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**Abstract:** This article discusses exertional heatstroke. Issues surrounding heat illness remain somewhat controversial, and our understanding of exertional heatstroke is evolving. The ability of humans to compete and work in hot, humid conditions with a low incidence of exertional heatstroke demonstrates tremendous adaptability. For the most part, the brain does control body temperature during exercise, as apparently demonstrated by researcher Paula Radcliffe and others who drop out of races and football practices when they feel ill and cannot go on. Assuming the brain can modify muscle function to control heat production and turn off activity when it reaches a set temperature or fatigue index, it seems logical that there would be times when the regulatory function could fail. From an evolutionary perspective, the "fight or flight" response could override the central governor function of the brain, as early humans were probably hunted as often as they were hunters. Prompt recognition and immediate total-body cooling will resolve or mitigate the problems of hyperthermia. Late cooling, even if it is rapid, is not as protective or therapeutic in athletes with heatstroke.

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**Section:** Commentary

## Common Threads in a Random Tapestry

### Another Viewpoint on Exertional Heatstroke

Issues surrounding heat illness remain somewhat controversial, and our understanding of exertional heatstroke is evolving. The ability of humans to compete and work in hot, humid conditions with a low incidence of exertional heatstroke demonstrates tremendous

adaptability. On the other hand, the occurrence of exertional heatstroke in conditions that should be prime for all-out physical effort remains puzzling and problematic.

### **The Role of the Brain**

As noted by Dr Noakes (page 39), heatstroke develops when the rate of heat production by the body exceeds the rate of heat loss and total body temperature rises to a level that leads to organ dysfunction and collapse. The athlete's brain should regulate both the rate of heat production by the body and, to some extent, the rate of body heat loss during exercise. It seems illogical that the brain would ever "choose" to allow an athlete to develop heatstroke, because the final outcome will be fatal if the condition is left untreated. The brain should always choose to protect the tissues and the body.

For the most part, the brain does control body temperature during exercise, as apparently demonstrated by Paula Radcliffe and others who drop out of races and football practices (American, international, and Australian rules) when they feel ill and cannot go on. This probably represents a form of brain-mediated "heat exhaustion" that is protective for those working in the heat. Equally important in the overall picture is the relatively rare occurrence of both fatal and nonfatal exertional heatstroke in high-risk conditions. In most cases of exertional heatstroke, there are many other athletes on the practice field or in the competition who handle the workload and heat stress with no apparent problems. What makes those athletes more tolerant of the heat on that given day?

Assuming the brain can modify muscle function to control heat production and turn off activity when it reaches a set temperature or fatigue index, it seems logical that there would be times when the regulatory function could fail. From an evolutionary perspective, the "fight or flight" response could override the central governor function of the brain — as early humans were probably hunted as often as they were hunters.

In today's world, the "fight or flight" response may be mimicked by athletes delivering an extraordinary effort to make the team, qualify for Boston, set a personal record, or respond to a trusted coach to push harder. In most circumstances, the human brain does indeed appropriately set the work rate and number of muscle fibers recruited during exercise according to the rate of accumulating body heat. And I agree with Dr Noakes that this anticipatory response ensures that exercise is almost always terminated before the core temperature reaches dangerous levels. However, note that the use of the word "almost" leaves room for brain failure and other problems associated with the evolution of exertional heatstroke.

Exertional heatstroke, in this model, occurs when the brain fails in its regulatory function to limit muscle work to a level that does not overwhelm the heat dissipation systems. Does this failure occur when the brain itself overheats, and can an individual force failure by voluntary exertion that overrides the regulatory functions?

### **Common Threads to Random Presentations**

The lessons that I have learned from the sidelines, the finish lines, and the court documents seem to support a somewhat random "out of the blue" presentation of exertional heatstroke. Much of what we know about exertional heatstroke as an outcome of exercise comes from these settings, because institutional review boards for human research will not allow us to push athletes into the necessary temperature zones to study the clinical picture of true exertional heatstroke. Of interest, very few of the exertional heatstroke cases that I have treated or reviewed post mortem had a history of heatstroke or heat exhaustion. The military services no longer automatically discharge soldiers who

experience heatstroke during basic training, because recurrence is not that great. However, there are common threads that weave through the individual cases.

One common thread is the lack of acclimatization — gradual exposure to increasing heat loads and work volumes — in athletes who race or practice in hot, humid conditions. Hence, the increased incidence of collapse and heatstroke death in American football during the first few days of preseason practice and the heatstroke cases in road races that are unexpectedly hot. As noted by Schnirring, [ 1 ] there were three other "heat injured" football players from the same team as one of the high school players who died last year. This occurred on the first day of practice, after 16 sprints at the conclusion of a 3.5-hour session on a hot, humid day, leaving in its wake one dead and three in the hospital.

A second thread involves some "other factor" in a given individual that seems to tip the scale of temperature regulation, like concurrent viral illness, use of ephedra, genetic markers for malignant hyperthermia, [ 2 ] loss of intravascular volume with heat transport and sweating capacity deficits, or progressive day-to-day dehydration. It could be that the "other factor" is the difference in the evolution of exertional heatstroke.

A third thread that ties many of the cases together is an "extraordinary effort" to make the team, impress the coach, or reach a race goal. When the combination of voluntary extra effort, lack of acclimatization, and one or more "other factors" allows the body to work above the normally controlled level, sudden collapse occurs when the tissue temperatures inhibit normal brain and cardiac function. At this point, all tissues are heated to critical or near-critical levels, and life hangs in the balance.

The fourth thread is lack of recognition early in the evolution of individual fatal cases of heatstroke. Prompt recognition and immediate total-body cooling will resolve or mitigate the problems of hyperthermia. Late cooling, even if it is rapid, is not as protective or therapeutic in athletes with heatstroke.

### **Reducing Heatstroke**

That said, what is the most effective way to reduce the risk of exertional heatstroke that seemingly strikes from "out of the blue" in athletes? First, activity in hot, humid conditions should be introduced gradually, similar to what is now required in college football practices, which allows athletes to acclimatize to the heat. Second, athletes, coaches, administrators, and parents must be educated regarding the risk of participating in vigorous activity in the face of factors like fever, viral illness, and supplements (like ephedra) — and this must be re-emphasized regularly. Third, monitoring athletes for signs and symptoms during training by coaches and fellow athletes should become a part of sports culture so that athletes will know they can take the safe route when the situation arises.

And finally, recognition and rapid cooling are paramount to survival. Most of the athletes with heatstroke whom I cared for or whose cases I have reviewed had been sweating profusely with cool, wet skin. This single finding, which indicates to me that the body is still trying to cool, is probably the cause for many of the "missed" early diagnoses, because we still teach the classic heatstroke findings of hot, dry skin, as opposed to the pale, wet, and cool skin that manifests early in the course of exertional heatstroke. Another interpretation of this cool, wet skin is that the cardiac output is not adequately supporting blood pressure, and we are seeing a picture consistent with shock.

Athletes who collapse or who are "different" during or following exercise should have a rectal temperature measured to rule out hyperthermia, and, if the temperature is high,

immediate total-body cooling will save lives and tissue. Cooling reverses or prevents cardiac muscle failure, brain failure, liver dysfunction, and clotting abnormalities. Rhabdomyolysis is a prime example, as the heat-damaged muscle will leak its contents with ensuing renal failure in the face of poor renal blood flow. Rapid body cooling to stop the muscle cell leak — along with hydration to flush the kidneys — can prevent symptomatic rhabdomyolysis.

### **Staying Vigilant for Anomalies**

Like many who follow heatstroke in athletes, I am also taken by the relatively rare occurrence of the malady considering the number of people who participate in hot-weather activity. It lends credence to the supposition that some control system must have to fail for heatstroke to occur during activity. It seems well accepted that metabolic heat generated during activity is the most critical factor in the evolution of heatstroke.[ 3]

I have come to accept exertional heatstroke in hot, humid conditions as a relatively predictable, but rare, occurrence. Exertional heatstroke occurs in about 1 in 350,000 US high school and college football players per year. What we do not know is the rate at which this occurs in hot, humid conditions, as many players across the country probably start the season in reasonable environmental conditions. For prevention, we need to look at the environment and the actual time exposure for these rare events to see if we can determine with better precision which players are at most risk.

The football players who die of heatstroke are predictably working out in hot, humid conditions; losing large volumes of sweat; not acclimated to the heat, humidity, and stress of the uniform; and, for the most part, are large men or boys. Some have used ephedra products, some have not eaten well, some are away from home and have not slept well, some have lost large volumes of sweat, but all were working hard and not looking healthy in the eyes of their peers. Perhaps there is some cumulative effect or combination of factors that disrupt the normal governor function of the brain. How long does it take a person who has gone beyond the threshold of heat tolerance into heatstroke to spontaneously cool? Spontaneous cooling likely may not occur in time to prevent death or permanent damage.

The picture of exertional heatstroke is less clear in road racing. Heatstroke is relatively predictable during 10- to 15-km races run in heat and humidity. I am continually amazed that heatstroke can also develop at ambient temperatures as low as 10° C (50°F) in a marathon, both in faster and slower runners. In the past 2 years, I have been on site for 8 cases of heatstroke (among about 80,000 runners) that occurred in temperatures under 18°C (65°F). Of the runners that I have been able to question in some detail about the episodes, most had been ill the week prior to the race, had worn extra clothing to stay warm at the start, or had outside pacing in the latter part of the race.

Once an athlete has collapsed and there is no voluntary muscle contraction, is there continued metabolic heat generation or is there just continued tissue "cooking" with the latent heat? At the finish line and sideline we do not intentionally wait to see what will happen in a collapsed athlete who is identified as having hyperthermia. We do know that when the diagnosis is delayed, the cascade of tissue changes leading to fatal outcome begins and at some point becomes irreversible. Is there continued heat generation beyond basal heat, or is the combination of trapped and basal heat maintaining high core temperature in a system that has already failed to adequately remove metabolic heat? Most athletes who collapse during or after activity got hot because they produced more heat than they could remove, and there is little to support the concept that they would

cool spontaneously in the same environmental conditions at a rate that would preserve tissue.

In most of the deaths that I have reviewed, the athletes had initial rectal temperatures above 42°C (108°F), sometimes measured as much as 2 hours after the collapse. Athletes who collapse from heatstroke in cooler conditions, where the air and ground temperatures are much cooler than body temperature, have been in the 41°C (106°F) range 20 to 30 minutes after the collapse. In my view, it does not take much to maintain a hot core in hot, humid conditions or even in cool air, especially for large men with high heat capacitance. So is there a muscle abnormality that causes all heatstroke casualties to continue producing heat after exercise, or is there stored heat that cannot be dissipated? That question is yet to be resolved.

Heatstroke is not the normal response to exercise. Field experience does support applying the "normal precautions," like maintaining hydration to replace sweat losses during and after activity, avoiding exercise in extreme heat, allowing acclimatization sessions, and avoiding intense activity in the presence of fever, viral illness, vomiting, significant dehydration, and vasoactive substances like ephedra. Even with these precautions, heatstroke will occur. The key to reducing morbidity and mortality lies with a high index of clinical suspicion, the courage to check a rectal temperature, and the ability to provide active cooling on site.

For the small group of athletes who carry a gene for malignant hyperthermia, onsite cooling will not save the day, and intravenous dantrolene is life saving. That requires rapid transport to an emergency department or the capacity to deliver the medication on site. Exertional heatstroke due to known genetic links is too rare an occurrence to support having dantrolene on site for all heatstroke-prone activities.

### **Proven Therapy**

While we do indeed look for obscure explanations for most cases of heatstroke, we should continue to provide field-proven therapy for the heatstroke victims at sport sites. Although it is said that lightning never strikes twice, we now know that there are places where lightning is more prone to strike (and sometimes often). Heatstroke then, like lightning, is unpredictably predictable. Until we can reliably identify "who, when, and where," our current precautions, a high index of suspicion, and quick recognition with rapid total-body cooling constitute the best strategy for reducing death and disability related to exertional heatstroke.

**Disclosure information:** Dr Roberts discloses no significant relationship with any manufacturer of any commercial product mentioned in this article. No drug is mentioned in this article for an unlabeled use.

For author disclosure of financial relationships and mention of unlabeled use of drugs, see the next page.

PHOTO (COLOR)

### **REFERENCES**

- 1. Schnirring L: Heatstroke fatalities fan discussion: two deaths in high school football. Phys Sportsmed 2004; 32(9):8-10*
- 2. Muldoon S, Deuster P, Brandom B, et al: Is there a link between malignant hyperthermia and exertional heat illness? Exerc Sport Sci Rev 2004;32(4):174-179*

*[erratum in Exerc Sport Sci Rev 2005;33(1):60]*

3. *Noakes TD, Myburgh KH, du Plessis J, et al: Metabolic rate, not percent dehydration, predicts rectal temperature in marathon runners. Med Sci Sports Exerc 1991;23(4):443-449*

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