

CHANGES IN CUTANEOUS BLOOD FLOW DURING INCREMENTAL EXERCISE WITH AND WITHOUT EXTERNAL THERMAL STRESS

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INTRODUCTION

At the commencement of exercise, blood is redistributed from the viscera, inactive muscles and skin to exercising muscles (2,6). The reduction in blood flow to these organs is proportional to exercise intensity (5). As exercise continues, heat accumulates and the need for dissipation increases. Cutaneous vasoconstrictor tone is inhibited, and the resultant rise in skin blood flow (SkBF) ensures that heat flows from the core to the skin, eventually being lost to the environment. As exercise intensity and metabolic heat production increase, the demand for blood by active muscles increases, leaving a smaller portion of cardiac output available for SkBF. In a hot environment, reduced availability of blood for heat dissipation, and the return of cutaneous constrictor tone, results in an increase in core temperature which exceeds that observed without SkBF attenuation. It was the aim of this study to identify relative exercise intensities at which SkBF attenuation occurs during incremental exercise in two different thermal environments.

METHODS

Six males (21.3 yr, aerobic power [$\dot{V}_{O_{2peak}}$] 4.92 ± 0.23 l.min⁻¹; $\bar{X} \pm S.D.$) participated in two trials, conducted seven days apart at the same time of day in neutral ($19.6 \pm 0.3^\circ\text{C}$, rh $50.2 \pm 1.4\%$) and hot ($36.7 \pm 0.2^\circ\text{C}$, rh $46.1 \pm 3.2\%$) environments. Trials consisted of a 25 min adaptation period, with data collection commencing 5 min prior to exercise to obtain resting values. Cycling began at 40% \dot{W}_{peak} for 10 min, increasing every 4 min by 5% \dot{W}_{peak} . All cycling was conducted on an electronically braked cycle ergometer (Excalibur, Quinton). Tests ended after 30 min or if auditory canal temperature (T_{ac}) was $\geq 39^\circ\text{C}$. In the hot condition, five subjects completed 26 minutes of exercise with the sixth terminating after 22 minutes. Thirty minutes of cycling was completed by all subjects in the neutral environment. Measures included: T_{ac} (zero gradient aural thermometry), skin temperatures at eight sites (T_{sk} , YSI EU mini-thermistors), forearm SkBF (laser Doppler velocimetry, TSI Laserflo BPM², Vasamedics; $\lambda=780$ nm, fibre separation of 0.5 mm, and SkBF expressed in voltage units), cardiac frequency (f_c), and thermal sensation and perceived exertion (RPE) votes.

RESULTS

T_{ac} , f_c and \bar{T}_{sk} were always greater in the heat ($p < 0.05$), while terminal f_c was lower in the neutral state ($91.6 \pm 1.9\%$ versus $98.1 \pm 1.6\%$ f_{cpeak} ; $p < 0.05$; $\bar{X} \pm S.E.M.$). The exercise-related vasodilatory threshold occurred earlier in the heat (318.3 ± 26.6 s versus 761.8 ± 48.3 s; $p < 0.05$), but at a similar T_{ac} ($37.3^\circ\text{C} \pm 0.04$ versus $37.3^\circ\text{C} \pm 0.1$; $p > 0.05$). SkBF attenuation occurred at different exercise intensities between the environments, being lower in the heat ($40.8 \pm 0.8\%$ versus $55.8 \pm 3.0\%$ \dot{W}_{peak} ; $p < 0.05$). T_{ac} did not differ between the conditions at the point of attenuation ($37.7^\circ\text{C} \pm 0.3$ {hot} versus $37.8^\circ\text{C} \pm 0.04$ {neutral}; $p > 0.05$; Figure 1), and SkBF at this point was similar between conditions (1.07 ± 0.07 Volts {hot} versus 1.11 ± 0.09 Volts {neutral}; $p > 0.05$). However, the slope of SkBF: T_{ac} relation was greater in the hot environment (3.65 ± 1.03 Volts. $^\circ\text{C}^{-1}$ versus 1.01 ± 0.14 Volts. $^\circ\text{C}^{-1}$; $p = 0.05$). \bar{T}_{sk} at the point of SkBF attenuation differed significantly between conditions ($36.9^\circ\text{C} \pm 0.1$ {hot} versus $32.0^\circ\text{C} \pm 0.4$ {neutral}; $p < 0.05$). Thermal sensation and RPE votes were all greater in the hot condition ($p < 0.05$).

CONCLUSIONS

The attenuation of SkBF in both the hot and neutral conditions at an equivalent T_{ac} indicates that body core temperature (T_c) may dominate the control of this phenomenon. Attenuation occurred at different exercise intensities and T_{sk} in the two environments, giving further support to the possibility of greater T_c control. Figure 1 indicates that data from the two trials may fall at opposite ends of an apparently continuous, ailinear relationship. Environmental temperature seems not to have affected this relationship, although it is probable that local skin temperature (T_{sk}) causes the greater gain in the hot condition.

The attenuation in SkBF is in close agreement with the work of Brengelmann *et al.* (1) who observed reduced SkBF gain at a T_c of 38°C , and Smolander *et al.* (7) who found a similar T_c at attenuation in different environments. A reduced vasodilator gain to T_c elevation, combined with progressive vasoconstriction associated with regulation of arterial pressure were suggested as explanations for this SkBF decline. The concept of a reduced gain of T_c elevation seems probable, since in two different environments, at different

T_{sk} , attenuation occurred at the same T_{ac} . SkBF was equivalent in the two environments at the point of attenuation, contrary to the findings of Smolander *et al.* (7), implying that this phenomenon may be related to changes in central blood volume, which has been shown to decrease rapidly during even moderate exercise (4).

The present data indicates that SkBF attenuation occurs at an exercise intensity between 40-55% \dot{W}_{peak} during incremental exercise, depending on air temperature. This supports previous observations of Wenger *et al.* (9) who found SkBF to be attenuated at 50% $\dot{V}_{O_{2peak}}$ in a neutral environment, but conflicts with the observations of Smolander *et al.* (8) who observed reduced SkBF gain at 80% $\dot{V}_{O_{2peak}}$. However, both these studies used a constant exercise intensity in any one trial, whereas this study was primarily interested in identifying the exercise intensity where SkBF becomes attenuated during incremental exercise.

This SkBF attenuation may well be exercised-induced vasoconstriction, however it may also indicate that a maximum flow through the blood vessels has been attained, although subsequent local heating has been shown to increase SkBF after attenuation has occurred (1,3). It may also be an artifact of the apparatus, with the output signal becoming saturated. This is unlikely since the output at attenuation was less than 50% of the output range, with a larger SkBF being observed following application of an exogenous vasodilatory agent.

Current data also show the maintenance of a stable SkBF in the heat, 20-25 minutes after attenuation, in the presence of a progressively increasing exercise intensity. The work rate increased by 71.5 (± 11.1) Watts with no further reduction in SkBF. The blood flow demands of the active muscles would have continually increased during this period. Two possibilities may account for this observation. Muscle blood flow was maintained through redistribution from the viscera, or it may have been compromised during the latter stages of exercise. The elevated RPE and reduced exercise duration in the heat tend to support the latter alternative.

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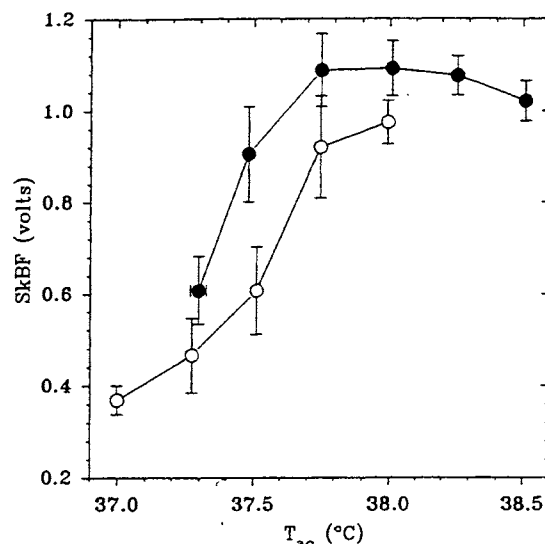


Figure 1 SkBF as a function of T_{ac} during incremental exercise in neutral (○) and hot (●) environments.