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CORRESPONDENCE

Cardiovascular Determinants Involved in Pacing Under Heat Stress

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Dear Editor,

I read with interest the review by Roelands et al. [1] regarding the "Neurophysiological Determinants of Theoretical Concepts and Mechanisms Involved in Pacing." The manuscript explains much of the recently published literature pertaining to the novel mechanisms suggested to regulate or influence pacing and pacing strategy during time-trial exercise. As the authors highlight, the regulation of prolonged self-paced exercise is multifactorial and as such is influenced by a variety of factors, including the development of hyperthermia, brain neurotransmitters, the perception of effort, neuromuscular function, and metabolism. However, a particular mechanism that is conspicuously absent from the discussion, particularly when referring to prolonged efforts in the heat, is that of cardiovascular limitations. The first goal of the manuscript was to review the existing literature on pacing strategies in different climatic conditions [1]. Rightly, it is underscored that during exercise in the heat, a redistribution of blood from the core to the skin may decrease available blood flow to the exercising muscle. Yet, this potential influence on performance is not further discussed. Rather, the authors conclude that "recent literature showed that during exercise in the heat, a reduction in power output and muscle activation occurs before a critical core temperature is reached, indicating that subjects can anticipate the exercise intensity and heat stress they will be exposed to; thereby preventing catastrophic outcomes" [1].

Although the concept of cardiovascular limitations during prolonged exercise in the heat dates from the early work of Rowell [2–4], it is in itself not dated. The premise of this mechanism lies with the redistribution of blood flow from the central to the peripheral circulation for the purposes of thermoregulation. During exercise in the heat, core and skin temperature increase. This increase narrows the core-to-skin temperature gradient, which in turn increases the skin blood flow requirement for heat dissipation [5]. The rise in cutaneous blood volume and a temperature-mediated increase in intrinsic heart rate result in a decrease in cardiac filling, which ultimately leads to the reduction in stroke volume [3, 6–9]. Consequently, when exercise is performed in the heat, maximum cardiac output decreases [10, 11] and the cardiovascular system is forced toward a functional limit at submaximal workloads and oxygen uptake (i.e., maximal aerobic capacity is reduced) [12–15]. This reduction in cardiovascular reserve is purported to be the primary factor limiting constant rate aerobic exercise in the heat, and is manifested as an increase in relative exercise intensity [% maximum oxygen uptake ($\%VO_{2max}$)] and perceived exertion [10, 11, 16].

While the previous observations pertain to constant-rate exercise and have been reported outside the literature search range (2004–2012) defined by the authors, two self-paced studies that fall within the literature search parameters were not included. Indeed, Ely et al. [17] examined well-trained runners performing an 8-km time trial in warm (~30 °C) and cool (~17 °C) environments. As with most studies, it was observed that time to completion was longer in warm conditions. However, the authors provided evidence against both the attainment of a critical core temperature and changes in heat storage regulating pacing in

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an anticipatory manner. They observed that despite the attainment of a core temperature of 40 °C (i.e., the proposed critical core temperature) in the warm condition, running velocity did not decline. In fact, several runners were able to perform an end-spurt at core temperatures above the critical threshold. Moreover, the rate of heat storage did not mediate any modifications in pacing strategy in either the warm or cool environments [17]. Of note, the premise on which the anticipatory model of pacing was developed, calculations of early changes in the rate of heat storage [18, 19], has been shown to be erroneous, suggesting no feedforward mechanism associated with the rate of heat storage [20]. Thus, Ely et al. [17] concluded that the simplest and most plausible explanation for the decline in performance time observed in warm conditions was a reduction in peak aerobic capacity.

In a recent review, Cheuvront et al. [5] acknowledged that the central nervous system and skeletal muscles provide valuable afferent feedback to the conscious brain. They also acknowledged that this sensory input influences performance under heat stress. However, they suggested that the requirements and limitations on blood flow during aerobic exercise in the heat implicate a much larger performance contribution by the cardiovascular system. Accordingly, we recently demonstrated that a thermoregulatory-mediated rise in cardiovascular strain during a 40-km time trial in hot (35 °C) conditions was associated with reductions in sustainable power output, peak oxygen uptake, and maximal power output, compared with the same effort undertaken in cool conditions (20 °C) [21]. More specifically, we observed that heart rate was higher throughout the hot exercise trial, while stroke volume, cardiac output, and mean arterial pressure were significantly depressed compared with exercise in the cool condition. Furthermore, an 18 % greater reduction in peak aerobic capacity was measured during the final kilometer of the time trial at maximal effort in the heat, relative to the cool condition. This suggests the utilization of a progressively greater percentage of peak aerobic capacity during the development of thermal and cardiovascular strain for a given power output. As a result, power output in the heat was reduced in order to continue exercising at the physiological limit, or critical power (i.e., fatigue threshold), which requires balancing the relative contribution of aerobic and anaerobic energy metabolism [22, 23]. This adjustment in pacing supports the typical time-trial profile observed in the heat, whereby greater reductions in power output occur in the early stages of exercise after self-selection of an aggressive pacing strategy [1].

In summary, it is clear that the interplay between peripheral and central afferent inputs mediates a behavioral response in consciously regulating pace. The authors have presented a comprehensive review of these concepts, as well as the role of manipulating brain neurotransmitters in further affecting endurance performance, especially in the heat. However, it is also clear that cardiovascular limitations induced by adjustments in thermoregulation can significantly influence the maintenance of a given absolute intensity or pace. Although this premise does not involve the complex interplay and integration of conscious and subconscious signals, it remains a fundamental concept in human physiology.

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