

HEAT STRESS AND ELECTROLYTE IMBALANCE

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Abstract: Core body temperature, water, electrolyte, and sugar control are intimately connected and respond rapidly to specific environmental changes. The ability of the human body to regulate core temperature varies amongst individuals and in response to a variety of environmental factors. Athletes, the elderly, and the very young are particularly at risk [1]. This short review focuses on the first of these groups: young athletes performing in high environmental temperatures. Excessive core temperature elevations, depletion of electrolytes, water, and sugars, and the rapid, chaotic shifts in electrolyte balance can end in terminal heat stroke if not adequately treated. Application of mechanical cooling aids and repeated ingestion of a solution of electrolytes, sugars, and water whose concentrations approximate known human physiological requirements are the treatments of choice. Ingestion of water alone can increase the risk of cerebral edema and catastrophic collapse.

The most common and life threatening challenges to temperature regulation occur during periods of extreme exercise in high temperature environments. When core temperature of the human body rises, basal metabolic rate increases beyond what would be required for the physical task at hand. A 0.6°C rise in temperature produces a 10% elevation in energy expenditure and depletion of both circulating sugars and replacement carbohydrate stores [2]. The perspiration characterizing exercise in high temperature conditions is the body's attempt to transfer excess core heat by dumping massive quantities of electrolytes and water to the outside environment [3]. During exercise in high heat, sweat output of both water and electrolytes (sodium, chloride, potassium, calcium, and phosphates) can quickly exceed intake [4]. As total water volume falls and electrolyte imbalances appear, removal of the excess heat and an athletes performance of required tasks are both impaired.

It has been clear for more than three decades that core temperature elevation and the accompanying electrolyte, sugar, and water imbalances are a metabolic shift that places young athletes at risk, impairs athletic performance, and produces detectable alterations in blood and urine chemistries. These maladaptive changes can be prevented or treated, but, if unattended, increase the risk of injury and likelihood life-threatening collapse [5-7].

Muscles and nerves (including brain) are particularly at risk in hyperthermia. The electrolyte disturbances produce alterations in sodium, potassium, calcium, and phosphate concentrations impairing the sodium-potassium pump that is crucial to neuronal and muscle function. Normally protecting the brain, the porous bony skull can act during core heat elevations as both a thermal blanket slowing radiant heat loss and as a fixed limit on brain volume. The brain is essentially a cluster of membranes built out of proteins and fats and suspended in massive amounts of water, electrolytes, and sugars. As core temperature rises, molecular agitation, by definition, increases. Solids, liquids, and gases all expand with increasing temperature. Brain tissue is constrained by the fixed volume of the surrounding skull. With increasing temperature there is a resulting increase in intracranial pressure. If the pressure increases excessively there will be a loss of control over water and electrolyte ratios in the intracellular, extracellular, and vascular compartments, and the first signs and symptoms of cerebral edema.

The most common early manifestation of this edema in young adults includes mild to moderate confusion, poor judgment, and diminished impulse control. In athletic events, additional injury can occur as a result of the confusion that accompanies early stage cerebral edema as the afflicted athlete may be slow to recognize danger, forgets previously learned safety techniques, and exhibits alterations in

fundamental reaction times. In young adults, such confusion can be easily demonstrated with dehydration of less than 1-2% of body weight [8]. Unfortunately, the human body is a work in progress. The thirst signals telling us to replace our fluid loss may not be activated until we have lost fluid stores equivalent to ~2% or more of our body weight.

Heat stroke is the most life-threatening aspect of untreated hyperthermia [9]. Heat stroke resulting simply from elevated environmental temperatures will most often affect the very elderly, the very young, and individuals with a variety of contributing metabolic disorders. Heat stroke is characterized by a rise in core temperature to 40°C (104°F) or more, loss of the ability to perspire, and a wide variety of individual neurological responses ranging from confusion and disorientation, to difficulty standing, or even deep coma or seizures, all reflecting a rapidly progressive cerebral edema.[10] The detection of the disorder is often obvious due to the paradox that a neurologically impaired, relatively inactive individual with elevated temperature suddenly cannot perspire in spite of elevated environmental temperature. If treated promptly, recovery is rapid with few if any sequelae. Untreated, the disorder quickly produces multiple system damage ending with seizures, coma, paralysis, or death.

Unfortunately, the heatstroke most commonly appearing in young adults (known as exertional heatstroke) often does not include an absence of sweating. The first series of clues will be entirely neurological since all of the individual's teammates will also be hot, sweaty, and exhausted. Signs include a sudden drop in performance, difficulty concentrating, memory defects, inappropriate behavior (angry after a successful play on the field or happy and unconcerned after a clear performance failure), dizziness, and the appearance of careless, potentially dangerous mistakes. The person afflicted may not be able to make intelligent decisions about his own level of impairment, much like a diver experiencing nitrogen narcosis or a pilot exposed to a high altitude oxygen deficit. Heat stroke is a life threatening medical emergency. The afflicted athlete can collapse suddenly in a grand mal seizure, regurgitate and aspirate stomach contents, or collapse following a sudden drop in blood pressure. Core temperature cooling and restoration of electrolyte, water, and sugar balance are the primary mandatory treatments.

Regardless of the apparent severity of the hyperthermia, the disorder should be treated with great respect and the ultimate goal is constant: prevent the onset of the disorder where possible and avoid the seizures, coma, and death accompanying terminal heat stroke. Two fundamental interventions are required and need to be implemented across the course of athletic competition. Mechanical cooling of core temperature during athletic events is probably best accomplished with cold wet ice packs applied to large surface-accessible arteries and veins in the neck, under the arms and in the groin. The classic application is a cold compress or ice pack to the neck accessing jugular veins and carotid arteries. Even the children's 'ice-cold-bottle-of-water-over-the-head' is not without its uses. Many football fields and soccer fields now use synthetic polymer turf whose surface temperatures have been reported at above 60°C (140°F). In spite of their confusion and debilitation athletes should not lie down on these surfaces since closer proximity to the surface will increase heat infusion into a body already hyperthermic and at risk. For more severe cases, placing the athlete on a tarp on top of ice from a drinks cooler while waiting for paramedics is a reasonable emergency measure.

Mechanical cooling will not alleviate the electrolyte and sugar disturbances that will have already compromised central nervous system and muscle performance. While an individual athlete will most assuredly be depleted in all three components (salts, water, and sugars), individual variation in glucose metabolism, sweat gland function, pre-competition nutritional state, and delay in recognizing symptoms makes it unlikely the examining physician will be able to estimate relative percentages of depletion. The solution originally pioneered by R. Cade is still the gold standard. For a popular accessible account of the development of the electrolyte solution that would later become Gatorade see the article written four years ago by A. Phillips-Han [11].

The fluids bathing all the cells of the human body are essentially minor modifications to the water, sodium, chloride, calcium, magnesium, phosphates, sugars, and other salts that have covered our planet and served as a home for living forms for the past 3.5 billion years. But the minor modifications are

critical to our functioning as a complex multicellular organism. If we lose some of those fluids and try to replace them with, for example, only water, then if the original imbalance actually includes a relative increase in loss of electrolytes and sugars compared to the loss of water, we have just made things worse by tipping the balance further away from equilibrium. If we administer a sugar-rich soda, and the primary deficits were with electrolytes and total water volume while sugars were relatively less depleted, our treatment attempt has again tipped the balance away from equilibrium. Essentially, if we administer any one of the triad alone, we have a two out of three chance to make a potentially life threatening condition worse.

These are far from being esoteric medical concerns. Imagine we knew in advance that the total water content for a young athlete was more depleted than his electrolytes and sugars. It might seem logical that we would try to give him extra water. But the brain would have already made some alterations we need to consider. As the young man lost water, his blood and the fluids bathing the outside of the neurons in his brain would now contain a higher concentration of sodium than is normal. Brain neurons, even though much richer in potassium than in sodium would have raised their sodium concentration in order to maintain a needed equilibrium with the fluid outside the cell. If we suddenly add extra water to the fluid bathing the cells, the first response will be a flood of water molecules into brain cells to move sodium ratios back to equilibrium [1]. Our treatment will have caused cerebral edema. The exact phenomena we wish to avoid.

The correct response to the dilemma is to treat with a solution that contains approximately the human-specific ratios of water, salts, and sugars. That insight led to the development of a water-electrolyte-sugar solution tested on the Florida Gator football team beginning in 1966 and demonstration that injuries of all types (including heat exhaustion and heat stroke) could be decreased with continuous balanced replacement therapy during competition. Subsequent work investigating fluid replacement with mixtures of electrolytes, sugars, and water has demonstrated significant performance improvements for athletes in a wide variety of sports when compared to athletes using only water for fluid replacement. Of the many insights gained over the past three decades, three are most intriguing and concerning. First, untreated elevations in core temperature significantly reduce maximal performance limits [12]. Second, core temperature increases and fluid losses are greater in marathon runners [13] and tennis players [14] training in high temperature when athletes replace fluid losses with water rather than an electrolyte-glucose-water solution during competition. Third, elevations in enzyme markers for muscle damage (lactic dehydrogenase and creatine kinase) in swimmers indicate increased muscle damage during high-intensity training when fluid replacement was accomplished using only water instead of an electrolyte-glucose-water solution [15]. The implications of these data are quite clear. Not only does rapid replacement of electrolytes, sugar, and water prevent heat stroke, but it also improves maximal performance. For once we have a single, easily implemented strategy for preventing a potentially life-threatening event and simultaneously optimizing an athlete's performance.

Finally, to put all of this in perspective for school athletic programs, it is important to understand how close to 'heat stroke territory' athletes come during routine competition. Temperature elevations up to 41°C (105.8°F) have been recorded repeatedly from marathon runners performing in an environmental temperature of only 25°C (77°F). This above the elevation characteristic of classical heat stroke. Our young athletes, as we always knew, are continually pushing the boundaries. We need to increase the odds that they will survive their efforts.

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Gators football team to his first attempts to introduce a mixture of electrolytes, sugars, and water that would later become Gatorade. The overwhelming memory is of an incredibly descent, energetic, and creative physician who loved sports and science. He recognized from the start of his efforts that a simple shift in how we prevent and treat expected water, electrolyte, and sugar losses during athletic competition could not only eliminate countless injuries and deaths, but enhance performance. It is unclear at this point how many lives have been affected by his early insights, but I suspect we owe Professor Cade more than most of us can imagine. I certainly do.

References

1. Wexler, R.K. 2002. *Evaluation and treatment of heat-related illnesses*. American Family Physician, **65**(11): p. 2307-2314.
2. Cecil, R.L., Wyngaarden, J.B., Smith, L.H., and Bennett, J.C. 1992. *Cecil Textbook of Medicine 19th ed.*, pp 2358-61, Philadelphia: Saunders.
3. Maughan, R. and Shirreffs, S. 2004. *Exercise in the heat: challenges and opportunities*. J. Sports Sci., **22**(10): p. 917-927.
4. Sawka, M.N. and Montain, S.J. 2000. *Fluid and electrolyte supplementation for exercise heat stress*. Am. J. Clin. Nutr. **72**(suppl), **72**(supp): p. 564S-572S.
5. Millard, M.L., Zauner, C.W., and Cade, J.R. 1981. *Effects of intense training on blood parameters in national class collegiate swimmers*. Med. and Sci. in Sports and Exercise, **13**(2): p. 110.
6. Burch, G.E., Cade, J.R., and Tintinalli, J. 1974. *When patient's problem is heat*. Patient Care, **8**(11): p. 42-43.
7. Vaccaro, P., Zauner, C.W., and Cade, J.R. 1975. *Changes in body weight, hematocrit and plasma protein concentration due to dehydration and rehydration in wrestlers*. Med. and Sci. in Sports and Exercise, (1): p. 76.
8. D'Anci, K.E., Constant, F., and Rosenberg, I.H. 2006. *Hydration and cognitive function in children*. Nutri. Rev., **64**(10): p. 457-464.
9. Amore, M. and Cerisoli, M. 1992. *Heatstroke and hyperthermia*. Ital. J. Neurological Sci., **13**(4): p. 337-341.
10. Yaqub, B. and Al Deeb, S. 1998. *Heat strokes: aetiopathogenesis, neurological characteristics, treatment and outcome*. J. Neurolog. Sci., **156**(2): p. 144-151.
11. Phillips-Han, A. 2003. *Dr. Robert Cade...saga of the world's best-selling sports drink and the creative physician scientist behind it*. <http://www.news.health.ufl.edu/story.aspx?ID=703>.
12. Craig, E.N. and Cummings, E.G. 1966. *Dehydration and muscular work*. J. Appl. Physiol., **21**: p. 670-674.
13. Cade, J.R., Packer, D., Zauner, C.W., Kaufmann, D., Peterson, J., Mars, D., Privette, M., Hommen, N., Fregey, M.J., and Rogers, J. 1992. *Marathon running- physiological and chemical changes accompanying late race functional deterioration*. Eur. J. Appl. and Occup. Physiol., **65**(6): p. 485-491.
14. Bergeron, M.F., Waller, J.L., and Marinik, E.L. 2006. *Voluntary fluid intake and core temperature responses in adolescent tennis players: sports beverage versus water*. Brit. J. Sports Med., **40**(5): p. 406-410.
15. Cade, J.R., Reese, R.H., Privette, R.M., Hommen, N.M., Rogers, J.L., and Fregey, M.J. 1991. *Dietary intervention and training in swimmers*. Eur. J. Appl. and Occup. Physiol., **63**(3-4): p. 210-215.