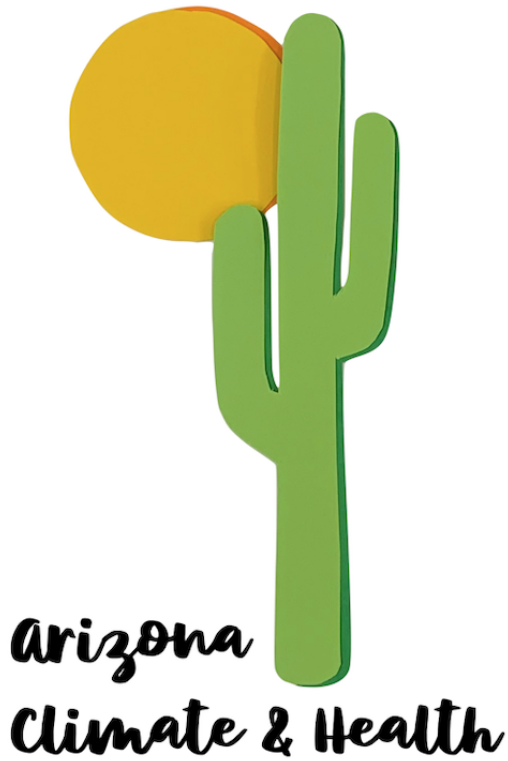

Faculty Handbook

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HEAT RELATED ILLNESS

FACULTY HANDBOOK

Introduction: With the change in Step 1 Exams from numeric scores to a pass / fail report, and with the need to encourage critical application of basic science knowledge into the clinical venue, there is an increasing demand that medical students demonstrate their skill at explaining medical phenomena using pathophysiology and scientific underpinnings. These questions and answers should serve to help develop these skills in the clerkship student and will help familiarize the attendings/preceptors with applying this educational directive in the clinical settings.

1. **How does the body detect and compensate for heat gain[1–4]?**

- The preoptic nucleus of the anterior hypothalamus senses core temperature changes above the set point from both peripheral thermoreceptors in the skin and from central thermoreceptors located in the hypothalamus, spinal cord, viscera and great veins. It appears the central thermoreceptors play a more important role than receptors in the periphery in the determination of core temperature. Signals sent from the presynaptic neurons in the spinal cord to the periphery via sympathetic pathways result in peripheral vasodilation, shunting of blood from splanchnic beds to skin, and increased sweat production predominantly in eccrine sweat glands
- Blood flow to the skin can increase from 250 ml/min to 6-8 L/min—up to 16x basal level!

➢ **Faculty Follow Up Discussion: What part of the arterio-venous system do you think would show the most vasodilation and why/how would this enhance cooling?**
Because we want to encourage the blood to collect at the skin surface, the pre-venous vessels (arteriolar and arteriovenous anastomotic areas) would preferentially dilate to maximize the amount of fluid held in the superficial vasculature. This not only helps increase the conduction or convection heat transfer, but also may initially allow the sweat glands access to more blood from which to produce larger volumes of perspiration. The variation in flow is tremendous, which suggests that this is an important adaptive mechanism. Why is the determination of internal temperature so much more reliant upon central receptors than upon peripheral receptors? Sports researchers have noted that the temperature recorded at the skin can vary significantly from that detected via core sensors. The core temperature reflects the temperature that the blood cells, proteins, organs, and heat-susceptible components are exposed to, and thus it makes sense that the core temperature is what determines whether our bodies act to dissipate heat to the environment.

2. **What 4 mechanisms of energy transfer impacts the body's temperature[1,2,5,6]? Can you give an example of each mechanism?**

- Conduction (heat transfer involving direct contact between surfaces; putting your warm hand on a cool metal computer would facilitate conduction from the higher temperature at your palm down the temperature gradient to the cooler metal; placing a patient with heat illness on a cold metal surface with direct skin-to-metal contact would similarly facilitate heat transfer from the patient's skin down the temperature gradient to the metal gurney directly via conduction.)

- Convection (heat transfer to moving liquid or air; as cold air blows over hot skin the heat energy is transferred down the temperature gradient by way of convection; can be enhanced by the use of fans to increase the movement air over the skin)
- Evaporation (heat transfer through change of state from liquid to vapor, such as sweating; just as a pot of water requires heat from a stove to boil into steam and the energy is absorbed by the liquid to transform into a gas, the transformation of liquid sweat into a gas uses available sources of energy, and thus effectively decreases heat energy at the skin.)
- Radiation (heat transfer by electromagnetic waves; the rapid warming you experience when you move from a shaded spot into a noon sun is an example of radiation energy transfer via energy from the sun's rays.)

3. Which mechanisms are the most effective for decreasing body temperature in conditions of thermal stress? How does increased ambient air temperature, increased humidity and/or decreased air flow affect these mechanisms for cooling [1,4,6–8]?

- Convection and evaporation are the most effective means for the body to dissipate heat under conditions of thermal stress, particularly in a dry environment. A hot human can dissipate heat to air that is lower temperature via **convection**. This can be aided by increasing the amount of blood flow (moving fluid) in the skin as well as through increasing convective currents (moving air). You can imagine air adjacent to the body accepting the exuded heat from the skin, and becoming warmer and less dense. Even without aid of a breeze, convection can facilitate the rising away from the body of warm air that is then replaced by cooler air. **Evaporation** involves the conversion of a liquid to a gas, an act which absorbs energy. The energy as heat is taken from the skin to enable the phase transformation from water to gas.This results in cooling of the skin.
- Methods such as wetting the skin and using a fan provide augmented cooling by increasing both evaporation and convection.
- Many of these mechanisms become less effective as ambient air temperature, humidity and air stagnation increase. Evaporation depends upon the water vapor pressure gradient between the skin and environment, so evaporation becomes less effective as humidity rises, At a heat index of 95^{o}F/35^{o}C, as you might imagine, convection or conduction become essentially ineffective for cooling. Evaporative heat loss is the only effective means of intrinsic cooling when the ambient air temperature exceeds the body temperature. Of course, air currents can augment convective cooling (and evaporation).
- Under normal resting conditions in a relatively cool and dry environment with still air, convection contributes little because there isn't much blood flow to the skin at baseline, and, instead radiation and evaporation (probably related to respiratory water losses) provide the major mechanisms for the body to dissipate the heat that is generated by basal metabolism, with conduction and convection contributing significantly less

4. How does increased body heat affect the cardiovascular system[1]?

- Cardiac demand is increased due to peripheral vasodilation and decreased peripheral resistance; eventually, hypovolemia from sweating without sufficient fluid replacement can also contribute to this.
- Right heart blood flow and, hence, preload volume is reduced from shunting of blood to the skin.

- Contractility is increased while stroke volume remains the same or slightly increased (while the body is still able to compensate).
- Cardiac demand is maintained predominantly by increased heart rate stimulated by sympathetic response in the heightened adrenergic state of hyperthermia. This is due to a direct heat effect on the SA node.

5. What pre-existing medical conditions, biologic characteristics and/or medications can affect the body's ability to regulate heat[1,9–17]?

- Poor baseline cardiac conditioning due to myocardial damage or medications that suppress heart rate, or cardiac work and/or contractility, such as beta blockers
- Type 2 diabetes may cause increased risk, possibly due to peripheral neuropathy, poor peripheral blood flow and the effects of fluid and electrolyte disturbances related to glucose concentration
- Relative dehydration from diuretic use and other medications
- Decreased sweat production due to prescription and illicit medications, particularly those with anticholinergic effects
- Pregnancy may cause increased vulnerability due to pregnancy-related weight gain and the attendant lowering of the ratio of body surface area to mass. Decreased surface area can make it harder to dissipate internal heat, while increased fetal metabolic demands can generate more heat. The increased susceptibility to dehydration can increase the clinical risks.
- Older age can increase vulnerability due to the higher presence of pre-existing medical conditions, possible attenuated physiologic responses to heat, potential for impaired cognition resulting in inability to behaviorally thermoregulate, and a decreased perception of thirst and decreased sweat response.
- Younger age (particularly less than 4 years) can cause increased risk due to higher surface area which could allow greater absorption of environmental heat. Additionally, infants have less effective intrinsic heat dissipation mechanisms as compared to adults, and they are often unable to intentionally thermoregulate through behavioral means.
- Patients with respiratory disease, particularly chronic obstructive pulmonary disease, have a higher risk of death during heatwaves, and respiratory deaths are a common secondary cause of death during heatwaves. The mechanism is unclear, but has been suggested to be related to the heat-induced inflammatory and coagulation responses. Hyperventilation associated with heat dissipation as well as the inhalation of hot air may trigger bronchoconstriction.
- Mental health conditions may increase vulnerability to heat through the actions of medications with anticholinergic effects (e.g. tricyclic antidepressants) as well as through potential interference with behavioral thermoregulation
- Obesity may worsen one's vulnerability to heat-caused illnesses due to an increased body mass relatively greater than surface area, resulting in a greater heat storing capacity and lower surface area to dissipate heat. Furthermore, adipose tissue may have lower thermal conductivity, impairing the transfer of heat from the core to the periphery. Higher cardiovascular events may be seen in obese individuals given a predisposition to coronary vascular disease and heart failure as well as an increased inflammatory response. Finally, obesity has been shown to be associated with alterations in water homeostasis and an increased propensity to dehydration, plasma hyperosmolality and kidney dysfunction.

- Chronic kidney disease may exacerbate the heat-associated risk of development of cardiovascular events, electrolyte imbalances and acute kidney injury. The kidneys in CKD have a blunted ability to concentrate urine, as well as impairment of potassium excretion and of regulation of acid-base balance. In addition, there is upregulation of coagulation pathways in end-stage renal failure as well as potentially with moderate CKD.

6. What would you put on your differential diagnosis list for a patient presenting with hyperthermia and CNS dysfunction?

- **Faculty discussion:** Given your patient population, help the student determine a differential diagnosis for a patient they might see in your practice setting who presents with CNS dysfunction and hyperthermia.
    - **i. Possibilities include**: status epilepticus, encephalitis, anticholinergic toxicity, amphetamine use, and thyroid storm

7. Which organ systems are involved in heat stroke?

- **Faculty discussion:** Help the student go through the organ systems and determine the effects of heat stroke on each system
    - **i. They can** consider how the effects of peripheral blood redistribution, activation of inflammatory and coagulation pathways, dehydration and direct cytotoxic effects of heat contribute to injury throughout the organ systems.

8. Consider which groups of patients might be at greater risk of severe disease from heat exposure related to their *social or environmental conditions* [13,15,18]?

- Those without secure housing or without adequate means to reliable cooling.
- Those who are socially or physically isolated
- Those who engage in high-risk occupations (e.g., construction, agriculture worker, factory worker, military, etc)
- Those who engage in high-risk activities (e.g., athletes)
- Those with lower incomes

9. How can the social determinants of health impact heat vulnerability [13,15,18]?

- Living environment: A lower socioeconomic status associated with living near greater heat absorbent surfaces, having less access to cooling green spaces/trees, having less access to transportation and air conditioning and/or cooling centers. These geographically have been linked to the historically disadvantaged peoples who have been impacted by “redlining”).
- Access to health care: Lower literacy levels and lower paying jobs, as well as limited access to transportation can be associated with less health care access, and can contribute to poor control of pre-existing conditions, reduced access emergency medical care, and lack of education regarding heat vulnerability
- Occupation: Lower income jobs can be associated with working in higher risk occupations (agriculture worker, construction, etc), with potentially less ability to control one’s working environment or to take time off without penalty.

- Higher rate of high-risk pre-existing conditions are seen in those already marginalized due to several factors, including access to healthy diet, exposure to air pollution, increased stress, less access to medical care, among others

10. How accurate is the picture we have of heat-related morbidity and mortality in the US [15,19]?

- It is likely underreported. Mortality data is based on death certificate reporting that explicitly includes hyperthermia. However, it is thought that the contribution of heat to death is often not recognized or is not included on the death certificate. In addition, death data typically excludes non-US citizens. Reporting heat-related illness that does not directly result in death is not required of hospitals or health-care providers (but is reportable to OSHA for places of employment), thus we have a limited picture of the extent of this.

11. Do we see higher rates of heat-related deaths in certain regions, genders and/or ethnic groups[7]?

We do see higher rates of deaths in certain populations. Higher heat-related deaths are seen in the following populations:

- Males (70% of deaths)
- American Indian/Alaska Native people
- Black people
- Arizona, California and Texas have a greater proportion of deaths per capita
- Large metropolitan and small rural populations

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