**Heat Stroke**


Mechem, C. “Severe hyperthermia: heat stroke, malignant hyperthermia, neuroleptic malignant syndrome.” *UpToDate v11.2.*

**Key Points:**
- **Heat stroke involves hyperthermia and CNS changes, with a risk of multi-organ failure**
- **Cause:** failure of internal / external heat-dissipating mechanisms (environment, drugs, disease)
- **Treatment is supportive**

**Definitions:**
- Hyperthermia: elevated core temperature from impaired heat-dissipating (external / internal)
- Heat stroke: T>40C + CNS abnormalities (delirium, seizures especially during cooling, coma)
  - Classic – due to environment
  - Exertional – due to exercise
- Heat exhaustion: water / salt depletion with thirst, weakness, (pre-)syncope, headache, with any core temperature
- Heat stress: discomfort / physiologic strain
- Heat wave: T >32.2C for 3+ days
- DDx: infection, NMS, malignant hyperthermia, dystonic reaction, status epilepticus, CNS event, thyroid storm, drug toxicity (lithium, anticholinergics, sympathomimetic, salicylate), withdrawal

**Physiology:**
- Hypothalamic and peripheral heat receptors
  - Reset by thalamic disease
- Sympathetic cutaneous vasodilation and thermal sweating
  - Anticholinergic
- Thermal gradient between body and environment
  - Increased ambient temperature / humidity >75%
- Secondary: tachycardia, increased cardiac output, increased minute ventilation
  - Volume depletion (from dehydration or diuretics) as a cause as well as consequence
- Acute-phase response: interleukin-1 and –6, cell adhesion, proliferation, angiogenesis
- Heat-shock proteins: allows cells to survive (prevent denaturation)
  - Decreased expression due to aging, lack of acclimatization to heat, genetics

**Pathophysiology:**
- Heat -> denatured proteins / cytotoxicity
- Decreased splanchnic blood flow -> endotoxin release and hemodynamic instability
- Increased release of reactive oxygen / nitrogen
- Activation of leukocytes, endothelial cells, cell adhesion
- Activation of coagulation (decreased protein C, S, antithrombin III) and inhibition of fibrinolysis
- End-organ damage:
  - Encephalopathy – residual CNS deficits in 20%
  - Rhabdomyolysis and acute renal failure
  - ARDS
  - Myocardial injury
  - Hepatocellular / pancreatic injury and intestinal ischemia
  - DIC
- Mortality up to 20%: infection, multi-organ failure; CNS recovery during cooling is prognostic

**Rx:**
- Cooling without triggering cutaneous vasoconstriction / shivering (massage / lavage)
- Benzodiazepines for seizures / shivering
- Dantrolene ineffective in RCT; aspirin / acetaminophen ineffective
- Supportive care
- Prevention