Non-Thermal Factors Are Important in the Control of Skin Blood Flow During Exercise Only Under High Physiological Strain

C. BRUCE WENGER, M.D., Ph.D.

U.S. Army Research Institute of Environmental Medicine, Natick, Massachusetts, and Harvard University School of Public Health, Boston, Massachusetts

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Several authors have argued that skin blood flow (SkBF) during exercise is less than during rest at the same levels of body core and whole-body skin temperatures ($T_c$ and $T_w$). Since such an effect does not prevent SkBF during exercise from rising above pre-exercise levels, it is sometimes called a relative cutaneous vasoconstriction. Such a vasoconstriction is considered to be either part of a thermoregulatory adjustment during exercise (elevated thermoregulatory "set-point") or a compensatory response to allow adequate perfusion of exercising muscle. In this paper, some of the pertinent experimental evidence is reviewed, and the following conclusions are reached: (a) the evidence does not support a change in thermoregulatory set-point during exercise; (b) under conditions of high physiological strain (high $T_w$ and intense exercise), there is quite clearly a relative cutaneous vasoconstrictor effect of exercise; (c) the evidence does not support such an effect under more moderate conditions; and (d) it is likely that, under mild to moderate conditions, other compensatory cardiovascular responses are sufficient to allow adequate perfusion of exercising muscle and are invoked in preference to relative cutaneous vasoconstriction, which has been demonstrated only at higher levels of strain. The thermoregulatory SkBF required during sustained exercise is thus maintained as much as possible.

Skin blood vessels participate in a number of non-thermoregulatory reflexes, not all of which are well understood, but some of which may participate in responses to exercise. There is little doubt that exercise under certain conditions is associated with changes in skin blood flow that cannot be explained by changes in body temperatures. Since the purpose of this symposium is to debate controversial issues in thermal physiology, it will be useful to begin this article by defining the terms of the discussion so as to identify and focus on the points of controversy concerning cutaneous vascular responses during exercise. Following Rowell [1], I shall begin by asking whether skin blood flow during exercise is different from what we should expect during rest at the same levels of the relevant body temperatures, which I shall take as skin and body core temperatures. This means that we are considering not the effects of individual non-thermal reflex loops, but the algebraic sum or net effect of all such loops that participate in the response to exercise, and it implies further that exercise is to be compared to rest under otherwise similar conditions, so that factors that affect resting subjects as much as exercising ones, but are not produced by the exercise itself, are not under discussion here. Thus, for example, we will consider responses to exercise-related

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Address reprint requests to: Dr. C. Bruce Wenger, USARIEM, Kansas St., Natick, MA 01760-5007

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changes in intravascular pressure, but not, say, effects of circadian rhythms, which seem to affect thermoregulation in the same way during rest and exercise [2].

A non-thermal effect of exercise need not be independent of the thermal state of the subject, and we cannot say a priori how it is related to exercise intensity. A second stipulation, therefore, is that rest should be compared to exercise over a range of core and skin temperatures and exercise intensities. Much of the least arguable evidence for non-thermal effects on skin blood flow during exercise comes from experiments conducted under conditions of fairly severe thermal and cardiovascular strain, such as whole-body skin temperature ($T_{sk}$) of about 38°C, or exercise near maximal $O_2$ consumption ($V_{O_2}$. max). It is not entirely clear whether, on the one hand, such non-thermal effects are characteristic only of exercise under fairly severe conditions and have little importance for exercise under mild to moderate conditions, or whether, on the other hand, they are characteristic of exercise in general, but because of experimental limitations are most easily demonstrated under relatively severe conditions (cf. [3]). It therefore seems most appropriate to focus attention on exercise under mild to moderate conditions, that is, at exercise levels reasonably below maximum and $T_{sk}$ well below, say, 36°C, and without the addition of other severe stresses, such as exhaustion or major disturbances in blood volume or osmolality. In view of the foregoing, I will consider the question “Is skin blood flow during exercise in conditions of mild to moderate physiological strain different from what we should predict from the same levels of body core and skin temperatures during rest”?¹

**EFFECT OF EXERCISE ON THERMOREGULATORY RESPONSES**

The notion that exercise alters the way the body responds to thermal stress has been around for a long time. In 1938, Marius Nielsen [6] reported that over a fairly wide range of ambient temperatures ($T_a$) the increment in body core temperature ($T_c$) at steady state during exercise is proportional to exercise intensity, but does not depend markedly on $T_a$. These observations have been confirmed many times and have been extended by the finding that the rise in $T_c$ during exercise is more closely related to the relative exercise intensity (i.e., the fraction of the subject’s $V_{O_2}$. max) than to the absolute intensity [7]. These observations have been taken to indicate that exercise raises the thermoregulatory “set-point” by an amount proportional to the exercise intensity. More recent studies have shown that the heat-dissipating responses, viz., sweat rate and peripheral blood flow, are approximately linearly related to $T_c$, demonstrating the proportional control nature of the thermoregulatory system. The upper panel of Fig. 1 shows how a set-point elevation would affect the control of a heat-dissipating response, R. The $T_c$ threshold for R is elevated in proportion to

¹Nearly all of our knowledge of the control of SkBF is based on measurements of ABF and FBF, because of difficulties in measuring SkBF on the trunk and head. However, FBF and ABF may not be quantitatively reliable indices of SkBF in other regions. Blair et al. [4], measuring changes in regional skin temperatures during whole-body heating and cooling, concluded that vasoconstrictor innervation is less important in the thermoregulatory control of SkBF in the trunk and head than in the limbs. Although their findings need not mean that vasoconstrictor influences are less important in the responses of trunk and head SkBF to other stimuli, such an interpretation is supported by the findings of Kenney et al. [5]. They reported that healthy hypertensive patients showed far less increase in ABF than did controls during cycle exercise, even though their $T_a$ and their increase in $T_a$ were the same as the controls', indicating that the patients achieved nearly normal increases in heat loss, core-to-skin thermal conductance, and presumably also SkBF over most of the body.
exercise intensity, and at any $T_c$ above threshold, $R$ is lower at higher exercise intensity.

In particular, if the response $R$ is skin blood flow (SkBF), then—since SkBF is lower during exercise than it would be during rest at the same $T_c$—we may say that there is a relative cutaneous vasoconstrictor effect of exercise, even though, as exercise continues, SkBF will probably rise higher than it was before exercise began. Generalizing slightly, we may speak of a relative vasoconstrictor effect of exercise whenever SkBF is lower than it would be during rest at the same $T_c$ and $T_{sk}$, whether such an effect is brought about by a set-point elevation or otherwise. For convenience, I shall often refer to a relative cutaneous vasoconstrictor effect simply as a vasoconstrictor effect.

Some increase in $T_c$ during exercise is a necessary consequence of the proportional control nature of the thermoregulatory system, since, in the face of a thermal load, a certain increase in $T_c$ above threshold (a so-called “load error”) is necessary to elicit heat-dissipating responses sufficient to restore thermal balance. The observed steady-state increase in $T_c$ during exercise is thus the algebraic sum of the set-point shift, if any, and the load error. For this reason we can reconcile the rise in $T_c$ during exercise with an elevation, a lowering, or no change of thermoregulatory set-point during exercise, by appropriate choice of sensitivity for the response of effector mechanisms to changes in $T_c$ (Fig. 1). Indeed, all of these possibilities have been proposed. (Curiously, although a fair number of authors have proposed that exercise causes a lowering of the thermoregulatory set-point, which implies a relative vasodilator effect of exercise, all discussion of non-thermal effects of exercise on SkBF seems to be concerned with vasoconstrictor effects.) Thus, though possible, a set-point shift is not established (see [3] for further discussion), and it is necessary to look for other evidence of a relative vasoconstrictor effect of exercise.

Since SkBF serves other functions in addition to thermoregulation, a change in thermoregulatory set-point is not the only mechanism by which exercise might alter
the control of SkBF. A true set-point shift would imply corresponding shifts in the thresholds for all thermoregulatory responses [3], but for the present purpose, we are concerned with shifts in the relation of SkBF to $T_c$ (at a given $T_{sk}$), regardless of whether or not control of other thermoregulatory responses (e.g., sweating) is altered in the same way. An interesting recent variant of the hypothesis of an elevated set-point during exercise is Rowell's suggestion [1] that perhaps "the elevation in $T_c$ during exercise may be the result of a high background bias of sympathetic nervous activity that reduces SkBF in order to maintain mean arterial pressure . . . this might explain, for example, why $T_c$ is more closely related to the percent of $V_{O_2\ max}$ required during exercise (and thus to total sympathetic nervous activity) than to absolute heat production."

**EFFECT ON SkBF OF EXERCISE WITH HIGH CARDIOVASCULAR STRAIN**

Most experimental evidence for a relative vasoconstrictor effect of exercise consists of observations made under somewhat extreme conditions, such as high $T_{sk}$ or high exercise intensities. John Johnson and his colleagues [8,9] compared the relations of forearm blood flow (ABF) to esophageal temperature ($T_s$) during exercise and rest in subjects whose skins (except for their heads and the forearms on which ABF was measured) were heated to 38°C. Use of such high $T_{sk}$ allowed them to drive $T_s$ to fairly high levels, even in resting subjects. Exercise shifted the threshold for vasodilation toward higher $T_s$ (equivalent to a relative cutaneous vasoconstriction), and sometimes also reduced the slope of the ABF:$T_s$ relation. Although these experiments represent an ingenious solution to the problem of obtaining ABF:$T_c$ relations over similar ranges during both rest and exercise, their findings may have little bearing on cutaneous vasomotor responses during exercise at lower $T_{sk}$. In addition to the reflex vasodilator effect of high $T_{sk}$, skin heating causes vasodilation by a direct action that is independent of nervous signals (Fig. 2). Extending the effect shown in Fig. 2 to the skin of the whole body, we can compute that the direct effect of heating the skin to 38°C can increase SkBF by 2.5 l/minute, in addition to the reflex effect of skin heating. Since such a degree of skin heating is likely also to impair vasoconstrictor and venoconstrictor responses in the heated skin, the subjects participating in those experiments must have had much more peripheral pooling of blood than they would have had with cooler skins. A major function of cutaneous vasoconstriction seems to be to limit peripheral pooling of blood and defend cardiac filling (see discussion following). In the face of so much peripheral pooling, adequate perfusion of skeletal muscle during exercise may have required compensatory adjustments that are not manifested at lower $T_{sk}$.

Other arguments for a cutaneous vasoconstrictor effect of exercise are based on evidence that, even at more moderate $T_{sk}$, SkBF during heavy exercise is less than during lighter exercise or at rest. Rowell [10] points out, for example, that $V_{O_2\ max}$ is not reduced by levels of $T_s$ and $T_c$ which at rest are associated with elevated SkBF. (At still higher temperatures, however, $V_{O_2\ max}$ is compromised.) This finding implies that, during maximal exercise, a warm subject can reduce SkBF to levels prevailing in cooler conditions, unless in such cooler conditions the cardiovascular system can deliver more oxygen than the muscles can consume. Rowell [1] makes another similar argument for a cutaneous vasoconstrictor effect of exercise, based on previous observations which his group made on men performing fairly intense treadmill
exercise. They reported [11] that, at any exercise intensity, cardiac output was lower at $T_a$ of 43.3°C than at 25.6°C, and the difference was more pronounced at higher exercise intensities. Rowell interpreted this finding to indicate that the cutaneous vasodilation expected from the higher $T_a$ (and thus $T_{sk}$) was more than overridden by an exercise-related vasoconstrictor effect, which serves to maintain cardiac filling. However, it is not clear what stimulus could account for SkBF in the heat being lower not simply at any $T_a$ (i.e., for a relative vasoconstriction), but at any point in exercise. Unless there was much more peripheral venodilation in the experiments at 43.3°C $T_a$, lower SkBF in those experiments should have caused less peripheral pooling of blood and thus a smaller stimulus for vasoconstriction. Furthermore, in another study from the same laboratory [12], in which the subjects were performing milder treadmill exercise (same speed, less steep grades), heating the skin from 32.9° to 38.3°C was followed by substantial increases in cardiac output, implying that under less severe conditions, the skin does vasodilate as expected in response to an increase in $T_{sk}$.

To extrapolate findings and interpretations like those above so as to argue for a similar (if smaller) cutaneous vasoconstrictor effect of exercise in more moderate conditions, we must assume that the vasoconstrictor effect of exercise is invoked not only in severe conditions, above some threshold level of strain, but rather is graded according to the level of strain over essentially the whole range of exercise intensity and thermal strain. This assumption appears plausible by analogy with splanchnic and renal beds [13], in which vasoconstriction during exercise is graded according to exercise intensity over a wide range. Moreover, in heated resting subjects these vascular beds constrict in proportion to the increase in $T_c$ [13]. However, vasoconstrictor signals to vascular beds in the skin may be governed differently, because of the thermoregulatory need for cutaneous vasodilation during exercise, and it may indeed be that relative cutaneous vasoconstriction is invoked only above some threshold level of strain. To distinguish between these two interpretations, I will devote most of the
following discussion to data obtained under conditions of mild to moderate strain. It will be seen that, under such conditions, any vasoconstrictor effect of exercise is so small that it is not needed to give a good quantitative account of the control of SkBF.

**EFFECT ON SkBF OF EXERCISE WITH MILD TO MODERATE CARDIOVASCULAR STRAIN**

*Effect of Exercise as Compared to Rest or Recovery*

Several previous studies [14,15,16] showed reductions in ABF or finger blood flow (FBF) after the start of leg exercise and, in some cases, increases in FBF or ABF after the end of five- or six-minute exercise bouts [15,16]. Although unaccompanied by measurements of $T_c$, such observations seem at first to provide clear evidence of a vasoconstrictor effect of exercise. However, the initial constriction could be wholly a transient response, as in the concept of Christensen et al. [14], who took it to be part of the cardiovascular adjustment associated with the transition from rest to exercise (cf. Fig. 3). In fact, the onset of exercise is often accompanied also by a transient burst of sweating, not explained by changes in $T_c$ or $T_{sk}$ [18]. Although such sweating has been taken as evidence of a reduction in thermoregulatory set-point during exercise, its transience indicates that it, too, is part of a burst of sympathetic nervous activity associated with beginning exercise. The increase in FBF or ABF after the end of five- or six-minute exercise bouts is almost certainly due to increasing $T_c$, since $T_{sk}$ continues to rise for up to several minutes after the end of short exercise periods (cf. Fig. 4). Furthermore, no such increase in blood flow follows the end of exercise which has been continued to near steady-state $T_c$ (Fig. 5). Figure 5 also shows that in a subject who was near the point of vasodilation before beginning exercise, ABF during exercise rose above resting levels almost as soon as $T_c$ did, contrary to what we should expect if exercise elevated the threshold for vasodilation.

The evidence cited in the previous paragraph points up the importance of taking concurrent changes in $T_c$ and $T_{sk}$ into account when interpreting changes in SkBF. Ideally, one would like to compare blood flow during rest and exercise over a range of levels of $T_c$ and $T_{sk}$, covering much of the range observed in subjects exercising in a variety of experimental conditions. Unfortunately, such data are rather scarce, partly because of the limitations of means for raising $T_c$ in resting human subjects without resorting to high $T_{sk}$. One unusual and rather elegant example of an experimental attempt to make just such comparisons is provided by the work of Nielsen and Nielsen [20], who studied the effects of cycle exercise and diathermy at intensities which produced the same rate of total internal heat production (415 or 431 W). In $T_c$ of 9 to 27°C, both means of heating produced similar levels of steady-state rectal temperature, about 37.8°C, in agreement with the hypothesis that the set-point was unchanged by exercise. The authors further reported that at steady state, core-to-skin thermal conductance, which they took as an index of skin circulation, depended only on $T_{sk}$ (rectal temperature not differing systematically between conditions) and not on the means of heating. In spite of the limitations of core-to-skin conductance as an index of SkBF, this result indicates that at the same $T_c$ and $T_{sk}$, SkBF was similar at steady state in both conditions.

Roberts and Wenger [17] looked for non-thermal effects of exercise on the SkBF:$T_c$ relation by measuring FBF, ABF, and temperatures during protocols consisting of alternating three-minute periods of cycle exercise and recovery (Fig. 4). These
experiments were conducted at 25°C $T_a$ on seated subjects, so that although exercise intensity ranged from 70 to 100 percent $V_o_2$ max, thermal stress and peripheral pooling of blood were not great. Relating blood flow to $T_{es}$ and $T_{sk}$, they found transient non-thermal effects (i.e., departures from the predicted relation of blood flow to $T_{es}$ and $T_{sk}$) in the first 30 seconds or so of exercise and recovery (Fig. 3). Thereafter,
although they found a relative vasoconstrictor effect at high exercise intensity in the fingers of some subjects, they found no vasoconstrictor effect in the forearm. (They actually found, if anything, a slight dilator effect.) Though recovery may not be identical to continued rest, arterial pressure returns to resting levels 30–60 seconds after end of exercise [15,21], following a time course which is probably consistent with that of the non-thermal effects on FBF and ABF. The fact of vasoconstriction in the skin of the finger but not of the forearm under the same conditions is not surprising in light of the fact that reflex thermal control of FBF is almost entirely through vasoconstrictor nerves, whereas most of the reflex increase in ABF during whole-body heating is by the action of vasodilator nerves [22].

**Effect of Exercise Intensity**

In some of the earliest work on SkBF at the Pierce Foundation, Wenger et al. [19] measured ABF and $T_s$ in subjects performing cycle exercise in a seated posture at ambient temperatures up to 35°C, and found no vasoconstrictor effect of increasing exercise intensity from 30 to 70 percent $V_O_2$ max (Fig. 6). More recently, John Johnson [23] studied a broader range of intensities, 25–200 W, of cycle exercise in the upright posture and at 24°C $T_s$, and confirmed that exercise intensity does not affect the ABF:$T_s$ relation. The simplest interpretation of these observations is that, under these conditions, exercise does not affect the SkBF:$T_c$ relation. However, it is not impossible that the relation between the degree of non-thermal cutaneous vasoconstriction and exercise intensity has a broad plateau, which is reached with mild exercise and exceeded only when severe conditions, such as heavy exercise at high $T_s$, evoke further constriction.

The lack of any effect of exercise intensity over so wide a range has several important implications: (a) unlike vasoconstriction in the gut and kidneys [13], any non-thermal vasoconstriction in the skin is not graded according to exercise intensity, so that it is not valid to extrapolate observations made under more severe conditions to infer what happens to SkBF under less severe conditions; (b) even if there is a non-thermal cutaneous vasoconstriction during exercise at mild to moderate intensities, its magnitude is independent of exercise intensity, so that such a constriction cannot explain why the increase in $T_c$ during exercise is proportional to exercise.
intensity (cf. Fig. 1); and \( (c) \) in environments cool enough that heat dissipation is dominated by vasodilation, an elevation in the threshold for vasodilation should by itself cause an elevation in \( T_c \), on which any load error necessary to dissipate the increased heat production of exercise would be superimposed. If such a threshold elevation is independent of exercise intensity, then the relation of steady-state elevation of \( T_c \) to exercise intensity should not pass through the origin, but should extrapolate to a positive intercept on the elevation-of-\( T_c \) axis. Such an effect on the \( T_c \) elevation has never been reported, although the available data may not have sufficient resolution to demonstrate it, if the putative constrictor effect is small enough.

**CONCLUSIONS**

*Cardiovascular Homeostasis*

At this point I will propose a general interpretation of the data concerning non-thermal effects of exercise on SkBF and will begin by considering whether and how such non-thermal effects might be useful. Since cutaneous vasodilation serves a vital thermoregulatory function during sustained exercise, why should SkBF be compromised by a relative vasoconstriction during exercise before maximum cardiac output is approached? Rowell [1,10] in addressing this question points to the fact that the superficial veins are very compliant and engorge as SkBF increases. Thus, a reduction in SkBF opposes peripheral pooling of blood, and helps maintain adequate cardiac filling and perfusion of working muscle during exercise. However, a relative vasoconstriction need not operate during exercise under all conditions, since there are other compensatory mechanisms. These mechanisms include changes in cardiac contractility and responses which mobilize blood from the visceral beds, as well as those which directly oppose peripheral pooling, such as peripheral venoconstriction in response to decreases in central venous pressure [24], and the muscle pump. For example, during stationary walking in Henry and Gauer’s study [25], the muscle pump was able to keep dorsalis pedis venous pressure almost as low as 33° as at 25°C \( T_a \), although pressure was considerably higher at 39°C. In their data, then, there hardly seems any evidence of a need for compensatory vasoconstriction up to at least 33°C \( T_a \). Moreover, other evidence suggests that the various responses for maintaining cardiac
output during exercise are not all invoked simultaneously and in proportion to each other, but rather are invoked in an order that provides for preserving thermoregulatory SkBF uncompromised until some rather substantial level of cardiovascular strain is reached. As was pointed out above, reductions in splanchnic and renal blood flow are proportional to exercise intensity over essentially the entire range studied, whereas over much of the same range of exercise intensities, the SkBF:Tc relation is independent of exercise intensity. Furthermore, two recent reports [26,27] show breaks in the slope of the ABF:Tc relation during exercise in certain rather stressful conditions (70 percent \( \dot{V}_{O_2} \max \) at about 35.5°C Tsk [26], and about 60 percent \( \dot{V}_{O_2} \max \) at 38°C Tsk [27]). Such breaks are not intrinsic to the thermal control of ABF, since data obtained on supine subjects—and even on the same subject [26] (Fig. 7)—in the same laboratories show the ABF:Tc relation continuing with undiminished slope well above the levels of ABF at which the breaks were observed. It is probable that these breaks mark the point beyond which relative cutaneous vasoconstriction becomes important during exercise, and that below this point other responses limit peripheral pooling sufficiently to maintain adequate cardiac output.

**Net Effect on SkBF of Non-Thermal Reflex Loops During Exercise**

Although a number of specific reflex loops may affect the ABF:Tc relation during exercise differently from rest, their effects may nearly cancel each other over a fairly broad range of conditions. Arterial pressure, for example, is higher during exercise than rest, and under mild to moderate conditions may be enough to counteract any cutaneous vasoconstrictor effect of other reflexes. Several authors report an effect of arterial baroreceptors on ABF, and Ebert [28] reports further that neck suction (which stimulates the carotid baroreceptors) causes a greater and more sustained increase in ABF when superimposed on a vasoconstriction induced by lower body suction. Under
more severe conditions, several changes may occur to upset the balance between non-thermal factors tending to produce cutaneous vasoconstriction and those tending to produce vasodilation: \((a)\) with increased peripheral pooling of blood, arterial pressure (which apparently has not been measured under such conditions during exercise) may not rise so high during exercise and may no longer be sufficient to overcome the total effect of the factors that tend to cause vasoconstriction; \((b)\) greater muscle ischemia may stimulate chemosensitive free nerve endings in muscle, which are reported to participate in pressor reflexes [29,30]; and \((c)\) unloading of cardiopulmonary baroreceptors may increase the tendency to cutaneous vasoconstriction enough to overcome the effect of elevated arterial pressure. The foregoing scheme is not meant to include all of the heterogeneous set of stimuli which may participate in reflex responses to exercise: other factors have been proposed as well (see also [3]), including stimulation of joint mechanoreceptors and radiations from the motor cortex. Furthermore, during prolonged exercise, blood volume will be reduced by fluid shifts into the tissues (e.g., [26]) and losses in sweat, thus further unloading cardiopulmonary baroreceptors.

**Effect on Thermoregulation**

What are the thermoregulatory consequences of a relative cutaneous vasoconstriction during exercise? As a fair approximation, let us assume that at steady state, the gradient between \(T_c\) and \(T_{sk}\) is inversely proportional to SkBF, thus: \(\text{SkBF} = k \cdot H/(T_c - T_{sk})\), in which \(H\) is rate of heat production in the body, and \(k\) is a proportionality factor. A subject exercising in a cool environment has a wide \(T_c - T_{sk}\) gradient and relatively low SkBF. A given reduction in SkBF would represent a relatively large fraction of total SkBF, and would be accompanied by a relatively large increase in \(T_c - T_{sk}\), and thus in \(T_c\), since \(T_{sk}\) is determined largely by environmental conditions. Thus, a small cardiovascular advantage would be bought at large thermoregulatory cost. On the other hand, a subject exercising in a warm environment has a much higher SkBF and smaller \(T_c - T_{sk}\). In comparison to the subject exercising in the cool environment, a much larger decrease in SkBF would then represent the same fraction of total SkBF and would be associated with the same proportional increase in \(T_c - T_{sk}\). However, since \(T_c - T_{sk}\) is small in this subject, this would result in a relatively small increase in \(T_c\), so that a large cardiovascular advantage can be bought at rather small thermoregulatory cost. Thus the thermoregulatory consequences of a given decrease in SkBF are least in just those circumstances where such a vasoconstrictor effect is most needed, and, conversely, the thermoregulatory consequences of the same decrease in SkBF are greatest in circumstances in which such a vasoconstrictor effect is least needed and probably does not occur to any significant degree. It may seem paradoxical that a given reduction in SkBF can be better tolerated by a subject exercising in the heat, since it is well known that high environmental temperatures increase the risk of heat disorders. The resolution of this paradox is as follows: Even though a given reduction in SkBF during exercise represents a smaller fraction of the total in a warm environment than in a cool one and has a smaller effect on regulation of \(T_c\), yet a subject exercising in a warm environment is likely to experience greater cardiovascular strain, because of greater peripheral pooling of blood and loss of plasma volume, and thus to experience much larger reductions in SkBF, which may be sufficient to impair thermoregulation seriously.
Summary

A number of authors have proposed that SkBF during exercise is less than during rest at the same $T_c$ and $T_{sk}$ or, in other words, that there is a relative cutaneous vasoconstriction during exercise. Such an effect is thought to be either a consequence of an elevation in thermoregulatory "set-point" during exercise or a specifically cardiovascular response which helps to support perfusion of exercising muscle. The available evidence does not support the hypothesis of a change in set-point during exercise. Nevertheless, there is convincing evidence for a relative cutaneous vasoconstriction during exercise under conditions of high cardiovascular strain (high $T_{sk}$ or intense exercise), and several authors have argued that it occurs under more moderate conditions also, with a magnitude proportional to exercise intensity. Comparison of control of SkBF during rest with that during exercise under more moderate conditions is complicated by the difficulty of raising $T_c$ and SkBF of resting human subjects without using high $T_{sk}$. However, data from one study in which this was accomplished with diathermy show no difference in steady-state $T_c$, $T_{sk}$, or core-to-skin thermal conductance between exercise and rest, with internal heat production matched in both conditions. In addition, comparison of the relation of FBF or ABF to $T_c$ and $T_{sk}$ during moderate exercise with that during the first few minutes of recovery does not support a relative cutaneous vasoconstriction during exercise. Furthermore, exercise intensity over a fairly wide range seems not to affect the relation of ABF to $T_c$ and $T_{sk}$. Thus it is likely that under mild to moderate conditions, other compensatory cardiovascular responses are sufficient to allow adequate perfusion of exercising muscle, and are invoked in preference to relative cutaneous vasoconstriction, which has been demonstrated only at higher levels of strain. The thermoregulatory SkBF required during sustained exercise is thus preserved as much as possible.

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