A Case of Heat Stroke in the Era of Climate Change

FRED VARONE, MD; WILLIAM BINDER, MD

From the Case Records of the Alpert Medical School Residency in Emergency Medicine

DR. FRED VARONE: Today's patient is an elderly male of unknown age (later found out to be 68 years old) who was brought into the Emergency Department (ED) by Emergency Medical Services (EMS) after being found unresponsive in a park. The patient was fully dressed and wearing a coat, lying face down, despite outside temperatures hovering at 99°F and > 90% humidity.

Initial vital signs were notable for a systolic blood pressure of 70 mm Hg, pulse 130s, and a rectal temperature of 43.3°C (110°F). The patient was noted to be unresponsive to painful stimuli and his skin was dry and warm to touch. Pupils were 3 millimeters bilaterally and sluggishly reactive. His cardiac exam revealed tachycardia with a regular rhythm. Breath sounds were clear although he was noted to have sonorous respirations.

The patient was given a bolus of crystalloid IV fluids and push dose phenylephrine for hypotension. He was intubated for airway protection via rapid sequence intubation using ketamine and rocuronium.

DR. AMY MATSON: What laboratory and imaging studies were obtained?

DR. VARONE: Notable labs are seen in **Table 1.** The patient's laboratory abnormalities are not unusual in heat stroke. Rhabdomyolysis and injury to the kidneys and liver are often noted by elevations in the creatinine phosphokinase (CPK), creatinine and liver function tests. Thrombocytopenia is also common. Troponin is frequently elevated as well, reflecting a type 2 myocardial infarction.[1,2,3,4,5]

Table 1. Heat Stroke Criteria

Lab	Initial presentation	Day 1 of hospitalization	Day 3 of hospitalization
Hemoglobin/Hematocrit	10.9/33	11.8/34	
Platelets	64 x 10^9/L	21 x 10^9/L	74 x 10^9/L
Creatinine	1.80 mg/dl	2.51 mg/dl	5.43 mg/dl
Troponin I	0.166 ng/ml	68.194 ng/ml	65.271 ng/ml
AST/ALT	125/36 IU/L	4,060/354 IU/L	949/302 IU/L
CPK	243 IU/L	6,638 IU/L	4,555 IU/L
Lactate	9.3 mEQ/L	4.6 mEQ/L	

There are no specific findings on radiographic imaging suggestive of heat stroke. When acute lung injury and ARDS are suspected, chest X-ray will show the typical bilateral infiltrates. In our patient, a chest X-ray after intubation showed the endotracheal tube in good position and no acute cardiopulmonary abnormalities. CT imaging of the brain and cervical spine without IV contrast was unremarkable for acute changes. CT imaging of the abdomen and pelvis with intravenous contrast revealed diffuse thickening of bowel loops with enhancing walls concerning for bowel ischemia, as well as bibasilar airspace disease.

DR. JOSEPH LAURO: With a temperature of 110°F, this patient has heat stroke. Is it unusual for a temperature to rise so high without exertion? Can you review the different types of heat stroke?

DR. VARONE: Heat-related illness exists on a spectrum of disease severity, with heat stroke being the most severe manifestation. Other classifications for heat-related illnesses include heat stress, heat injury, heat exhaustion, heat syncope, and heat cramps. The loss of fluid and salt in sweat can lead to muscle cramping and intravascular volume depletion leading to syncope. Heat exhaustion can be considered the precursor to heat stroke, with symptoms such as headache, dizziness, nausea, weakness, and a temperature that is typically between 36 and 40°C. While these are all readily reversible clinical conditions with cooling and hydration, they can progress into organ failure if untreated, leading to the syndrome of heat stroke.

Our patient has heat stroke, classically defined as high environmental heat exposure combined with CNS dysfunction and elevated core body temperature > 40°C (104°F).[6] Additional definitions have been proposed which do not include temperature criteria (See **Table 2**).[7]

Heat stroke can be divided into classic heat stroke (CHS) and exertional heat stroke (EHS). CHS occurs more often in vulnerable populations, such as the elderly, as their ability to compensate is often impaired by comorbidities and medications. EHS, on the other hand, occurs in

Table 2. Comparison of different criteria for diagnosis of heat stroke

All definitions	• High Environmental Heat Exposure
Bouchama	• Core temperature > 40°C • CNS effects (coma, delirium, seizures)
JAAM	• CNS symptoms (Japan Coma Scale score of ≥ 2, cerebellar symptoms, convulsions seizures) • Hepatic or renal dysfunction • DIC by JAAM criteria
JAAM-HS-WG	• GCS ≤ 14 • JAAM DIC score ≥ 4 • Creatinine or bilirubin sym 1.2 mg/dl

Glasgow Coma Scale, Japanese Association of Acute Medicine (JAAM), Japanese Association of Acute Medicine heat stroke committee working group (JAAM-HS-WG)

young, healthy individuals during strenuous physical activities (athletes, military personnel).[7,8] Temperatures can rise substantially in both forms of heat stroke.

In patients with heat-related illness, heat loss is mediated by several different mechanisms. These include radiation, conduction, convection, and evaporation. In cool temperatures, the majority of body heat (55%–65%) is lost by radiation, or the emission of energy from the skin. Convection occurs through the transfer of heat from the body to a gas (air), while conduction occurs by the transfer of heat between two surfaces with different temperatures. In warm temperatures, evaporation is the primary means to dissipate heat, but it has limited efficacy when humidity rises above 60%.[1] Unfortunately, our patient was unable to dissipate heat due to his clothing and the high heat index (a combination of air temperature and relative humidity).

DR. ELIZABETH SUTTON: This patient's temperature reached 110°F. Is that usually survivable? What happens to the patient's organs?

DR. VARONE: While organ systems may start to exhibit dysfunction at 40°C (104°F), higher temperatures are survivable, and patients may have temperatures up to 43.3°C (109.9°F) and survive with good neurologic outcome.[9] Some studies suggest that time to cooling may be more important than maximum temperature.[3,10,11]

The cause of an elevated temperature is important. Fever is created when pyrogens (such as bacterial endotoxins or cytokines produced from cell damage) act on the hypothalamus to increase the natural temperature set point through increased prostaglandin production.[7]

In heat stroke, however, temperatures rise without an adjustment in the hypothalamic set point. In heat injury, cell membrane physiology is disrupted, leading to endothelial and organ cell death.[2,12,13,] Proteins start to denature around 40°C, leading to enzyme disruption, mitochondrial failure, and loss of oxidative phosphorylation. In response to injury, cells produce heat-shock proteins, which are protective against heat, hypoxia, and ischemia, but these quickly can become overwhelmed in severe heat stroke.[1] The end result is cell death across multiple systems.[14]

The impact of hyperthermia on organ systems is significant. In the CNS, thermal injury can lead to cellular denaturing, neuronal death, and excitotoxicity. Cytokine release and resultant vascular damage, as well as local ischemia and systemic changes in blood flow can result in cognitive impairment, seizures, and cerebral injury.[15]

Gastrointestinal tract physiology is altered by hyperthermia and contributes to mortality. To dissipate heat, blood flow shunts from the GI tract to the skin, resulting in bowel ischemia, mucosal barrier disruption, and bacterial translocation. Bacteremia, circulating endotoxin and sepsis can ensue.[1,3,13] Additional inflammatory mediators, including IL-1 and IL-6, are released from injured cells, causing a systemic inflammatory syndrome response (SIRS).[1,7] Vascular endothelial cells also sustain damage, which can lead to a consumptive coagulopathy and systemic microthrombi.[2,13]

Other organ systems are impacted. Myocardial infarction has been reported in up to 7% of admitted heat stroke patients.[4] Multi-organ dysfunction and systemic manifestations including disseminated intravascular coagulation, rhabdomyolysis, hepatic necrosis, and acute kidney injury have all been reported.[5,16]

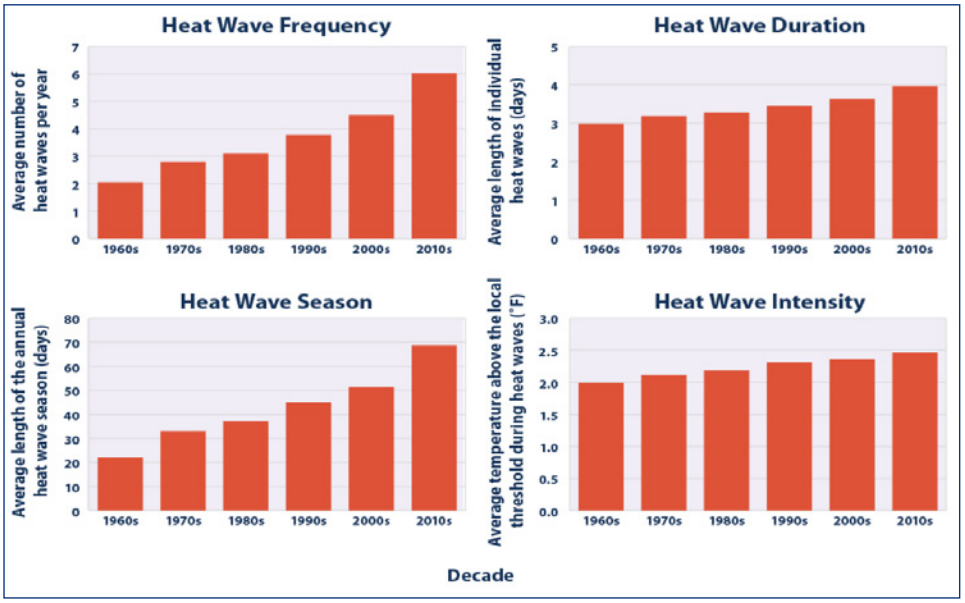
DR. OTIS WARREN: Are heat-related illness occurring with greater frequency?

DR. WILLIAM BINDER: There has been an increase in the frequency of extreme heat events (EHEs) over the last several decades. Data analyzed between 1961 and 2019 for 50 metropolitan areas in the US shows an increase in heat wave frequency, duration, intensity, and an increase in the length of the heat wave season (see **Figure 1**).[17]

Heat related mortality has risen concordantly. While it is reported that approximately 700 individuals die annually in the US due to heat, it is felt that this number is grossly underestimated as comorbid diseases are often reported as a primary cause of death, thereby concealing environmental heat's role.[1,18] The recent Lancet countdown on health and climate change suggests that in 2018, 19,000 deaths in the US were related to high environmental temperatures.[19] Underscoring this, our patient's problem list and discharge summary in the electronic health record acknowledges NSTEMI, encephalopathy, and acute kidney injury, but makes no mention of heat stroke.

While data on heat-related mortality in Rhode Island is not available, average temperatures have risen over the past several decades. Between 1981–2010 there were an average of 9 days ≥ 90°F in the Providence Metro area; this number increased between 2010 to 2014, and in 2021 there were 19 days ≥ 90°F. Rising summer daily temperatures have been linked to increased EMS utilization and ED visits.[20,21]

Figure 1. Heat-wave frequency, duration, intensity, and length of season between 1961–2019 for 50 metropolitan areas in the US.



DR. VARONE: Anthropogenic climate change from increasing urbanization has resulted in heat islands, areas where the average temperature is higher than surrounding areas. Developed cities tend to have higher day and nighttime temperatures due to increased heat production, replacement of vegetation by concrete and other manmade materials, and disruption of natural airflow by large buildings, streets, and city geometry.[22] Higher evening temperatures prevent humans from recovering from daytime heat exposure and injury. Analysis of the 2003 Paris heat wave shows a clear association between increased nighttime surface temperature in heat islands and risk of death, particularly in elderly people.[22]

While cities only make up around 2% of the Earth's surface, they are responsible for 75% of the world's emissions.[23] In 2007 there were 19 cities with 10 million or more inhabitants; by 2018 there were 33 "mega-cities." Increasing urbanization will lead to more heat islands and more heat-related illness, particularly in developing countries where populations and urbanization are still accelerating.[23,24]

DR. CATHERINE CUMMINGS: What was your management of this patient with hyperthermia?

DR. VARONE: Rectal temperatures should be checked in patients with suspected heat stroke. External measurements have been shown to correlate poorly with rectal temperature and underestimate the degree of hyperthermia.[8,11] In one study of collapsed marathon runners, only two of 17 collapsed runners with true hyperthermia (rectal temperature ≥ 39.4°C) were identified correctly by temporal measurement.[25]

Rapid cooling is the mainstay of treatment for heat stroke and improved outcomes are related to early recognition and treatment.[6,8,10,11,12] Patients should be cooled at a rate of 0.1–0.2°C per minute until evidence of clinical improvement, with careful attention paid to preventing overcooling.[6,26] While there is no specific temperature goal and the patient's clinical picture should guide cooling, some studies have used specific temperature cutoffs to prevent overcooling (38–39°C).[6,10] Techniques used for cooling in the ED include cold packs and ice (conduction), and evaporative cooling using cold water and fans. Cold-water immersion, frequently used in the pre-hospital setting for patients with EHS, is often impractical in the ED and potentially harmful in older patients with CHS requiring monitoring. Invasive methods for cooling include bladder, gastric and peritoneal lavage, and cardiopulmonary bypass methods such as ECMO-based cooling.[26]

Our patient received intravenous fluids, cold packs and ice, and evaporative cooling with fans. His temperature decreased to 39°C within 45 minutes of arrival to the ED.

Of note, antipyretics have no utility in hyperthermia due to environmental exposure, and some studies suggest they may be harmful in heat-related injury.[26]

DR. NICK MUSISCA: What happened to this patient?

DR. VARONE: After intubation, orogastric and rectal tubes were placed. His ED course was complicated by runs of atrial fibrillation with rapid ventricular response, and cardioversion was attempted but was unsuccessful. His rate decreased with fluid administration and temperature control. He received benzodiazepines, to prevent shivering and agitation, piperacillin-tazobactam for ischemic bowel, and anti-epileptic medications (AEDs).

The patient was admitted to the medical intensive care unit and multiple medical services were consulted. Continuous electroencephalography was performed for 24 hours, and the patient did not demonstrate seizure activity. On hospital day 4 he was extubated, although he was confused and encephalopathic. On hospital day 10 he was able to provide health care workers his name, and social work was able to locate his daughter. She reported a history of chronic alcohol abuse and dementia - known risk factors for heat stroke. The patient improved to his baseline and was discharged to a skilled nursing facility on hospital day 19.

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Authors

Fred Varone, MD, PGY-3, Brown Emergency Medicine Residency.

William Binder, MD, Associate Professor of Emergency Medicine, Warren Alpert Medical School of Brown University.

Correspondence

william_binder@brown.edu