Review Article

Cardiovascular Drift and Critical Core Temperature: Factors Limiting Endurance Performance in the Heat?

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Prolonged severe exercise stresses the cardiovascular system with the need to address the competing demands of blood flow to both the exercising muscles and the skin, while maintaining arterial blood pressure for adequate blood flow to the brain. With the imposition of an environmental heat load in addition to a high metabolic heat load, core temperature steadily increases along with a number of associated circulatory adjustments that inevitably signal an increasing strain on homeostatic control. Whether endurance performance under these conditions is limited by the rise in core temperature (critical core temperature hypothesis) or is limited by the inability to meet the competing demands for blood flow is debatable. The "critical core temperature" hypothesis is centered on an elevated body temperature providing peripheral afferent feedback from temperature-sensitive sites to the central nervous system (CNS), resulting in reduced CNS drive to the exercising muscles. Alternatively, exercise endurance in the heat may be limited by the rise in heart rate (HR) toward maximum HR that accompanies the increase in core temperature. The rise in HR is thought to compensate for a decrease in stroke volume (SV) as central venous pressure declines due to increased skin blood flow. However, recent research suggests that the upward drift in HR is the primary event that causes a decline in SV. Thus, the circulatory strain associated with HR approaching maximum and an inability to maintain cardiac output may signal fatigue in prolonged exercise in the heat.

Keywords: cardiovascular drift, fatigue, prolonged exercise

Introduction

The early research by Marius Nielsen (1938) demonstrated that humans can regulate their core temperature during exercise, independent of the climatic conditions over a relatively wide range of ambient temperatures. The range over which a steady-state core temperature is maintained independent of the ambient temperature is referred to as the "prescriptive zone." Within the prescriptive zone, Saltin and Hermansen (1966) showed that the rise in rectal temperature during "steady-state" exercise is related to the intensity of exercise when expressed relative to maximum oxygen uptake (%$\dot{V}O_{2max}$). Thus, there appears to be a linear relationship between %$\dot{V}O_{2max}$ and rectal temperature when exercise is performed at submaximal intensities in climatic conditions conducive to heat dissipation. However, as the intensity exceeds ~85% $\dot{V}O_{2max}$, this linear relationship appears to become curvilinear suggesting

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that the rate of metabolic heat production cannot be matched by heat dissipation mechanisms. Davies (1979) subsequently confirmed the earlier research finding of Lind (1963), which demonstrated a reduced prescriptive zone with increases in exercise intensity. With the imposition of an elevated environmental heat load in addition to a high metabolic heat load during intense exercise, core temperature steadily increases and may precipitate collapse. With the rise in core temperature, there are a number of associated circulatory adjustments (cardiovascular drift) that inevitably signal an increasing strain on homeostatic control.

Current research concerning the limiting factor(s) for prolonged exercise performance in hot environmental conditions is centered on two avenues of enquiry.

1. A decline in mean arterial blood pressure occurs together with an upward drift in heart rate (HR) until maximum HR (HR_{max}) is reached whereupon cardiac output (CO) is compromised, muscle and/or skin blood flow (skBF) decrease and exercise terminated.
2. The attainment of a so-called "critical core temperature" signals a reduced central nervous system (CNS) activation of the exercising muscles leading to a reduced power output or the cessation of exercise.

Is exercise in the heat limited by circulatory strain?

Rowell (1973) first presented a compelling hypothesis implicating the phenomenon of "cardiovascular drift" in the fatigue associated with prolonged exercise in the heat. The most obvious manifestation of cardiovascular drift is a steady upward drift in HR that is directly associated with increases in body temperature and dehydration (Montain et al. 1992a). HR has been reported to increase by 7 bpm per 1.0°C increase in rectal temperature during exercise (Jose et al. 1970).

The conductance of heat from the exercising muscles to the skin requires a large increase in blood flow to the skin, without compromising muscle blood flow, if exercise is to continue unimpaired by the stress of an accumulating heat load. To meet this requirement a redistribution of the central vascular volume to peripheral regions occurs through a reduction in splanchnic and renal blood flow along with a gradual change in compliance of the capacitance vessels (Johnson 1977; Johnson & Rowell 1975). Rowell (1986) proposed that increased skBF during exercise in the heat is only partly met by a redistribution of CO away from the splanchnic and renal vascular beds. Since the increase in skBF is greater than the reductions in splanchnic–renal blood flows and any increase that may occur in CO, Rowell (1986) argued that muscle blood flow is ultimately compromised. That muscle blood flow may be compromised is indirectly reflected in an elevated blood lactate concentration (Febbraio et al. 1996). The decrease in blood flow to the liver, kidneys, and gut is linearly related to increases in ambient temperature and exercise intensity (%$\dot{V}O_{2max}$). Problems of clinical significance may arise through ischemia and hyperthermia as a result of extended visceral vasoconstriction during prolonged severe exercise (Dancaster & Whereat 1971). Furthermore, the increase in cutaneous venous volume to offset the possibility of metabolic hyperthermia has been implicated as a potential limiting factor for prolonged exercise, especially when this circulatory adjustment is heightened by the addition of an environmental heat stress (Ahmad et al. 2003; Nadel et al. 1977). Arterial and pulmonary pressures decline along with central venous pressure (Johnson & Rowell 1975). Thus with diminished blood flow into the left ventricle, stroke volume (SV) declines.

Rowell's (1973) hypothesis thus proposes that the upward drift in HR is a consequence of increased skBF reducing central blood volume, arterial and central venous pressures so that SV decreases. The increase in HR offsets the decline in SV so that CO is maintained. However, if HR has drifted up toward HR_{max} then CO cannot be maintained if SV continues to decline. Sawka et al. (1979) suggested that the high metabolic rates and elevated body temperatures encountered by competitive distance runners accentuates cardiovascular drift and may eventually result in either decreased cutaneous blood flow or a compromised CO. In a laboratory-based study of Gonzalez-Alonso et al. (1999b) subjects were exercised to fatigue following three different pre-exercise treatments: pre-cooling, pre-heating, and a normothermic condition. While exercise time to fatigue varied with the pre-exercise treatment, subjects were exhausted in all three trials

when HR had drifted to 99% HR_{max}. Note that the CO was lower at the point of fatigue in the pre-heating trial compared with the pre-cooling and normothermic conditions.

In investigations of distance runners competing in road races, researchers have noted the considerable demands placed on the circulatory system in meeting the dual demands of metabolism and temperature regulation (Gisolfi et al. 1977; Costill et al. 1970; Pugh et al. 1967). Telemetered HRs in excess of 90% HR_{max} have been measured in the course of a marathon race (Costill & Winrow 1970) with estimates of exercise intensity between 80% and 88% $\dot{V}O_{2max}$ being maintained (Davies & Thompson 1979). Blake & Larrabee (1903) noted elevated rectal temperatures of subjects at the conclusion of a marathon race. Similar observations of rectal temperature exceeding 40°C have been reported following marathon races on cool, moderately warm, and hot days (Costill et al. 1970; Wyndham & Strydom 1969; Pugh et al. 1967). Even in shorter running races (5–14 km) the high metabolic heat load when combined with an environmental heat load results in rectal temperatures >40°C (Hoffman et al. 1999; Robinson 1963). Robinson (1963) reported data on several elite athletes competing in approximately 5–10 km events where ambient temperature was 30–31°C and humidity and solar radiation were high, rectal temperature increased above 41°C in 15 min. Further observations of unusually high core temperatures have been reported following a 20 km time trial run at 30°C ambient temperature and 89% relative humidity (Thompson et al. 1997). Twenty-six elite endurance runners uneventfully completed the time trial with the highest rectal temperature being 41.8°C, while the mean rectal temperature of the group was 40.87°C. Note that this elevation in rectal temperature is well above the reports of researchers who ascribe to the critical core temperature hypothesis.

In prolonged exercise, the dissipation of heat by sweat evaporation results in relatively large body water losses (Wyndham & Strydom 1969; Pugh et al. 1967). Dehydration which may amount to >5% body weight with marathon running exacerbates the rise in body temperature and associated upward drift in HR. The direct effect of graded dehydration on cardiovascular drift has been well demonstrated by Montain and Coyle (1992a) who observed the magnitude of increase in esophageal temperature and HR to be linearly related to the level of dehydration in cyclists undertaking 2 h of cycling exercise.

If temperature regulation, and thus increased skBF takes precedence over perfusion of the exercising muscles then performance will be impaired unless arteriovenous oxygen difference (A-V O_2) can be increased. However, if under these circumstances the A-V O_2 cannot be increased further, then the muscle will become partially hypoxic and more reliant on anaerobic metabolism. With increasing exercise duration, limited muscle glycogen stores would be available to support anaerobic glycolysis, and as such exercise can only continue if the intensity is reduced. In the event that muscle glycogen availability was adequate to support the increase in anaerobic meta-bolism, pH would inevitably decrease and exercise performance would be impaired.

Note that increased sympathetic drive directed at skeletal muscle could also decrease muscle blood flow. There is a well-documented significant increase in circulating catecholamines with exercise-heat stress and Donald et al. (1970) have demonstrated vasoconstriction to contracting muscle during direct sympathetic stimulation. Similarly, artificial elevation of catecholamines during prolonged exercise has also been shown to cause hyperthermia by vasoconstricting the skin (Mora-Rodriguez et al. 1996).

There is a considerable volume of research that supports the proposition that increasing compliance of the skin capacitance vessels inevitably reduces central venous pressure and SV, leading to an increase in HR to maintain CO. The upward drift in HR is reduced with upright cycling exercise performed in water. This experimental approach provides hydrostatic pressure to the body and, therefore, limits skBF. HR drift is also reduced when exercise is performed in a supine position where there is a greater central blood volume so SV is less likely to be compromised. Further studies on the effect of intravenous fluid infusion (glucose with water) during exercise in the heat have resulted in a significant reduction in the upward drift in HR while a saline infusion has been shown to have no effect on HR drift (Hamilton et al. 1991). The efficacy of fluid replacement in averting HR drift during prolonged

exercise in the heat is based on large fluid losses through sweating causing a reduction in plasma volume. There are a number of studies that report an association between exercise-induced dehydration and an elevated HR response. A decrease in plasma volume increases circulatory strain by increasing the blood viscosity and limits blood perfusion of tissues and organs throughout the body unless there is an increase in blood pressure and/or HR. Conversely, an increase in plasma volume, as observed with heat acclimatization, results in a reduced HR response to a standard exercise load and reduced HR drift (Thompson et al. 2004).

More recent research has questioned the Rowell (1986) hypothesis in part because skin temperature (a crude *de facto* indicator of cutaneous venous volume) appears to plateau at ~38°C while HR continues to increase. The leveling off observed in skBF is likely to occur when baroreflex activity is reaching a limit in maintaining blood pressure, thus ensuring brain blood flow. Thus, the regulation of blood pressure and perfusion of muscle may appear to have precedence at this point with the consequences being severe hyperthermia. Measures of forearm blood flow (FBF) also plateau in hot conditions while HR continues to rise (Gonzalez-Alonso et al. 1997). The addition of radiant heat in these laboratory experiments is likely to further vasodilate the peripheral blood vessels and increase the cutaneous venous volume, which in turn may precipitate collapse. Alternatively, CO will have to increase to avert imminent collapse. CO has been shown to increase with heating of the skin in a rested state, but there are no reports of this adjustment during prolonged intense constant load exercise. That HR continues to increase when a plateau appears to occur in FBF and skBF challenges the link between the upward drift in HR and decrease in SV associated with increased skBF. This is further tested by the observation that SV was similar during 30 min of cycling exercise at ~70% $\dot{V}O_{2max}$ both in hot and cool conditions despite a significantly greater skBF occurring in the heat (Gonzalez-Alonso et al. 2000). Thus, peripheral displacement of blood volume may not be the only cause for the fall in SV with prolonged exercise in hot conditions. The question now arises as to what is causing the upward drift in HR at this point?

There are a number of plausible explanations to consider in addressing this question. First, there may be a gradual decline in the vasoconstrictor activity of the splanchnic and renal beds with local heating of these tissues, thus increasing blood flow to this region. In a hypothetical hierarchical needs assessment, it could be hypothesized that these tissues would possibly take priority over skBF in terms of impending heat injury. However, the limited available research on humans indicates a progressive splanchnic vasoconstriction with increasing HR, although this is not seen in dogs. Reports of liver and kidney damage during prolonged exercise in the heat support the assumption that splanchnic vasoconstriction is maintained despite an injurious heat load on these tissues (Dancaster & Whereat 1971).

The upward drift in HR may in part be a consequence of additional motor units being recruited as muscle fatigue occurs with exercise duration. The evidence is equivocal in supporting this explanation. Studies have reported a significant increase in $\dot{V}O_2$ with prolonged constant power–velocity exercise in association with an increase in HR (Davies & Thompson 1986). However, other studies have not found $\dot{V}O_2$ to increase during prolonged exercise in hot conditions. In addressing this issue, we have recently undertaken some prolonged (1 h) cycling experiments in hot conditions where we have "clamped" HR so that power output is gradually decreased to maintain a constant HR. In these experiments we have found a significant decrease in economy over time i.e., there is a dissociation between $\dot{V}O_2$ and power. Our observations have shown a ~20% increase in the $\dot{V}O_2$ cost of exercise under hyperthermic conditions (Thompson & Chapman, unpublished observations).

We have recently found HR to show a significant upward drift during prolonged negative work (40 min of downhill walking) while no change was observed in SV, resulting in an increased CO. Thus, the upward drift in HR is not a product of a decrease in SV during negative work. However, $\dot{V}O_2$ also showed a significant increase in parallel with HR indicating an increased motor unit recruitment. At 40 min, the rectal temperature had increased by 1.2°C whereas, the muscle temperature increased by 6.2°C (Chapman et al. 2004). There is some evidence to suggest that high muscle temperatures and prolonged exercise may impair mitochondrial

function (Tonkonogi & Sahlin 2002; Brooks et al. 1971). Tonkonogi and Sahlin (2002) showed an increase of 18% in noncoupled respiration of human skinned muscle fibers after prolonged exercise. Such a decrease in exercise economy would require the recruitment of additional motor units to ensure maintenance of force generation. Note that when subjects are trained in downhill walking, indicators of eccentric exercise-induced muscle damage disappear, but $\dot{V}O_2$ still exhibits a significant increase over time (Thompson & Balnave 1996).

There is evidence to suggest that an increase in the intrinsic HR causes a decline in SV. Fritzsche et al. (1999) used beta-blocker drugs to prevent an increase in HR after 15 min during a 60-min bout of exercise and observed no change in SV. They further observed in their placebo control experiment that SV continued to decline when cutaneous blood flow had leveled off, although the largest decrease in SV occurred when cutaneous blood flow increased by more than 600% above resting measures over the first 20 min of exercise. Thus an increase in HR appears to be the primary determinant for the decrease in SV, but this study does not exclude increased skBF precipitating the decline in SV over the first 20 min of the exercise.

As a primary event, an increase in HR would decrease ventricular filling time and end-diastolic volume, therefore causing a decrease in SV. HR could increase as a result of an increase in sympathetic activity, a loss of baroreflex regulation of arterial blood pressure (Norton et al. 1999), a direct local effect of blood temperature on the Sinoatrial node, or in response to an altered central command with increased perception of effort. There may also be an effect of cardiac fatigue with a decrease in left ventricular filling and contractility (Ketelhut et al. 1992).

From a more pragmatic perspective, Arngrimsson et al. (2002) suggest that maximal aerobic power is reduced during the course of prolonged exercise and have shown an association between the decrease in $\dot{V}O_{2max}$ capacity and the upward drift in HR. They suggest that the increase in HR reflects the increasing intensity (% $\dot{V}O_{2max}$) of constant load exercise because the $\dot{V}O_{2max}$ capacity declines. A similar proposition has been suggested by Wingo et al. (2005) who found a relationship between the increase in HR during exercise in the heat and the subsequent decrease in $\dot{V}O_{2max}$. These studies may partly explain the findings of Vautier et al. (1994) where fatigue during continuous exercise occurred earlier than what was predicted when extrapolating the upward drift in HR to HR_{max}.

Summary

While the underlying mechanism for the "cardiovascular drift" associated with prolonged exercise in the heat is contentious, there is little doubt that the ensuing circulatory strain may ultimately limit endurance performance. The unresolved question is which of the three following competing demands dominates during prolonged exercise in the heat:

(1) Increased blood perfusion of the active skeletal muscle to match the metabolic requirements of the exercise?
(2) Increased skBF and volume to dissipate heat generated by the active skeletal muscle?
(3) Maintenance of arterial blood pressure for adequate blood flow to the CNS and to the other vital tissues?

A significant compromise in any one of these three competing demands will lead to the cessation of exercise, collapse, and/or a potentially fatal heat stroke (Sutton & Thompson 1998).

Is exercise in the heat limited by a "critical core temperature"?

The "critical core temperature" hypothesis is centered on an elevated body temperature providing peripheral afferent feedback from temperature-sensitive sites to the CNS, resulting in reduced central neural drive to the exercising muscles. This hypothesis proposes that the critical core temperature for reduced CNS activation of the exercising muscles may vary among individuals and may be an inhibitory protective mechanism against potentially lethal increases in body temperature. Testing the critical core temperature hypothesis is fraught with difficulty as there are ethical considerations in exercising subjects to imminent collapse associated with high body temperatures. Further, subjects need to be highly motivated to endure elevated body temperatures and the associated circulatory strain for researchers to be confident that an

impaired performance in hot environmental conditions is a consequence of reduced CNS drive.

The studies supporting this hypothesis (Todd et al. 2005; Gonzalez-Alonso et al. 1999a,b; Nielson et al. 1993) are laboratory based and invariably report body temperatures following exercise-induced hyperthermia that are lower than those reported on athletes at the conclusion of endurance events (Hoffman et al. 1999; Pugh et al. 1967; Robinson 1963). Whether a cause–effect relationship exists between an elevated core temperature and reduced CNS activation of exercising muscle is debatable.

Nielson et al. (1993) reported the observation that during the course of a heat acclimation program lasting 9–12 consecutive days, exhaustion coincided with an esophageal temperature of 39.7 ± 0.15°C in eight "well-trained" subjects performing seated cycling exercise (50% of estimated $\dot{V}O_{2max}$ for 48–80 min) in hot conditions (40–42°C and 10–15% relative humidity). However, they failed to demonstrate a reduced central neural drive as the subjects were able to fully activate the muscles (quadriceps) that had worked to "exhaustion" when they subsequently performed a post-exercise maximal voluntary isometric contraction (MVIC). The authors concluded that "physical endurance for exercise in hot, dry environments appears to be limited by the attainment of a critical level of core temperature, perhaps due to temperature, reducing motivation". It is difficult to reconcile the reduced motivation explanation with the reported small increase in MVIC of the quadriceps and no change in the MVIC of the elbow flexors following exhaustion in the hot condition.

In a further study from the same laboratory, Gonzalez-Alonso et al. (1999a,b) reported that a group of seven cyclists reached exhaustion at the same esophageal temperature of 40.2 ± 0.3°C when exercising at 60% $\dot{V}O_{2max}$ in hot conditions (40°C and 17% relative humidity) despite experimental manipulation of their body temperature (immersion in water at 17°C, 36°C, and 40°C) prior to exercise. The authors concluded that "fatigue during exercise in the heat was related to high internal body temperature". Close scrutiny of Gonzalez-Alonso et al.'s (1999a,b) results reveals that the HR at exhaustion was 99% HR_{max} in all three conditions, yet the authors did not propose this extreme circulatory strain to have limited exercise performance. These researchers concluded that "high body temperature *per se* causes fatigue in trained subjects during prolonged exercise in uncompensable hot environments". Since SV and CO were significantly lower in the preheated condition one could speculate that exercise was terminated when CO could no longer be maintained with HR virtually at maximum.

More promising evidence of a CNS role in the fatigue shown by hyperthermic subjects has been reported by Nybo et al. (2001) who used a superimposed electrical stimulus on an MVIC sustained for 2 min to determine whether full activation of the muscle was achieved. They found that under both control and hyperthermic conditions, superimposed electrical stimulation elicited additional force during the sustained MVIC of the quadriceps. This suggests a reduced capacity to voluntarily activate the quadriceps, a suggestion which is further supported by a reduced rectified integrated surface electromyography (iEMG) recording. However, there was no difference in the total force (MVIC plus electrical stimulation) when a brief MVIC was performed in both control and hyperthermic conditions. Thus, these results do not differentiate whether the subjects could not or would not fully activate the leg extensors during the sustained MVIC.

Conflicting observations have since been reported by White et al. (2002) who used passive heating (hot water immersion) to induce hyperthermia in five highly trained endurance athletes. Passive hyperthermia was achieved with hot water immersion that elevated rectal temperature to 39.52 ± 0.27°C and muscle temperature to 40.26 ± 0.30°C. The alternate leg was not immersed in the hot water, and thus the muscle temperature was more than 3.0°C cooler than the heated leg. Superimposed electrical stimulation and EMG were used to determine the level of muscle activation during a sustained 90 s MVIC of the triceps surae. The results indicated a small progressive decrease in the activation level of the calf muscles, but this did not exceed 15% in the hyperthermic-hot leg condition compared with 46% in the quadriceps reported by Nybo et al. (2001). The study by Nybo et al. (2001) reported a "core" temperature of 39.7°C and a muscle temperature of 40.7°C at the conclusion of the sustained MVIC, whereas the corresponding temperatures reported by White et al. (2002) were

taken immediately before the subjects undertook the sustained MVIC. Nevertheless, it is difficult to reconcile similar core and muscle temperatures reported in both studies resulting in such a profound difference in voluntary activation, although the sustained MVIC in Nybo et al.'s (2001) study was 30 s longer in duration. The explanation for the disparity in these results may arise as a consequence of differences in hyperthermia elicited via exercise and passive heating, as well as the muscle groups tested.

The sustained MVIC fatigue test requires considerable motivation to elicit a maximal effort throughout the duration of the test. If the motivation of the subject is compromised, then superimposed electrical stimulation will produce additional force to that attained by voluntary effort alone. Under these circumstances the researcher may conclude that there is evidence of central fatigue. The subjects in Nybo et al's. (2001) study showed a reduced capacity to fully activate the quadriceps (~18%) during the sustained MVIC in the control condition. Their performance of the same fatigue test under hyperthermic conditions further reduced the capacity to fully activate the quadriceps. This observation raises the question whether there are different afferent signals inhibiting CNS drive to the muscle under control and hyperthermia conditions.

Several researchers have recently proposed an anticipatory reduction in CNS drive during prolonged exercise in the heat as a protective mechanism (Marino 2004; Tucker et al. 2004). Tucker et al. (2004) observed a reduced power output and decline in iEMG activity during prolonged (20 km) self-paced cycling exercise in the heat, while rectal temperature and HR remained elevated. Whether this is a controlled regulatory response to protect against an excessive thermal load is a moot point. The so-called down regulation of CNS activation was first apparent when the elevation in rectal temperature was approximately 38°C (Tucker et al. 2004). Further, the subjects increased the power output by 80 W (~220 W to ~300 W) over the final 20% of exercise time in completing the 20-km cycle exercise. It is difficult to link a relatively modest increase in rectal temperature signaling an inhibition of CNS drive to the exercising muscle, let alone contend with a subsequent significant increase in power over the latter stages of the 20-km cycle time trial. When subjects are able to dictate the intensity of exercise, it requires highly motivated individuals to sustain intense prolonged exercise in hot conditions in a laboratory setting. Bruck and Olschewski (1986) have proposed that a high core temperature in some way affects the brain by reducing motivation. Perhaps these results reflect waning motivation or alternatively the explanation may be related to the circulatory strain experienced.

Recently, Todd et al. (2005) used transcranial stimulation of the motor cortex to investigate brief and sustained MVICs of the elbow flexors under passively induced hyperthermia (esophageal temperature ~38.5°C) and control conditions. Their results show a very small decline (~2.4%) in a brief 1–2 s MVIC following hot water immersion; however, a 12% decline in voluntary activation was observed with a 2-min sustained MVIC. It is difficult to reconcile the impaired muscle function reported with such a moderate increase in body temperature. Nevertheless, Todd et al. (2005) concluded that part of the failure in voluntary activation was at or above the level of motor cortical output, while the remaining central component was a consequence of temperature-related changes in the contractile properties of muscle. They found that the muscle relaxation rate increased by ~20% during hyperthermia, thus requiring faster motor unit firing rates to produce fusion. This is a particularly interesting finding that warrants further investigation. Thus, the etiology of weak hot muscles may be an altered contractile state that exceeds the activation rate capacity of the CNS.

While faster muscle peak relaxation rates may contribute to a CNS limitation under hyperthermia conditions, there are indications of a direct effect of exercise-induced hyperthermia on brain function. Prolonged exercise in a hot environment can lead to altered electroencephalographic (EEG) activity (Nielsen et al. 2001), reduced blood velocity in the middle cerebral artery (Nybo et al. 2002; Nybo & Nielsen 2001b), and altered brain metabolism (Gonzalez-Alonso et al. 2004; Nybo et al. 2002). However, the connection between these changes and the cessation of prolonged exercise in the heat is tenuous.

Several animal studies have reported that fatigue during prolonged exercise in the heat coincided with a similar core temperature despite differences in

experimental protocols (Walters et al. 2000; Fuller et al. 1998; Caputa et al. 1986). Caputa et al. (1986) found that elevating the temperature of the brain in goats exercising on a treadmill reduced the work rate, while elevating the temperature of the trunk had no effect on work rate even when elevated to 43.5°C for 1 h of walking. In the study by Fuller et al. (1998), rats were run on a treadmill under three different rest-exercise ambient temperatures, resulting in significant differences in exercise time to fatigue; yet the core temperature was the same at the conclusion of each exercise bout. The study by Walters et al. (2000) passively heated rats to three different core temperatures prior to running on a treadmill in 35°C ambient temperature to exhaustion. The core and hypothalamic temperatures at exhaustion were not significantly different across the treatments.

Thus these three animal studies (Walters et al. 2000; Fuller et al. 1998; Caputa et al. 1986) indicate that prolonged exercise in hot conditions is limited when core temperature is elevated to a critical degree, irrespective of the rate of heat storage or the duration of the exercise. Whether the elevated core temperature affects a peripheral or central fatigue outcome is unclear.

Summary

There is no dispute that prolonged exercise in the heat is limited by an early onset of fatigue. There is little evidence to implicate an accumulation of metabolites or depletion of energy-yielding substrate as a causative factor in the cessation of exercise. Recent studies have suggested that a reduced CNS activation of the active muscles occurs when a critical core temperature is attained during exercise in the heat. Both superimposed electrical stimulation in conjunction with voluntary contractions and EMG have been used to determine the extent of neural activation. However, the results may reflect a conscious reduction in voluntary effort rather than central fatigue. When brief MVICs are performed in a hyperthermic state, there is no apparent central fatigue component evident. However, when sustained MVICs are performed there is evidence of reduced CNS activation in both control conditions and when hyperthermia is induced either through passive heat exposure or as a consequence of exercising in the heat. More studies on well-trained, highly motivated endurance athletes is warranted to clearly establish the underlying mechanism(s) between the elevation of core temperature and reduced exercise performance in the heat.

It is of interest to note that Gisolfi and Mora (2000) concluded in their book, *The Hot Brain*, that the issue of what limits performance in the heat remains unresolved, pointing out that the arguments put forth by both Nielsen (1993; 1990) and Rowell (1986) were equally compelling.

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