Prolonged exercise is associated with extensive increases in blood flow to the active tissues (e.g., muscle) in response to increased oxygen demand as well as to the skin to permit adequate heat dissipation via evaporative heat loss and dry heat exchange. Consequently, exercise of increasing duration can result in marked strain on the cardiovascular and thermoregulatory systems. On the other hand, passive heat stress can cause substantial increases in skin blood flow (even up to ~8 l/min in extreme situations) relative to modest (if any) changes in muscle blood flow. Accordingly, it has been postulated that combining exercise with ambient heat stress in humans can pose one of the most severe challenges to the regulation of the cardiovascular system stemming from the extensive competition for blood flow between the active muscle tissue and the skin (i.e., the thermoregulatory system). Moreover, recovery from exercise has been associated with a rapid decline in local and whole body heat loss, which is thought to be mediated by factors of central origin. As a result, an elevated residual heat storage paralleled by sustained elevations of core and muscle temperatures relative to baseline levels are observed lasting for as long as 90 min after exercise and by a magnitude that is dependent on the intensity of the preceding exercise bout.

The postexercise period has also been associated with impairments in cardiovascular control such that mean arterial pressure is sustained well below baseline levels. Importantly, this disturbance in blood pressure regulation following exercise-induced heat stress has been found to elicit a reduction in orthostatic tolerance, although the mechanisms are unclear. Some information may be gleaned from cardiovascular responses during passive heating at rest, which is associated with progressive reductions in central venous pressure and central blood volume. When the cardiovascular reflexes are overwhelmed by the demand for circulation to the skin, syncope may ensue. This point at which the autonomic control of blood pressure fails is potentiated by inadequate circulation of blood to the brain. Passive heat stress has been shown to elicit failure of blood pressure control at an earlier point compared with a non-heat stress condition (i.e., the point of failure exhibits a leftward shift to a lower level). In particular, a recent study by Keller and colleagues compared orthostatic tolerance through the incremental application of lower body negative pressure (LBNP; represented by a survival curve defined as the level of LBNP that induced presyncope) under normothermic and hyperthermic conditions (e.g., core body temperature ~1.5°C above baseline levels). Through application of LBNP during heat stress, a greater strain is placed on the cardiovascular system as more blood is pooled in the lower limbs, thereby further reducing central blood volume and requiring greater increases in heart rate and total peripheral resistance to maintain mean arterial pressure. Findings from this study indicated that orthostatic tolerance was negatively influenced by heat stress. This was evidenced by the survival curve exhibiting a leftward shift to a lower point of autonomic failure such that the critical level of LBNP found to induce presyncope was ~40% lower in individuals rendered passively hyperthermic.

Given that there exist pronounced levels of blood pooling in the previously active muscle following a bout of exercise-induced heat stress, it is plausible to suggest that tolerance to LBNP would be further reduced compared with that during passively induced hyperthermia. However, in this issue of *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, Pearson and colleagues provide strong evidence to suggest that this is not the case. Rather, their findings implicate a novel role for skin temperature as the underlying modulator of LBNP tolerance for a given increase in core body temperature (increase of 1.2°C above baseline resting levels of ~36.9°C in this study) that was irrespective of the method by which heat stress was achieved (i.e., passive or exercise-induced). In particular, LBNP tolerance (quantified by the time at each level of LBNP, multiplied by that level and presented as the cumulative stress index) was similar following passive and exercise-induced hyperthermia when skin temperature was clamped at 38°C. Conversely, orthostatic tolerance was increased (i.e., the cumulative stress index was greater) following exercise-induced hyperthermia when skin temperature was clamped at 36.5°C. These results were specifically applied in context of soldiers who are at risk for a hemorrhagic injury during simultaneous exposure to passive (i.e., warm environments) and/or active heat stresses (i.e., foot patrols) while wearing body armor. Based on their findings, the authors recommended the implementation of measures to reduce skin temperature in an attempt to delay the point of cardiovascular failure as long as possible.

Other important implications for these findings include individuals such as workers in occupational settings, athletes, and others who as a result of short duration moderate-to-high intensity work performed in the heat experience an elevated state of hyperthermia (i.e., core body temperature ≥39.0°C) in combination with extensive dehydration. Under such conditions, inadequate fluid replacement can be associated with profound reductions in blood volume, which can reduce cardiac filling pressure and consequently alter blood pressure.
sure regulation. These conditions can be exacerbated in workers and soldiers who may be required to continue low intensity physical activity for prolonged periods especially when insulative protective clothing is worn. In extreme conditions, individuals may experience exertional heat stroke (i.e., core body temperature ≥40°C). Irrespective of the pathway to hyperthermia, a decrease in orthostatic tolerance can place the individual at greater risk for cardiovascular collapse. In fact, victims of exertional heat stroke often experience a rapid deterioration in cardiovascular control (i.e., a precipitous drop in blood pressure), which is paralleled by acute thermoregulatory dysfunction (i.e., decreases in heat dissipation and subsequent thermoregulatory failure), particularly with any delay in treatment (2). Under conditions that may require the individual to perform under extreme heat stress conditions (e.g., high ambient air temperatures and/or radiant heat sources such as the sun, boilers, mechanical systems, etc.) the elevated core temperatures can be paralleled by markedly elevated skin temperatures. Thus, based on the study findings by Pearson et al. (8), such individuals are likely at greater risk for heat-related syncope due to a significant leftward shift in the survival curve for the point at which they experience cardiovascular failure. It is important to consider, however, that their study employed LBNP in the supine posture (8). While this position would likely parallel that of a wounded soldier on the battlefield, it does not take into consideration any postural influences that may present an added stress for the athlete or occupational worker who may work and/or recover in the upright posture for an extended period of time.

In taking these findings together, it is clear that implementation of adequate cooling strategies are paramount to improve the health and safety of individuals at risk for developing heat stress (e.g., military personnel, firefighters, miners, and others). The method of cooling recommended by Pearson and colleagues (8) could incorporate a light-weight non-ice modality that can effectively reduce skin temperature. While this approach may be effective as a treatment modality for a soldier with hemorrhagic injury, the same intervention may not be sufficient for workers and/or athletes who may experience substantially greater levels of hyperthermia (i.e., core body temperature ≥39°C) even during prolonged light work in the heat. Under these conditions, cooling treatments that enhance heat dissipation by increasing conductive and/or convective heat transfer and/or evaporative heat loss are critical for the victim’s survival. This is because the primary goal of acute and emergency care in exertional heat-related illness is to restore core temperature to near-normal levels as rapidly as possible. The current gold standard treatment for exertional heat stroke is the immersion of the individual in a tub of cold water (i.e., 2–10°C) (2). While this method can be impractical in certain situations given the materials may not always be available, similar strategies (e.g., applying cold towels to cover the skin and changing them frequently) can be effective in reducing core temperature. In fact, though these methods are aimed first and foremost to manage hyperthermia and minimize the risk for heat-related injury, evidence also indicates that cardiovascular stability will benefit simultaneously (2). Finally, these findings raise important questions regarding the mechanisms governing cardiovascular control following passive- and exercise-induced heat stress. In particular, further examination is warranted to evaluate how orthostatic tolerance, and thereby the point at which cardiovascular failure occurs, would differ at greater levels of hyperthermia (i.e., ≥39°C) and whether the influence of skin temperature would be as impactful under such conditions when the competing effects of the thermoregulatory and cardiovascular systems for blood flow are enhanced.

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AUTHOR CONTRIBUTIONS

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