Autonomic Neuroscience: Basic and Clinical 196 (2016) 52–62

Contents lists available at ScienceDirect

Autonomic Neuroscience: Basic and Clinical

journal homepage: www.elsevier.com/locate/autneu

Review

Cardiovascular adaptations supporting human exercise-heat acclimation

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ARTICLE INFO

Article history:
Received 15 October 2015
Received in revised form 29 January 2016
Accepted 4 February 2016

Keywords:
Blood pressure regulation
Cardiac output
Exercise performance
Fluid balance
Heat acclimatization
Stroke volume
Thermoregulation

ABSTRACT

This review examines the cardiovascular adaptations along with total body water and plasma volume adjustments that occur in parallel with improved heat loss responses during exercise-heat acclimation. The cardiovascular system is well recognized as an important contributor to exercise-heat acclimation that acts to minimize physiological strain, reduce the risk of serious heat illness and better sustain exercise capacity. The upright posture adopted by humans during most physical activities and the large skin surface area contribute to the circulatory and blood pressure regulation challenge of simultaneously supporting skeletal muscle blood flow and dissipating heat via increased skin blood flow and sweat secretion during exercise-heat stress. Although it was traditionally held that cardiac output increased during exercise-heat stress to primarily support elevated skin blood flow requirements, recent evidence suggests that temperature-sensitive mechanisms may also mediate an elevation in skeletal muscle blood flow. The cardiovascular adaptations supporting this challenge include an increase in total body water, plasma volume expansion, better sustainment and/or elevation of stroke volume, reduction in heart rate, improvement in ventricular filling and myocardial efficiency, and enhanced skin blood flow and sweating responses. The magnitude of these adaptations is variable and dependent on several factors such as exercise intensity, duration of exposure, frequency and total number of exposures, as well as the environmental conditions (i.e. dry or humid heat) in which acclimation occurs.

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1. Introduction

It is well established that cardiovascular strain contributes to impair aerobic exercise performance in the heat (Rowell, 1974; Cheuvront et al., 2010; Nybo et al., 2014) and that cardiovascular adaptations are important contributors to the improved exercise capacity and reduced risk of serious heat illness conferred by exercise-heat acclimation (Sawka et al., 2011). Early physiologists recognized that a reduction in the elevated heart rate from heat stress likely was an important marker of adaptation to hot climates (Sundstroem, 1927). Lee and Scott (1916) were among the first to appreciate that the cardiovascular system likely imposed a physiological limitation to exercise performance under heat stress by "drafting blood away from the brain and the muscles to the skin". Subsequently, Hill and Campbell (1922) demonstrated that

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http://dx.doi.org/10.1016/j.autneu.2016.02.002

improved evaporative cooling of the skin reduces cardiovascular strain and "keeps off fatigue" during exercise-heat stress. Perhaps the most important modern contributions regarding cardiovascular control with heat stress were made by Rowell (1974); Rowell et al. (1996) and Johnson (1977); Johnson et al. (2014), and integrated adjustments in such controls are critical to human heat acclimation (Taylor, 2014; Périard et al., 2015a). This review examines the cardiovascular adaptations along with total body water and plasma volume adjustments that occur in parallel with improved heat loss responses during exercise-heat acclimation. These physiological adaptations/adjustments are critical to exercise-heat acclimation as upright posture, increased cutaneous vasodilation and greater sweat secretion provide severe challenges to blood pressure regulation, performance, and health during exercise hot conditions.

2. Adaptation to heat

When humans are repeatedly exposed to conditions that are sufficiently stressful to elicit profuse sweating and elevate skin and core temperature, adaptations develop that reduce the deleterious effects of heat stress: heat acclimation or acclimatization. Heat acclimation refers to repeated periods of heat exposure undertaken in artificial or laboratory settings, whereas heat acclimatization results from exposure to natural environments. Although both natural and artificial hot environments elicit similar physiological adaptations (Armstrong and Pandolf, 1988; Wenger, 1988), heat acclimatization provides more specific responses due to exposure to the exact conditions that will be encountered during work or competition (i.e. exercise task, solar radiation and terrain/geography) (Périard et al., 2015a). The phenotypic adaptations that develop during repeated exposure to hot conditions improve performance during submaximal exercise, increase maximal aerobic capacity ($\dot{V}O_2$max) (Sawka et al., 1985; Lorenzo et al., 2010) and enhance thermal comfort (Lemaire, 1960; Folk, 1974; Gonzalez and Gagge, 1976) in the heat. These benefits are achieved through plasma volume expansion, better maintenance of fluid balance, enhanced sweating and cutaneous blood flow responses, lowered exercising metabolic rate, and acquired thermal tolerance through the heat shock response, all of which contribute to improved cardiovascular stability and exercise performance during heat stress (Wyndham et al., 1976; Hori, 1995; Sawka et al., 1996; Horowitz, 2014; Périard et al., 2015a).

Physiological adaptations related to repeated heat exposure develop relatively quickly with 75–80% of the acclimation process occurring in the first 4–7 days (Fig. 1) (Pandolf, 1998; Shapiro et al., 1998). These adaptations can be categorized into short-term (<7 days), medium-term (8–14 days) and long-term acclimation (>15 days) (Garrett et al., 2011). Horowitz et al. (1993, 1996, 1998) argue that heat acclimation develops as a continuum of processes and have proposed a conceptual model characterized by a distinct biphasic pattern. The initial short-term phase is transient and manifested by a decreased effector organ output-to-autonomic signal ratio, such that accelerated efferent activity is required to override the suboptimal peripheral responsiveness and produce adequate effector output. The second long-term phase is stable and characterized by an increased effector organ output-to-autonomic signal ratio, as both central and peripheral adaptations enhance physiological efficiency and reduce the need for accelerated excitation.

Although passive heat exposure induces adaptations commensurate with that magnitude of strain (Takamata et al., 2001; Beaudin et al., 2009; Brazaitis and Skurvydas, 2010) and passive hot water immersion after exercise can improve endurance performance in the heat (Zurawlew et al., 2015), the inclusion of exercise with heat exposure provides additional strain that generally elicits more profound adaptations (Armstrong and Pandolf, 1988; Wenger, 1988). Accordingly, the magnitude of physiological adaptation induced by heat acclimation or acclimatization depends largely on the initial heat exposure status (i.e. recent heat exposure, season, fitness level), as well as the exercise intensity, duration, frequency, and number of heat exposures, along with the induction protocol (Sawka et al., 1996; Taylor, 2000; Taylor, 2014; Périard et al., 2015a).

Repeated exercise-heat exposure at a constant work rate (i.e. traditional occupational and military heat acclimation protocol) is not likely as effective in eliciting adaptation as maintaining hyperthermia at a given core temperature (e.g. 38.5 °C; controlled hyperthermia or isothermic heat acclimation) (Taylor, 2000, 2014). Indeed, the traditional heat acclimation model offers a constant forcing function (i.e. fixed endogenous and exogenous thermal loads), which as adaptations progressively develop, results in decreased physiological strain and reduced further adaptations (Eichna et al., 1950; Fox et al., 1963a; Rowell et al., 1967). In contrast, with controlled hyperthermia protocols the forcing function is increased in proportion to the adaptations by manipulating the endogenous and/or exogenous thermal loads (Garrett et al., 2011; Taylor, 2014). Therefore, it is suggested that greater physiological adaptations occur during a given period with controlled hyperthermia than traditional approaches. Interestingly, recent studies have not found greater adaptations with controlled hyperthermia and the explanation for those findings are unclear (Gibson et al., 2015a,b).

The similar adaptations between some traditional and controlled hyperthermia approaches may stem in part from different acclimation protocols inducing distinctive autonomic responses that result in

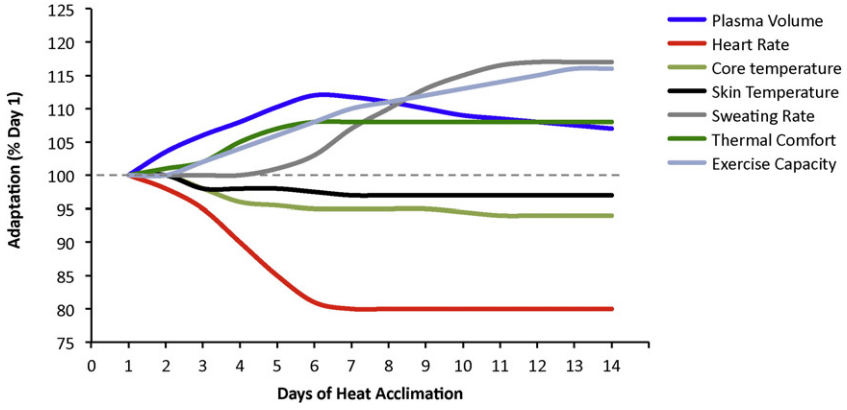

Fig. 1. The time course of adaptations to exercise-heat acclimation. Within a week of acclimation plasma volume expansion occurs and heart rate is reduced during exercise at a given work rate. Core and skin temperatures are also reduced when exercising at a given work rate, whereas sweat rate increases. Perceptually, the rating of thermal comfort is improved. As a result, aerobic exercise capacity is increased. Of note, the magnitude of these adaptations is dependent on the initial state of acclimation and the acclimation protocol (e.g. environmental conditions and exercise intensity). Adapted with permission from Périard et al. (2015a).