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The pathophysiology of heat exposure

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Heat exposure of the body can result in different physiological/pathological outcomes (**Slide 1**). Behavioral (e.g., cold seeking) and autonomic (e.g., skin vasodilation, sweating) warmth-defense effectors are recruited to maintain normal body temperature.[1] Physiologically, successful activation of the effectors results in warmth tolerance. In case of repeated, chronic heat exposure the tolerance intensifies leading to heat acclimation.

Heat-related illnesses develop when the pathological effects of heat load are not prevented. Syndromes vary from less severe, such as heat syncope to severe forms as lethal heat stroke.

Activation of autonomic heat-defense effectors affects the regulation of homeostatic systems other than thermoregulation. Increased cutaneous vasodilation and decreased venous tone reduce ventricular filling, which, in an orthostatic position, may lead to low brain perfusion and heat syncope (with unconsciousness). Electrolyte imbalance can develop due to sweating, and promote the occurrence of heat cramps, i.e. short-lived, painful contractions of skeletal muscles during or after prolonged work in the heat.[2]

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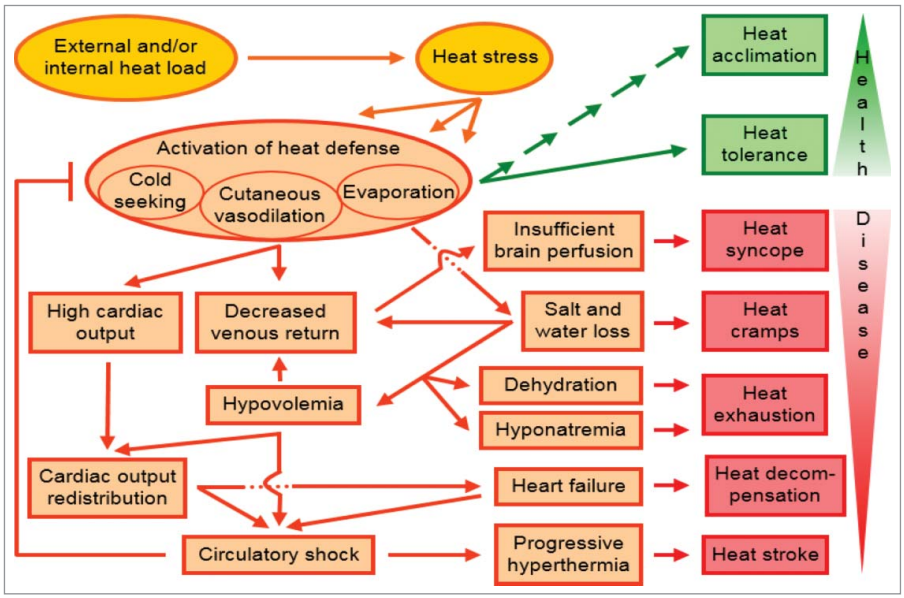

Slide 1. The consequences of heat exposure in health and disease.

In severe hypovolemia, heat exhaustion develops with domination of water- or salt-depletion.[3] The former is mainly due to insufficient fluid replacement and consists of thirst, progressive hypovolemia, hyperosmolarity and hyperthermia. Salt-depletion dominates when sweating-induced water loss is replaced, but salt is not (e.g., by soft drinks), leading to hypoosmolarity and its consequences (e.g., cell swelling).

In patients with compensated heart failure, the need for a higher cardiac output during heat exposure may manifest heart failure: high end-diastolic volume/pressure with backward (venous congestion, edema) and forward (insufficient muscle/renal/intestinal perfusion) failure symptoms. Consequently, the patient collapses because of muscle weakness. Although consciousness is maintained, the developing heat decompensation is more severe than heat syncope.

The most severe form of the heat-related illnesses is heat stroke, when body temperature usually exceeds 41°C, neurological dysfunctions, and in some cases anhydrosis are present. Classic heat stroke affects subjects with compromised warmth-defense capabilities (e.g., infants, elderly), while exertional heat stroke occurs in healthy, young adults during high-performance activities in hot environment. In heat stroke, the signs and symptoms of systemic inflammation, distributive/hypovolemic circulatory shock, multiple organ dysfunction, disseminated intravascular coagulopathy, electrolyte, pH, and osmotic imbalance are manifested simultaneously.[4]

Teaching Slide

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