result of this delayed cooling most probably played a major role in the deaths of the patients.
Summary and Conclusions

All four cases presented herein deal with young, healthy, physically fit subjects in whom EHS occurred after a short duration of strenuous activity held under moderate heat conditions. Thus, all of these cases are similar EHS cases in which the subjects suffered for only a few hours the noxious effect of the heat. Nevertheless, the outcome of the first two cases was totally different compared with the other two cases. In the first two cases, the length of time that elapsed from collapse to the initiation of cooling differed from the last two cases. The first cases were rapidly cooled soon after the collapse by simple available means of tap water, whereas in the other two cases efficient cooling was delayed for more than 3 hours. This was probably detrimental for prognosis. We can conclude from these cases that there is a limited “window time period” within which effective cooling can influence prognosis. Delaying cooling will result in poor prognosis or even death. The term “golden hour,” used in trauma to describe the period until the initiation of treatment, is also relevant for the cooling of heatstroke patients. Initiation of efficient cooling immediately after the EHS event (collapse), beginning at the site of the event, is of crucial importance and cannot be emphasized enough. A delay in the efficient cooling even at the hospital level may have a catastrophic implication on prognosis.

Using large quantities of tap water at the scenario site and during the evacuation has proven to be an effective cooling method, with a rate of 2.5 to 3.0°C/h (33.4–33.6°F/h) reduction in body temperature. Postponing the cooling treatment until more sophisticated equipment is available may be harmful or even life-threatening.

The use of antipyretics in the treatment of heatstroke is contraindicated because there is no change in the hypothalamic temperature set point. However, recent data have shown pyrogenic cytokines to be involved in heatstroke pathogenesis, thus, favoring their use. Case 4 is a “strong” clinical example of the potential hazard that exists in the use of antipyretics (e.g., paracetamol) to cool heatstroke victims. Therefore, our position is against their use in EHS victims.

References