# POLYCYTHAEMIA AND ITS AFFECT ON SUDOMOTOR AND CUTANEOUS BLOOD FLOW RESPONSES TO HEAT STRESS

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## INTRODUCTION

Polycythaemia has been shown to improve submaximal and maximal exercise performance in a neutral environment (1,5). Possible mechanisms responsible for this improvement are an increased arterial oxygen ($O_2$) content or an expanded blood volume (1,10). Since it is unlikely that blood volume changes, as plasma volume is decreased to maintain a constant blood volume (3), increased performance may primarily be attributed to an increased arterial $O_2$ content.

Thermoregulatory processes may benefit from the increased arterial $O_2$ content, since the fraction of cardiac output needed at the exercising muscles is reduced, allowing a greater proportion for heat dissipation. This is beneficial, since, during combined exercise and heat stress, competition for blood flow exists between the exercising muscles and cutaneous vasculature. At a given work rate, a polycythaemic subject may be exercising at lower relative work rate (1,10), and since body core temperature ($T_c$) is primarily a function of relative exercise intensity (8), polycythaemia may reduce thermal strain.

Sawka *et al.* (9,10) reported a reduction in cardiac frequency ($f_c$) and $T_{re}$ accompanied by an increase in local and whole body sweat rate, and sensible heat exchange at the arm, when heat acclimated subjects were rendered polycythaemic. Since we were investigating the affects of polycythaemia on other physiological functions, we decided to further examine its influence on thermoregulation.

## METHOD

Five trained males participated in two heat stress trials, each with 20 min seated rest, 20 min cycling at 30% peak power ($W_{peak}$), and 20 min at 45% $\dot{W}_{peak}$ at a dry bulb temperature of 38.3 ±0.7°C (relative humidity 41.4 ±2.9%). The same absolute work rates were used for each trial: 125.8 ±15.6 Watts (30% $\dot{W}_{peak}$) and 186.9 ±20.3 Watts (45% $\dot{W}_{peak}$; $\bar{X}$ ±S.D.; Monark). Trials were undertaken during normocythaemia (control) and isovolaemic polycythaemia (respective haematocrit 39.5 f1.8 & 43.1 ±1.7%, $p<0.05$; and red cell count 4.17 ±0.21 & 5.03 f0.38 x$10^{12}L^{-1}$, $p<0.05$). Polycythaemia was obtained by reinfusing 2-3 units of autologous blood*, approximately 12 weeks after withdrawal and glycerol freezing.

Core temperature was recorded at the auditory canal ($T_{ac}$; zero gradient aural thermometry, London Hospital), local sweat rates at the forearm and forehead ($\dot{m}_{sw}$; capacitance hygrometry: Multi-site Sweat Monitor, Clinical Engineering, Sydney), and skin blood flow (SkBF) was measured at the forearm, upper arm, head, back, chest and thigh (laser Doppler velocimetry: TSI Laserflo BPM², Vasamedics; λ=780 nm, fibre separation of 0.5 mm, and expressed in voltage units). SkBF was measured continuously at the forearm for the first 15 min of each test phase, then at each of the other 5 sites over the next 5 min. Other measures: skin temperatures at 8 sites ($\bar{T}_{sk}$ (after 4); YSI EU mini-thermistors), $f_c$ (polar PE3000), thermal sensation and rating of perceived exertion (RPE).

## RESULTS

In the polycythaemic state, $T_{ac}$ (collapsed across the last 20 min) and $f_c$ (across total test duration) were significantly reduced (38.0°C ±0.1 versus 38.2°C ±0.1, and 104.4 ±3.7 versus 115.4 f5.3 b.min$^{-1}$; $\bar{X}$ ±S.E.M.; $p<0.05$). SkBF, averaged across the exposure, was lower in the polycythaemic state (0.76 ±0.08 versus 0.97 ±0.18; $p<0.05$), primarily due to differences within the last 20 min. Similar responses were observed for SkBF in the upper arm and back when averaged across the 3 test phases ($p<0.05$). Local skin temperatures at the forearm were significantly higher across the exposure ($p<0.05$), as was mean skin temperature, during the polycythaemic trials. Thermal sensation and RPE remained equivalent between conditions ($p>0.05$).

Sweat onset at the forearm was earlier when subjects were polycythaemic (363.0 ±259.1 versus 1224.9 ±188.6 s; $p<0.05$), although there was no difference at the forehead ($p>0.05$), and the sweat threshold did not differ between conditions for either the forearm or forehead ($p>0.05$). $\dot{m}_{sw}$ gain was unchanged at the

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forehead, although forearm gain was elevated in the polycythaemic state (2.9 f0.6 versus 1.8 f0.4 $mg \cdot cm^{-2} \cdot min^{-1} \cdot °C^{-1}$; $p < 0.05$). During polycythaemia, forehead $\dot{m}_{sw}$ was significantly lower during the 45% $\dot{W}_{peak}$ work phase (averaging 3.43 ±0.15 versus 3.60 f0.13 $mg \cdot cm^{-2} \cdot min^{-1}$), while the forearm $\dot{m}_{sw}$ was significantly greater during all phases of the heat stress test (1.20 f0.13 versus 0.94 f0.11 $mg \cdot cm^{-2} \cdot min^{-1}$). Body mass loss did not differ between trials (0.84 f0.04 (control) versus 0.98 fO.11 kg; $p > 0.05$).

CONCLUSIONS

Polycythaemia *appeared* to reduce thermal strain, lowering $T_c$ and $f_c$. Since $\dot{W}_{peak}$ did not increase in the polycythaemic state, subjects were exercising at the same absolute and relative work rates during both tests. Therefore, reduced strain was the result of an altered haematocrit, and not differences in relative work intensity, producing an altered $T_{sc}$: % $\dot{W}_{peak}$ relationship. Lower thermal strain was possibly achieved by an elevation in $\dot{m}_{sw}$ at some skin surfaces, as reflected in forearm $\dot{m}_{sw}$, which was attributed to an increase in local skin temperature, since $T_{sc}$ was either equal or lower during the second exposure.

It was anticipated that SkBF would be greater in the polycythaemic state, as an elevated $O_2$ carrying capacity of the blood should allow for a greater redistribution of the cardiac output to the skin. The paradoxical reduction in forearm SkBF was also reflected in the SkBF of the upper ann and back, but not at the other *sites.* These changes were possibly associated with a lower $T_c$ over the latter 20 min of the polycythaemia exposure, and a reduction of active vasodilatory tone.

The slight reduction in forehead $\dot{m}_{sw}$, coupled with the rise in forearm $\dot{m}_{sw}$, and equivalent body mass changes, indicates a possible redistribution of sweat, favouring the more distal sites. We have found a similar redistribution of sweat production in heat stressed anaemic subjects (7). Shvartz (11), and more recently Regan *et al.* (6) have observed such a redistribution accompanying heat acclimation. It was found that the distal sites exhibited greater changes in $\dot{m}_{sw}$ than did more proximal sites. Steady state $\dot{m}_{sw}$ tends to be higher centrally (2), and consequently, there is little margin for further increments in $\dot{m}_{sw}$. However, our data show a clearly diminished forehead $\dot{m}_{sw}$ below that observed in the control state, supporting the notion of an active redistribution of sweat production when subjects were rendered polycythaemic.

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