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FRONT MATTER: DISCOVERY

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Hyperthermia during exercise – a double-edged sword*

Comment on: Buono MJ, et al. Increases in core temperature counterbalance effects of haemoconcentration on blood viscosity during prolonged exercise in the heat. Ex Physiol 2016; 101:332-342; http://dx.doi.org/10.1113/EP085504

An excessive elevation in body temperature is known to decrease exercise performance. The physiological mechanisms involved with hyperthermia-induced fatigue include central nervous system dysfunction and cardiovascular instability. Conversely, a moderate increase in body temperature has been shown to actually improve exercise performance. Asmussen and Boje[1] reported that sprint performance improved by approximately 5% for each 1°C increase in muscle temperature. The improvement was attributed to the well-known Q_{10} temperature effect on numerous biochemical processes. However, are there other potential benefits of hyperthermia during exercise?

Recently we published findings that suggest another possible benefit of moderate hyperthermia during exercise, namely improved blood viscosity.[2] This is important as blood viscosity plays a critical role in limiting exercise capacity and is significantly correlated to maximal oxygen uptake.[3] It is inevitable that during exercise there is an increase in hematocrit, induced by several mechanisms including fluid shifts due to increases in blood pressure, dehydration, and water trapping in working skeletal muscle. The exercise-induced hemoconcentration has traditionally been viewed to have both positive and negative consequences as it simultaneously increases both oxygen carrying capacity and blood viscosity. In fact, over the last 30 y at least 10 studies have reported that various forms of exercise increase blood viscosity by between 5 to 15%. However, in an effort to isolate the effect of increases in hematocrit on blood viscosity, they measured both the pre- and post-exercise blood samples at 37°C. This discounts the fact that exercise almost always elevates core and blood temperature. Thus, past studies that reported an increase in blood viscosity following exercise did not replicate real *in vivo* conditions and therefore likely missed any affect that exercise-induced hyperthermia may have had on mitigating change in blood viscosity.

The results of our recent study challenge the dogma that exercise increases blood viscosity. Specifically we found that exercise-induced hemoconcentration significantly increased blood viscosity by 9%. This finding agrees with all of the past studies. However, we also found that exercise-induced hyperthermia significantly decreased blood viscosity by 7%. When both factors were considered together, there was no overall change in blood viscosity. Thus, the effects of hemoconcentration and hyperthermia counterbalanced each other so that there was no change in blood viscosity, at a given shear rate, following prolonged, moderate intensity exercise in the heat. Therefore, we suggest that moderate hyperthermia during exercise may be beneficial because it attenuates the increase in blood viscosity brought about by hemoconcentration. Our results suggest that hyperthermia decreases blood viscosity by increasing red blood cell deformability. The exact mechanism is currently unknown; however increased temperature may result in more spontaneous dissociations of the spectrin network where in junctions with the phospholipid bilayer. This reduced tension within the cytoskeleton could ultimately decrease membrane stiffness and improve red blood cell deformability.

Our results may also help to explain why past findings[4] have reported that a high hematocrit decreases blood flow much more in resting muscle than in exercising muscle. It could be theorized that the blood flow preserving effect of muscle contractions reported in these studies was at least partially due to a hyperthermia-induced reduction in blood viscosity in the exercising muscle.

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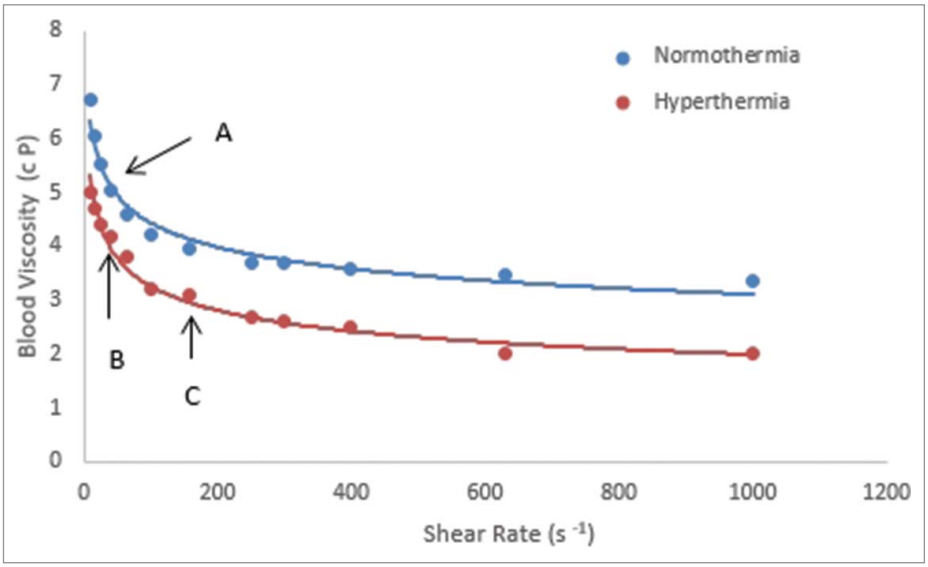

Figure 1. The blood viscosity vs. shear rate relationship.

Lastly, taking our results a step further suggests that during exercise *in vivo* blood viscosity may actually decrease. To envision this concept 2 facts must be integrated. First, it must be remembered that blood is a shear-thinning fluid and its viscosity is inversely related to shear rate (i.e. blood velocity), as shown in Figure 1. Second, to increase oxygen delivery to working skeletal muscles, cardiac output increases as does shear rate of the blood during exercise. Coupling these facts with the results of our study suggests that the combined effects of exercise-induced hyperthermia (point A to point B in Fig. 1) and increased shear rate (point B to point C) could theoretically decrease *in vivo* blood viscosity by 30% (point A to point C), despite the typical increase in hematocrit seen during exercise.

In conclusion, we have demonstrated that when hyperthermia was accounted for by measuring the post-exercise blood sample at the actual *in vivo* core temperature and not at a set temperature, as past studies have done, there is no change in blood viscosity during prolonged, moderate-intensity exercise in the heat. Thus, we suggest that moderate hyperthermia during exercise may be beneficial because it attenuates the increase in blood viscosity brought about by hemoconcentration. This may improve muscle blood flow at a time in which the body is metabolically active.

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