Non-Thermal Influences on the Control of Skin Blood Flow Have Minimal Effects on Heat Transfer During Exercise

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During exercise, circulatory reflexes ensure that the cardiac output is sufficiently elevated to meet the oxygen delivery requirements of the contracting skeletal muscles and the heat delivery requirements of the body to the skin. The latter requirements are met by increasing skin blood flow. These increases are largely driven by elevations in the body temperatures, although non-thermal effects on the control of skin blood flow occur in certain conditions. These effects are largely the consequence of high and/or low baroreflex stimulation. Even in the face of such non-thermal effects, which occur during exercise in the heat, the body’s requirements for heat transfer from core to skin are largely met by the increased skin blood flow. Thus, non-thermal effects on the control of skin blood flow are relatively unimportant in the body’s overall regulatory response to exercise.

Exercise is a unique state in which many of the body’s regulatory systems are activated to reduce the resultant physiological strain. Circulatory reflexes ensure that the cardiac output is sufficiently elevated to meet the oxygen delivery requirements of the contracting skeletal muscles and the heat delivery requirements of the body to the skin. Even during heavy exercise, when the external thermal load is not great, these regulatory mechanisms are able to meet the simultaneous demands for increased blood flow to muscle and to skin, and the individual is able to maintain a metabolic and thermal steady state for extended periods [1,2]. During heavy exercise in a hot environment [2], or during moderate exercise in underhydrated conditions [3], however, we have previously observed an inability to attain a thermal steady state and have attributed this inability to a modification in the control of skin blood flow [3]. This hypothesis would appear to support the notion that non-thermal effects are important in the control of cutaneous vascular resistance during exercise. While acknowledging that non-thermal effects exist, I will argue that they are of relatively small importance in the overall control of skin blood flow, and hence of the ability to transfer heat from the body core to the skin during exercise.

HEAT TRANSFER DURING EXERCISE

All of the energy released as a by-product of metabolism must eventually be accounted for as thermal energy or as physical work. The metabolic rate, and therefore the generation of heat, in resting skeletal muscle is low, on the order of 0.01 kcal · min⁻¹ · kg⁻¹. During moderate to heavy exercise, the metabolic rate in skeletal muscle can increase 100-fold, to about 1 kcal · min⁻¹ · kg⁻¹, or 20 kcal · min⁻¹ if we assume the involvement of 20 kg of muscle. This new rate of thermal energy production first
causes muscle temperature to rise, initially at a rate proportional to the rate of energy production, and this rise may be in excess of 1.0 °C · min⁻¹ over the first minutes of heavy exercise [4]. The precipitous rate of muscle temperature rise is attenuated as the rate of heat transfer from the muscle to the blood perfusing the muscle increases, due to (a) an increased temperature gradient from muscle to blood and (b) an increased rate of muscle blood flow (see [5] for a more detailed description of the factors governing the rate of heat transfer in such conditions). The excess heat is then carried into the body core by convection in the venous blood draining the contracting muscles. Thermal sensors in the body core, particularly in the hypothalamus, respond to an increased core temperature and reflexly stimulate the body’s heat dissipation activities. When internal body temperature exceeds a specific threshold, which itself is not fixed but depends upon a number of factors (e.g., the average skin temperature), the rates of skin blood flow and of sweating are increased in a manner proportional to the increase in core temperature [6]. Increased skin blood flow promotes an increase in the rate of convective heat transfer from the body core to the skin, from which the heat is readily eliminated to the environment by convection and radiation, down the skin-to-environment temperature gradient, and by evaporation, down the skin-to-environment water vapor pressure gradient. Since the storage of only about 60 kcal increases the body temperature of an average-size adult by 1.0 °C, it can easily be seen that the increases in skin blood flow and sweating rate, driven primarily by the rise in internal temperature, are essential during exercise to enable the transfer and dissipation of the thermal energy produced. Otherwise, exercise would be limited to relatively short periods, because body core temperature would rise to a level at which both central nervous and circulatory problems would begin to appear.

**THERMAL CONTROL OF SKIN BLOOD FLOW DURING EXERCISE**

Robinson [6], using computations of skin thermal conductance from measurements of the changes in body temperatures, gave the first adequate description of the physiological control of the skin circulation during exercise. It followed from M. Nielsen’s earlier observation (that the absolute core temperature rise during exercise was proportional to the exercise intensity [7]), that the rate of heat transport from core to skin was elevated to a new steady level which balanced the rate of heat production. However, Robinson’s description was novel in that it presented skin conductance as a function of internal (rectal) temperature, i.e., an increased core temperature caused a proportional increase in the transport of heat from core to skin. Years later, using techniques that enabled more reliable assessments of both skin blood flow and internal temperature, Wenger et al. [8] confirmed the basic description of Robinson and constructed a mathematical model that described the relation:

\[ BF = \alpha T_{es} + \beta T_{sk} + \Gamma |BF > 3.0| \]

where \( BF \) = forearm blood flow in ml · min⁻¹ · 100 ml⁻¹ and minimum \( BF = 3.0 \) ml · min⁻¹ · 100 ml⁻¹

\( T_{es} \) = esophageal temperature in °C

\( T_{sk} \) = average skin temperature in °C

\( \alpha, \beta \) = proportional control constants in ml · min⁻¹ · 100 ml⁻¹ · °C⁻¹

\( \Gamma \) = intercept constant in ml · min⁻¹ · 100 ml⁻¹

The values for \( \alpha \) and \( \beta \) were 8.5 and 1.3 ml · min⁻¹ · 100 ml⁻¹ · °C⁻¹, respectively, indicating that changes in internal temperature were considerably more important
than comparable changes in skin temperature in driving the skin circulation. It is important to point out that the studies of Wenger and colleagues were conducted on volunteers exercising at moderate intensities on a cycle ergometer in the semi-upright position.

With such a model, one can better understand the dynamics of heat transfer to the skin surface, as well as from the body, and the development of a new, elevated-state internal body temperature during exercise. The internal temperature is the primary determinant of an elevated skin blood flow (as well as of sweating rate [9]); mean skin temperature, which is fixed within a narrow range by the environmental temperature, determines the internal temperature threshold for increased skin blood flow. The proportional control constant determines the “rapidity” of the skin blood flow response to increases in core temperature and, therefore, the time necessary for the rate of heat transfer from core-to-skin to equal the rate of heat transfer from muscle-to-core (which itself is a function of the rate of energy production). If the rate of heat transfer from skin-to-environment is not limited (by factors such as high ambient humidity, for instance), a new steady state in internal body temperature will occur when the new rates of heat transfer are equivalent.

**NON-THERMAL INFLUENCES ON THERMAL CONTROL**

It is now well recognized that the skin circulation is the shared effector organ of two regulatory systems, the temperature regulatory system and the blood pressure regulatory system. Major changes in skin vascular resistance can be induced by body heating and/or by changes in the blood pressures. The skin vasculature has a great capacity to increase flow; in supine, resting humans exposed to a high ambient temperature, cardiac output and, by inference, the blood flow to the entire skin can be increased by 6 to 7 liters min⁻¹ [10]. In upright humans during exercise, the heart is not able to provide this magnitude of flow to the skin while maintaining the necessary flow to the contracting muscles. Nonetheless, in most conditions of exercise and ambient temperature, the heart is able to deliver adequate flow to both muscle and skin, thereby satisfying the demands for oxygen delivery to the former and heat delivery to the latter. The major determinant of blood flow to muscle is the oxygen demand, created by the exercise intensity. In cool or thermoneutral conditions, cardiac output and muscle blood flow are both roughly proportional to the metabolic rate; in such conditions the heart has no difficulty meeting the requirements for blood flow to the skin, as dictated by the increases in the body temperatures.

Exposure to a high environmental temperature during exercise places an increased demand for heat transfer on the body. Using the model described above, the elevated skin temperature serves to shift the threshold for cutaneous vasodilation to a lower internal temperature. Accompanying the greater skin blood flow at any combination of body temperatures in such conditions is an increased volume of blood in the skin, especially in veins below heart level. An increased skin blood volume reduces the central circulating blood volume, and in conjunction with other factors that tend to do the same (e.g., increased filtration of plasma water from the vascular compartment [11] and increased water loss from the body by evaporation of sweat), there occurs a reduction in cardiac filling pressure and, in keeping with Starling’s law of the heart, a decrease in cardiac stroke volume. In such conditions, the first line of defense in order to maintain cardiac output is cardioacceleration. Rowell et al. [12] found that cardioacceleration (by 20 beats min⁻¹) was not adequate to sustain cardiac output
during treadmill (upright) exercise in the heat. However, we [2] found that if subjects exercised in the seated position, this amount of cardioacceleration was sufficient to maintain cardiac output.

Reflexes that act to attenuate the fall in cardiac filling pressure during exercise in the heat are those that divert blood flow away from organs in which a high perfusion rate is not essential. A decrease in blood flow to an organ reduces the volume of blood contained in that organ and restores this volume to the central circulation. Rowell et al. [13] showed that splanchnic blood flow is reduced in proportion to the exercise intensity and, furthermore, that exposure to heat shifts the threshold for reduced splanchnic flow toward lower exercise intensities. We [2] and others [14] have shown that there is a relative cutaneous vasoconstriction that is superimposed upon the vasodilatory drive associated with the high body temperatures produced during exercise in the heat (Fig. 1). This effect would qualify as a non-thermal influence on the control of skin blood flow. The reflex constriction of the skin vasculature acts to shift blood centrally so that, at the point of cutaneous vasoconstriction, the progressive decline in cardiac stroke volume during moderate exercise in the heat ceases (Fig. 2). This reflex is important in that it contributes to the body's ability to maintain cardiac filling and arterial blood pressures in the face of events that would otherwise lead to a decompensation of the circulation. However, whether this reflex is an important non-thermal reflex, affecting the ability to transfer heat in the cutaneous circulation, is debatable. I will return to this.

A second and even more impressive non-thermal factor affecting the control of skin blood flow is that induced by hypovolemia. During prolonged exercise in a hot environment, hypovolemia will develop as a consequence of the considerable water loss due to evaporation. This water must be derived from all body compartments, including the vascular compartment. In order to characterize those circulatory reflexes that
compensate for the effects of hypovolemia, we [3,15] rendered volunteers hypovolemic by the use of diuretics, taken over a three-day period. This regime resulted in a blood volume contraction of 10 to 11 percent and is associated with a dramatic reduction in cardiac stroke volume (as well as in cardiac output) during moderate exercise in the heat [15]. Hypovolemia induces a marked increase in the internal temperature threshold for cutaneous vasodilation. This increase is on the order of 0.5 °C; a consequence of this shift is that hypovolemic people store heat at a faster rate than they would during comparable conditions when they are normovolemic (Fig. 3). Second, there is a 30 percent reduction in the maximal skin blood flow in hypovolemic conditions (also shown in Fig. 3). Thus, the maximal rate of heat transfer from core to
skin is reduced. Although heat transfer is lower in hypovolemia than in normovolemia during exercise in the heat, both of these circulatory adjustments serve to maintain cardiac stroke volume by reducing the volume of blood residing in the periphery [15]. During moderate exercise in the heat, signals related to a decreasing central circulating blood volume (presumably, these signals are triggered by falling arterial and/or cardiac filling pressure) stimulate an overriding of the normally occurring cutaneous vasodilator drive from the thermoregulatory system.

**IMPORTANCE OF NON- THERMAL INPUTS**

That non-thermal influences affect the control of the skin vasculature is clear. Dramatic changes in the control of skin blood flow are demonstrated in Fig. 3, and these changes contribute to a reduced heat transfer and produce an increased body core temperature in the hypovolemic condition. During 30 minutes of exercise, body core temperatures increase by about 0.4 °C higher in the hypovolemic as compared to the normovolemic condition, equivalent to a heat storage of approximately 50 kcal · hour⁻¹. Relative to the metabolic heat production in such conditions (around 700 kcal · hour⁻¹) this excess storage of 50 kcal · hour⁻¹ in hypovolemic conditions does not appear to be so large. Even considering that up to 25 percent of this thermal energy may have appeared as physical work (accounted for as heat transferred directly to the flywheel of the cycle ergometer), the difference in heat transfer between normovolemic and hypovolemic conditions was less than 10 percent, despite the seemingly large upward shift in internal temperature threshold for cutaneous vasodilation and decrease in maximal forearm blood flow from 18.6 to less than 13 ml · min⁻¹ · 100 ml⁻¹. In other words, an examination of the body's ability to transfer heat from core to skin via the cutaneous circulation reveals that the non-thermal effect (due to hypovolemia) has relatively little influence on skin blood flow during exercise.

An extension of this argument is that the body may be “overperfusing” the skin (relative to its heat transfer needs) in normovolemic conditions. Since the rate of heat transfer is the product of the heat flow to the skin (skin blood flow) and temperature gradient between core and skin, it follows that we can achieve the appropriate heat flux by changing both variables simultaneously. In conditions in which skin blood flow is reduced, the resultant increase in body core temperature (and the core-to-skin temperature gradient) will therefore maintain an appropriate heat transfer rate. Thus, it can be stated that non-thermal effects on the control of skin blood flow are not of great importance to the body's overall ability to transfer heat from core to skin during exercise.

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