CONVECTIVE AND METABOLIC HEAT IN HUMAN UPPER LIMBS DURING THERMAL STRESS IN WATER

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INTRODUCTION

The limbs are major sites for heat loss, contributing about half of the total body heat loss during cold water immersion in the absence of shivering (1). It has been reported that the major portion of the heat loss from the limbs (70 to 80%) is attributed to the local metabolic heat production of the tissue, and that the balance originates from the convective heat transfer between blood and tissue (2). These results, however, contrast with those obtained in the same study (2) for the extremities where the majority of the tissue heat loss from the fingers and hand during cold stress originated from the convective heat transfer with the blood. In addition, other studies have suggested that despite minimum tissue perfusion during cold stress, the convective heat transfer has possibly a greater role than suggested by previous studies (3, 4).

The objective of the present study was to investigate, using a 30 minute period of arterial occlusion of the forearm, the relative contribution of the convective heat transfer in the forearm and extremity to 1) the total heat loss during immersion in cold water (20°C) and to 2) the heat gain during immersion in warm water (38°C).

METHOD

Eleven healthy male subjects, aged 18-40 years, underwent two partial immersions at two different temperatures: 20 and 38°C. Before the immersion, the lightly dressed subject (T-shirt and casual pants) sat comfortably under thermoneutral conditions (Tair=25.0±0.2°C, HR=40±2%) for 1 hour. After the resting period, the subject immersed his left forearm and hand for 3 hours in a well-stirred water bath maintained at a constant temperature of either 20 or 38°C. At the end of the 3-h immersion, an arterial cuff, previously fixed on the left upper arm was inflated at 250 mmHg for 30 minutes while the forearm and hand were still immersed in water.

The heat fluxes from the skin of the forearm and finger were continuously monitored during the experiments with 23 recalibrated waterproofed heat flux transducers (HFTs). The HFTs were placed equidistant from each other along the circumference of a segment. Four were fixed around the middle phalanx of the middle finger, five around the distal segment of the forearm (wrist), six around the medial segment, and eight around the proximal segment (9 cm from the elbow). This resulted for the forearm in a similar coverage on the basis of surface area to segment volume ratio. True average heat flux value from the segment was thus obtained from the measurement of heat flux at all sites.

RESULTS

The heat flux values changed significantly during the 30-min occlusion period for all segments and both water temperatures tested. The heat flux values decreased during the occlusion period at Tw=20°C and increased at 38°C, and were significantly higher than 0 $W \cdot m^{-2}$ for all sites at both water temperatures at the end of the occlusion period, except for the proximal segment of the forearm at Tw=38°C (see Fig. 1). This indicates that by the end of the occlusion period at Tw=38°C, the heat flux values became positive (heat loss) despite a higher water temperature compared to the rectal temperature of the subjects (36.92 ± 0.05°C). The heat flux values reached steady state for the finger at Tw=20°C and 38°C after approximately 10 min of arterial occlusion, while the values were still changing for the other segments on the forearm.

The tissue metabolic heat production of the different sites at steady state before occlusion was assumed to equal the heat loss from the site at thermal stability during the arterial occlusion (the longitudinal conductive heat transfer was assumed to be negligible). It was calculated for the finger that the tissue metabolic rate was equal to 124.8±29.0 $W \cdot m^{-3}$ at Tw=20°C and 287.7±41.8 $W \cdot m^{-3}$ at Tw=38°C. Since thermal stability was achieved only for the finger during the occlusion period, the metabolic heat production of the tissue for the forearm sites were assumed to equal the measured asymptote values. This assumption holds only if the metabolic heat production of the tissues stays constant during the occlusion period. When the predicted metabolic rates (calculated from standard metabolic rate for every tissue type and taking into account the Q10 effect; $\dot{H}m$) are compared to the measured asymptote values it is observed that at Tw=20°C both values are close for every sites, but at Tw=38°C, $\dot{H}m$ is greater than the asymptote for the forearm sites. Hence, the assumption of a constant metabolic heat production seems correct for Tw=20°C but not for Tw=38°C.

The contribution of the convective heat transfer by the blood at thermal steady state during immersion at Tw=20°C was calculated by substracting the tissue metabolic heat production (predicted $\dot{H}m$ or measured asymptote) from the measured heat lost for all sites before the occlusion period. At thermal steady state during immersion at Tw=38°C, the convective heat transfer by the blood was calculated by adding the tissue metabolic heat production (predicted $\dot{H}m$) to the measured heat gain by the different sites before the occlusion period. It was calculated that at thermal steady state at Tw=20°C, the convective heat transfer with blood contributed 90% of the forearm heat loss but only 40% of the finger heat loss. At thermal steady state at Tw=38°C, 72% of the forearm heat gain originated from the environment and only 28% from the tissue metabolism, while 82% of the finger heat gain originated from the environment.

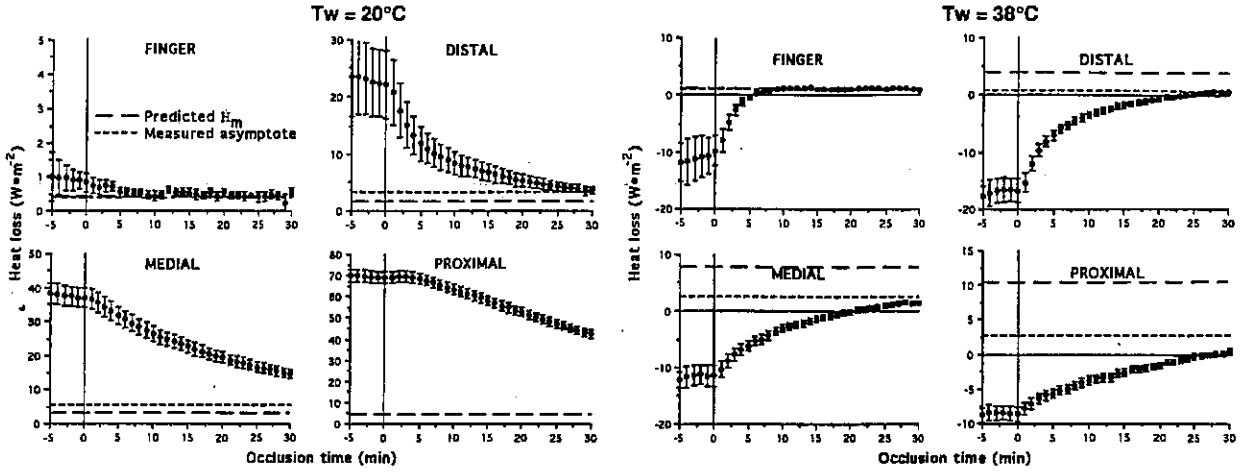

Figure 1. Finger and forearm heat losses during the 30 minutes of arterial occlusion in water at 20 and 38°C. Predicted tissue metabolic rate ($\dot{H}m$) and measured asymptote values are indicated.

DISCUSSION

The results of the present study support the theoritical studies of Mitchell et al. (3) and Tikuisis and Ducharme (4) which suggested that during cold stress the major contributor to the limb heat loss (or to the tissue temperature) is the convective heat transfer with the blood. The present study also suggests that the convective heat transfer with blood contributes as much as the tissue metabolism to the finger heat loss during cold stress, possibly due to an intense vasoconstriction at Tw=20°C, and to the very low temperature difference between blood and tissue. These results are in conflict with those of Rennie (2) which suggested that the tissue metabolism is mainly responsible for the limb heat loss, and that the convective heat transfer with blood is the main contributor to the finger heat loss during cold stress. The apparent discrepancy between the studies is attributed to 1) conclusions reached by Rennie (2) from data at non steady-state during a 15-20 min of arterial occlusion (for the proximal forearm), and to 2) low resolution of the finger heat flux measurements.

During heat stress at Tw=38°C, because of the large tissue perfusion, the blood acts as a heat sink, carrying away from the limb and extremities the heat gained from the environment and to a lesser extend (28%), the heat produced by the tissue metabolism.

In conclusion, this study suggests that despite minimum tissue perfusion in the forearm during cold stress, the convective heat transfer by the blood has a greater role in thermoregulation than previously suggested.

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