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Scorching Heat and Fragile Hearts: Hidden Cardiovascular Risks

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Rising summer temperatures lead to a surge in heat stroke cases in emergency rooms, typically marked by body temperatures of 40.5 °C or higher.[1] This spike in body temperature triggers the body's heat dissipation mechanisms, with the cardiovascular system playing a critical role by boosting cardiac output and peripheral blood flow. However, extreme heat intensifies the demand for the heart to supply adequate blood and oxygen, resulting in cardiac stress. Cardiovascular complications are common in heat stroke and significantly contribute to morbidity and mortality. Research estimates that up to 93,000 cardiovascular deaths worldwide are linked to heat exposure annually.[2] Additionally, cardiovascular mortality can increase by 11.7% during heat waves, especially among patients with preexisting heart disease, who are particularly vulnerable to these effects.[3]

Heat stroke occurs when the body generates more heat than it can dissipate, potentially leading to an exaggerated inflammatory response.[4] Thermoregulation, the process of heat dissipation primarily through sweat evaporation, governs the body's heat control. The cardiovascular system transports heat in the blood from arteries and veins toward the limbs, where most heat dissipation occurs.[5] During heat waves, metabolic activity increases, overwhelming the body's heat-dispelling capacity. To manage this stress, cardiac output must rise, yet this compensatory mechanism may fail, resulting in heat stroke.

Tachycardia is the primary sign of cardiac stress during heat stroke,[3] along with increased cardiac markers and ST-segment deviations on electrocardiograms, indicating potential ischemia and heart failure.[6] Heat stroke patients often show decreased left ventricular volume, which lowers cardiac stroke volume and coronary blood flow, increasing myocardial ischemia risk.[7] On a molecular level, heat stroke triggers the release of protective proteins like HSP40, HSP60, and HSP90 and cytokines to protect cellular structures.[8] The activation of immune and inflammatory responses to these heat shock proteins in blood vessel walls may be critical in triggering and sustaining atherosclerosis.[9] Heat waves notably increase sudden death risks, primarily due to underlying cardiovascular conditions, which may lead to heart failure from ischemia and stress-induced cardiomyopathy. Although the impact of heat stroke on the cardiovascular system is evident, comparative studies are needed to assess heat stroke-induced cardiomyopathy incidence and mortality.

In conclusion, heat stroke poses a serious threat to cardiovascular health, with severe complications like acute kidney injury, rhabdomyolysis, respiratory issues, and potentially death if not managed promptly.[10] Prolonged dehydration and high temperatures heighten cardiovascular strain, mainly through a supply-demand mismatch, underscoring the need for timely preventive measures. Staying hydrated, minimizing sun exposure, and monitoring for heat stress can mitigate heat-related illnesses, especially during heat waves and strenuous activity. Additionally, public awareness campaigns are essential to educate vulnerable populations. Large-scale studies are needed to fully clarify the clinical impact of heat stroke on cardiovascular mortality and morbidity.

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