Collapse and Neurological Dysfunction in a Young Athlete

Ronald N. Rubin, MD[1,2]—*Series Editor*

An 18-year-old boy has collapsed during a football practice. He is a high-school varsity player in a Southern city, and he has been attending the late summer (last 2 weeks in August) obligatory practices for varsity players.

He is in outstanding health, with a medical history positive only for appendectomy at age 10. He takes no medicines, and he had felt completely well this morning. This is the second week of practice, and he has had no difficulties until now. The morning practice was 2 hours in full pads, and he appeared to have no problems at all. After a 45-minute break for fluids and fruit if desired, the coaches had stated that better athletes (the starters) must return for another session, since the temperature was only 87 °F, and they wanted these boys to practice more so as to be "more ready for the season opener" and "just in case some college recruiters might be looking on."

Approximately 40 minutes into the afternoon session, the boy had slowed down markedly compared with his teammates. He took fluids, but his teammates noted that he was confused about where he was and what he was supposed to do. No head trauma was noted. The trainer took the boy to the locker room, where a spot neurological evaluation showed no focal findings, but the agitation and confusion continued. His pulse rate was 116 beats/min, his respiratory rate was 22 breaths/min, and his skin was wet and clammy. His oral temperature was 103.6 °F (39.8 °C), and the trainer sent for an ambulance to take the boy to an emergency department.

Which one of the following is an *incorrect* statement regarding the clinical entity being manifested by this patient?

A. A patient can experience this syndrome without exposure to excessive environmental heat stress.

B. Important corroborative clues to the diagnosis include wet rather than dry skin and abnormal results of a battery of biochemical tests (eg, potassium level, calcium level, liver function, kidney function)

C. Mortality in a population experiencing the described entity can exceed 50%.

D. There is probable association with enhanced risk for malignant hyperthermia.

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Classification schemes separate heat stroke into classic and exertional subtypes. The patient described is manifesting the exertional type in contrast to the classic form.

TAKE-HOME MESSAGE

Heat stroke is a life-threatening syndrome that occurs when an imbalance between heat exposure (endogenous of exogenous) and heat dissipation capacity of the body occurs, to the favor of heat accumulation in the body with a core temperature of 40.5 °C being a landmark for neurological dysfunction. Two distinct clinical variants exist—classic and exertional—with quite distinct epidemiology. Both manifest the triad of hyperthermia, neurological dysfunction, and appropriate exposure to heat-generation risk. Therapy involves rapid lowering of core temperature using cold-water immersion for the exertional type and conductive/evaporative methods for the classic type. Mortality rates for classic heat stroke exceed 50% due to the nature of the patients and the frequent delay in medical care. Mortality rates for exertional heat stroke are less than 5%, since the condition usually is observed, and thus immediate therapy can be put in place.

Answer: The *incorrect* statement is C, mortality in a population experiencing the described entity can exceed 50%.

The patient described is manifesting heat stroke, more specifically the exertional type in contrast to the so-called classic form. Both share the core pathophysiology of mismatch or imbalance between heat generation and/or absorption by the body vs the ability of the body to physiologically negate this with heat-loss mechanisms. The result in both cases is pathological overheating of the body core to and above the very critical level of 40.5 °C.

Current classification schemes separate heat stroke into classic and exertional subtypes. Classic heat stroke is the type most typically seen in elderly patients with a variety of serious comorbid conditions (and thus who takes many medications, which will be discussed below). The comorbidities and medications, as well as the sometimes difficult social conditions, render these people to have inadequate endogenous/physiological mechanisms as well as exogenous/environmental mechanisms for dissipation of metabolically and environmentally produced body heat. Frequently, during a prolonged heat wave, these cases cluster into "epidemics," with multiple cases encountered after 3 days or more of such a heat wave.[1] Such exogenous heat is the trigger for heat stroke development in these cases. More specifically, such well-defined historical facts will include cardiovascular insufficiency, which impairs cardiovascular adjustments to heat stress; medications such as β-blockers, anticholinergics, and especially monoamine oxidase inhibitors, selective serotonin-reuptake inhibitors, and tricyclic antidepressants; comorbid neurological disability that inhibits movements; and, very critically, social isolation and living conditions such as lack of ventilation, fans, and air conditioners.[1]

Conversely, exertional heat stroke does *not* require exogenous heat to occur, which makes **Answer A** a correct statement. The heat in these cases is endogenously created by very vigorous and intense physical activity that creates more heat than even a young, fit person with normal physiology can dissipate. These are the cases of military training deaths related to gung-ho drill sergeants and high-school athlete deaths related to overzealous coaches, such as the situation described in the case above. The symptoms and signs appear very acutely—from normal to life-threatening illness in a matter of hours rather than days (as with classic heat stroke). But in exertional heat stroke, the precipitating situation is usually quickly observed such that immediate treatment is the norm, and the mortality rate is less than 5%, compared with the often unobserved patients with classic heat stroke, the mortality rate of whom exceeds 50%,[1,2] which makes **Answer C** the *incorrect* statement here.

The diagnosis is essentially clinical and is made when the presence of the triad of hyperthermia (body temperature of 40.5 °C or above), neurological dysfunction (which can range from dizziness, confusion, weakness, and slurred speech into delirium), and recent exposure to hot weather (classic heat stroke) or violent, severe physical exertion (exertional heat stroke). Most patients will manifest tachycardia and hypotension. Interesting differences between the classic and exertional forms include dry skin in the former with absence of sweating, compared with wet skin and the presence of sweating in the latter; minimal abnormal metabolic test results (eg, creatine kinase, creatinine, calcium, and potassium levels) in classic heat stroke compared with frequent and significant abnormalities in such test results in the exertional heat stroke,[1,2] as was later determined in the presented patient, which makes **Answer B** a true statement.

The goal of therapy is simple: Lower the core body temperature as quickly as possible. The faster the cooling, the better the prognosis for neurological recovery and overall mortality.[1-3] Reductions of 0.20 to 0.35 °C/hour documented by rectal temperature monitoring are cited in the literature.[1,3] Again, differences in typical standard of care for classic vs exertional forms exist in therapy methods. Persons with classic heat stroke (usually an elderly, infirm patient population) are less tolerant

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of cold immersion such that conductive or evaporative techniques—infusion of cold intravenous solutions, ice or cold packs, cold wet sheets, and fans are literature-cited treatments of choice.[1]

The generally younger and more physically fit exertional heat stroke population can be treated with cold-water immersion, along with the conductive and evaporative maneuvers used as adjuncts.[1,3]

Answer D relates to certain interesting observations linking heat stroke risk to history of and risk for malignant hyperthermia, and the presence of increased exercise-related deaths without exertional heat stroke per se in patients with sickle cell trait.[4] However, to date, no specific genetic abnormalities disposing to heat stroke have been documented. And no pharmacologic therapy of any kind has been shown to be helpful. Early basic science research into both the genetics of heat stroke and the potential for pharmacologic intervention are in progress.[1]

PATIENT FOLLOW-UP

The acute development of hypotension, tachycardia, and neurological signs and symptoms, with documentation of a core temperature approaching 40.5 °C with association of hyperintense physical exertion was quickly recognized as heat stroke of the exertional form. The patient was quickly placed in cold-water immersion. Within 5 minutes, his core temperature had dropped to 38.5 °C, and his mentation started to clear. Intravenous fluids were administered (isotonic saline), and he was taken to a nearby emergency department, where he was admitted. Laboratory tests performed at admission revealed elevated levels of creatine kinase, creatinine, and potassium, which started to correct the next day and which normalized, along with all neurological findings, by day 3, allowing for discharge. ■

REFERENCES:

1. Epstein Y, Yanovich R. Heatstroke. *N Engl J Med*. 2019;380(25):2449-2459. doi:10.1056/NEJMra1810762
2. Bouchama A, Dehbi M, Mohamed G, Matthies F, Shoukri M, Menne B. Prognostic factors in heat wave related deaths: a meta-analysis. *Arch Intern Med*. 2007;167(20):2170-2176. doi:10.1001/archinte.167.20.ira70009
3. Heled Y, Rav-Acha M, Shani Y, Epstein Y, Moran DS. The "golden hour" for heatstroke treatment. *Mil Med*. 2004;169(3):184-186. doi:10.7205/milmed.169.3.184
4. Nelson DA, Deuster PA, O'Connor FG, Kurina LM. Sickle cell trait and heat injury among US Army soldiers. *Am J Epidemiol*. 2018;187(3):523-528. doi:10.1093/aje/kwx285