**Stay cool, fool.**

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**The Evaluation and Management of Heat Injuries in the Emergency Department**

Younggren BN, Yao C. EM Practice. ebmedicine.net. June 2006, Vol 8(6).

There are a few different kinds of heat injury, the most severe, and potentially fatal, being heat stroke. Temperatures over 41-42C (about 106-107F), cause cellular dysfunction. Temperatures over 49C (120F) cause immediate cell death. Hyperthermia-induced apoptosis occurs mostly in thymus, spleen, lymph nodes, and small intestine mucosa, as well as massive release of inflammatory cytokines and increased vascular permeability leading to subsequent development of DIC and multiorgan failure. Everything up to this level is not a big deal.

**Head Edema:** benign condition, thought to be 2/2 peripheral vasodilation and increased vascular permeability/leak. Dependent and usually mild, resolves with time/acclimatization. Treatment includes rest, elevation of extremities and staying cool. No role of diuretics, can actually worsen dehydration. Prevention – stay cool, fool.

**Heat Tetany:** carpopedal spasm 2/2 hyperventilation and subsequent hypocarbia. Treatment includes breathing into a paper bag. Prevention – stay cool, fool.

**Heat Syncope:**  orthostatic hypotensive event 2/2 peripheral vasodilation (and decreased venous return), often seen after prolonged standing or with quick upright postural change. Treatment includes oral re-hydration in a cool area. Prevention includes adequate hydration, avoid prolonged standing in the heat, and periodic flexing of lower extremities to increase venous return.

**Miliaria rubra, or “Prickly Heat”:**  pruritic papulovasicular rash that develops over clothing covered areas when exposed to heat. Occluded sweat glands deep in the dermis dilate and rupture, causing intensely pruritic vesicular lesions.

May progress to *Miliaria profunda* – duct obstruction is evident at dermoepidermal junction. Areas become anhydrotic and are often superinfected with staph. Treatment includes drying and cooling, chlorhexidine cream or salicylic acid (1%). Prevention – stay cool, fool.

**Heat Cramps:** Painful spasms of large voluntary muscle groups (usu. thighs, calves, abdominal wall and shoulders) after prolonged exertion, 2/2 dilutional hyponatremia. \*\*NO systemic symptoms\*\*

Non-acclimatized people have increased electrolyte concentration in sweat. Elevated temperature is **not** a requirement, and it may develop hours after exertion. Treatment includes oral rehydration with electrolyte solution, or IV NS, and rest.

**Heat Exhaustion:** Most common form of heat related illness. Temp elevated, but *<104F*, and symptoms are *systemic*. Malaise, profuse sweating, fatigue, headache, nausea, possibly vomiting. Tachycardic, some postural hypotension. \*\*Mental status INTACT.

**Water Depletion**: 2/2 inadequate rehydration, rapid, seen in day laborors, military, or athletes. Treatment includes rest, cooling and aggressive oral rehydration.

**Salt Depletion**: 2/2 overhydration with hypotonic solution. Dilutional hyponatremia/hypochloremia just like heat cramps, but producing *systemic* symptoms.

\*\*Can cause *altered mental status* or *seizures*, but if temp <104, can’t call it “heat stroke”.

Treatment includes aggressive electrolyte replacement (instead of rapid cooling). 🡪 this is the SAME as heat stroke, except core temp <104 = BADNESS. TREAT AGGRESSIVELY.

Significant free water deficits should not be corrected faster than 2mOsm/hr (or you’ll cause cerebral edema = frowned upon).

***HEAT STROKE***

**(Simply put) Temp >105 + altered mental status.**

But there are 2 real definitions.

First, by criteria: (1) temp>105, (2) CNS dysfunction, (3) exposure to heat stress and (4) absence of CNS infection, NMS, malig hyperthermia, or sepsis (some sources include (5) increased AST/ALT).

Second, by pathophysiology: hyperthermia assoc with systemic inflammatory response, leading to multiorgan dysfunction in which encephalopathy predominates.

High inpatient mortality (numbers vary wildly from 20% to 85%), complicated most commonly by ARDS, DIC, rhabdomyolysis, renal failure, cerebral edema, seizures, shock, hepatic dysfunction.

BAD SIGNS: metabolic acidosis (even if compensated by resp alk), CK>10,000, and elevated LFTs.

🡪 BE AGGRESSIVE.

**Epidemic, or “Classic” Heat Stroke**

2/2 environmental/external heat insult. Usually infants, debilitated and elderly. Develops over days, usually present anhidrotic (worsening their condition) and hyperventilating (respiratory alkalosis with metabolic acidosis). Risk factors in table below.

**Exertional Heat Stroke**

2/2 internal heat insult. Usually young, previously healthy, unacclimatized, and exercising in hot, humid env’t. Usually present still sweating, tachycardic, and hypotensive. More commonly develop DIC, ARF, lactic acidosis, hypokalemia, and rhabdo.

**Wilderness Management:** for most heat related illness, rest, cooling, and oral rehydration are key. If you have any suspicion for heat stroke (or salt-depleted heat exhaustion), EVACUATE, giving supportive therapy in route to nearest hospital. Don’t mess around, this can be a fatal condition.

**[just for fun, if you saw this pt in your ED…..**

**ED Management:**

These patients look like sepsis. BE AGGRESSIVE!! If they develop multiorgan failure, they will need aggressive medical resuscitation (ICU care: central lines with CVP monitoring, mechanical ventilation, hemodialysis, pressure support, frequent neuro checks, foley – strict I’s&O’s, etc). Don’t hesitate putting a central line in these folks.

LP?? If unable to obtain good history that is consistent with clear heat injury, CNS infection must be excluded.

Dantrolene? No, no. just stop. **SUPPORTIVE THERAPY.** They *can* get better if you adequately protect them from their own systemic inflammatory response. Cool them down to <39C (102.2F): ice bath immersion = fastest; tepid water and fans (evaporation/convection); ice packs in axillae and groin.

Invasive cooling methods; not well studied. Some case studies seem to have done well, but not recommended at this time. **]**