THE EFFECT OF SINGLE LEG KNEE EXTENSIONS ON THE CONTRALATERAL QUADRICEPS MUSCLE TEMPERATURE

G.P. Kenny[1], M.B. Ducharme[3], M.L. Reardon[2], W. Zaleski[2] and F.D. Reardon[1]

University of Ottawa, [1]Faculty of Health Sciences and [2]Faculty of Medicine, Ottawa, Canada, K1N 6N5 and [3]Defence and Civil Institute of Environmental Medicine, Toronto, Canada, M3M 3B9.

❦

INTRODUCTION

We have previously shown that esophageal temperature remains elevated by ~0.5°C for at least 65 min after exercise **(8).** In a subsequent study, we demonstrated that successive exercise/recovery cycles performed at progressively increasing pre-exercise esophageal temperature levels resulted in further and parallel increases of the esophageal temperature threshold for cutaneous vasodilation during exercise and the post-exercise elevation in esophageal temperature **(4).**

In recent studies, the exercise-related effect on the post-exercise threshold of cold (i.e., vasoconstriction and shivering) **(5)** and warm (i.e., cutaneous vasodilation and sweating) (6) thermoregulatory responses were evaluated. Results indicated that exercise exerts a residual effect on thermal responses by increasing **(-0.3** to $0.5°C$) the post-exercise resting threshold for cold and warm thermal responses. This residual effect **is** not a result **of** the exercise-induced elevation of whole body heat content (3), but most likely due to exercise-related factors such as cardiovascular (either central or peripheral), metabolic, endocrine or neurochemical (i.e., interleukin-1, a-interferon, dopamine, etc.) changes during exercise that alter hypothalamic temperature regulation **(7).**

However, recent evidence favors a baroreceptor-mediated influence, in that a post-exercise hypotensive effect has been observed following acute bouts of exercise likely due to a persisting peripheral tissue vasodilation (1). It has been suggested that the resultant peripheral vasodilation following exercise may cause a pooling of the 'warmed blood, thus entrapping the residual heat of muscle, thereby temporarily increasing local tissue heat content. This may result in a time-dependent transfer of the residual heat of muscle to the core.

It has been demonstrated that local tissue heat content is mainly determined by convective heat transfer with blood (2). Therefore, in the absence of **an** increased heat loss response (i.e., cutaneous vasodilation and sweating) (5,6), core temperature would remain elevated as long as the heat content of muscle remains higher than that of the core and/or the post-exercise hypotensive effect is removed. This study was designed to evaluate the role of nonactive tissue in the retention and dissipation of heat during and following intense isolated muscle activity.

METHODS

Subject Selection. Five males and 1 female participated in the study. They were physically active, although none engaged in daily or intensive training programs.

Instrumentation. Esophageal temperature (T_{es}) was monitored as an index of core temperature. Skin heat flux and temperature (T_{SKavg}) were measured from 12 sites by combined thermal flux transducer/thermistors and the area-weighted mean was calculated. Heart rate was monitored continuously. Oxygen consumption was determined continuously by an open-circuit method from measurements of expired minute volume and inspired and mixed-inspired gas concentrations sampled from a mixing box. Forearm blood flow was measured by laser-Doppler flowmetry.

Muscle temperature of the non-exercising leg was measured by a thermo-couple temperature probe inserted into the vastus medialis under ultrasound guidance. Temperature sensors were located at ~10, 25 and 45 mm (i.e., T_{mu10}, T_{mu25} and T_{mu40}) from the femur and deep femoral artery. The implant site was about midway between, and medial to, a line joining the anterior superior iliac spine and the superior aspect of the center of the patella.

Experimental **Protocol.** Subjects were required to perform an incremental isotonic test on the **KIN-COM** isokinetic apparatus to determine their maximal oxygen consumption for one-legged knee extension. The results of the test were used to establish the work level for the experimental trial.

Experimental trials were conducted in the morning. Following the insertion of the intramuscular probe (i.e., in the non-exercising leg), subjects rested in a semi-recumbent position for 60 min in an ambient condition of 22°C. During this period, the subject was instrumented appropriately and then remained seated for a minimum period of 30 min. Subjects then performed 15 rnin of one-legged right knee extensions against a dynamic resistance corresponding to 60% of maximal oxygen consumption. Exercise was followed by 60 min of seated rest.

Analysis of Results. Statistical analyses, for T_{es}, T_{SKavg} and T_{mu} were performed by ANOVA for repeated measures to compare values for baseline (average of final 5 min), end-exercise and at 10-min intervals post-exercise. Data are presented as means ± SE.

RESULTS

Baseline T_{es} and T_{SKavg} were 37.03 ± 0.1 "C and 31.81 ± 0.3 °C respectively. Resting muscle temperature was significantly lower than esophageal temperature (i.e., 36.60 ± 0.1, 36.58 ± 0.1 and 36.45 ± 0.1°C for T_{mu10}, T_{mu25} and T_{mu40}, respectively) (Figure 1. Following the onset of exercise, both Tes and Tmu increased for the duration of exercise. T_{Skavg} demonstrated a slight decrease at the onset of exercise followed by a gradual increase, ~5 min into exercise, to an end-of-exercise value of 32.18°C (N.S.). The increase in T_{Skavg} was paralleled by an increase in the rate of heat loss (Figure 2).

Exercise resulted in a T,, and T_{Skavg} increase of 0.27°C and 0.37°C, respectively, above pre-exercise rest (P < 0.05). Muscle temperature (T_{mu}) increased
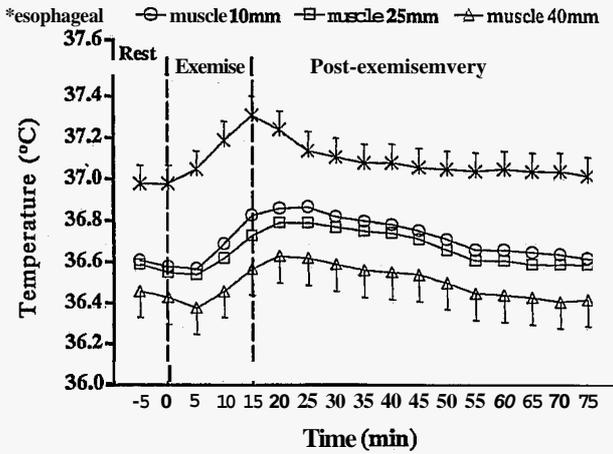
Figure 1. Mean (±SE) esophageal and muscle (non-exercising leg) temperatures during baseline resting, exercise and post-exercise recovery (n = 6).
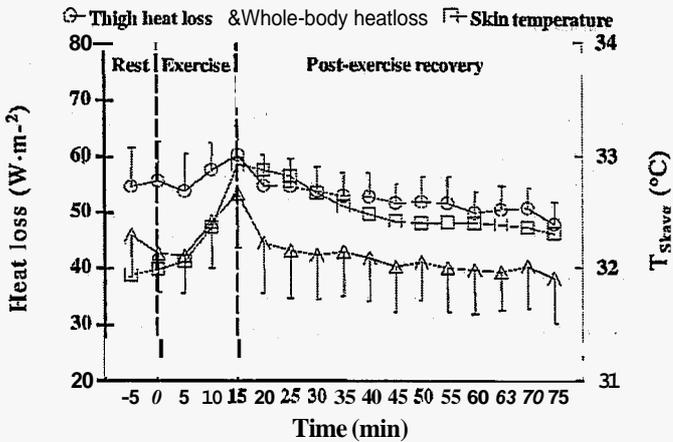
Figure 2. Mean (±SE) heat flux of the inactive thigh and whole body and average skin temperature during baseline rest, exercise and post-exercise resting (n = 6).

0.22, 0.14 and 0.11°C for T_{mu10}, T_{mu25} and T_{mu40}, respectively (P < 0.05). T_{es} continued to increase for –2 min following cessation of exercise after which Tes decrease to baseline values within –20 min. In contrast, T_{mu} continued to increase for –10 min post-exercise reaching a peak value of 36.86 ± 0.1, 36.78 ± 0.1 and 36.62 ± 0.2°C for T_{mu10}, T_{mu25} and T_{mu40}, respectively (P < 0.05). While T_{es} decreased to baseline values within –20 min of end-exercise, T_{mu} reached pre-exercise resting values following –40 min of recovery.

CONCLUSION

These data show that the rate of tissue heat production of the active muscle was sufficient to result in a significant elevation of esophageal temperature and a subsequent increase in the contralateral (i.e., inactive leg) muscle temperature. Although muscle heat loss, **of** the inactive leg, increases above resting during mid-exercise, it actually decreased to resting values before peak muscle temperature was reached (i.e., at –10 min of post-exercise rest). Our results may support the importance of convective heat transfer by blood to inactive muscle tissue in reducing the rate of core temperature increase during exercise. In addition, the sustained increase in muscle temperature (i.e., inactive leg) during the early stages of recovery, in contrast to the decrease of esophageal temperature and rate of heat loss, demonstrates the potential role of inactive muscle tissue **as** a heat sink during recovery. Although the transfer of heat to the cooler tissue regions may act **as** a heat sink during exercise, thereby reducing the thermal stress, it results in a significant residual heat load during subsequent recovery. This may in fact increase recovery time required to re-establish normal resting temperatures.

REFERENCE

1. Coats, A.J.S, Conway, J., Isea, J.E., Pannarale, G. and Sleight, P. **1989,** Systemic and forearm vascular resistance changes after upright bicycle exercise in man, Journal of Physiology (Lond.), **413,289-298.**

2. Ducharme, M.B. and Tikuisis, P. **1994,** Role of blood as heat source or sink in human limbs during local cooling and heating, Journal of Applied Physiology, **76,2084-2094.**

3. Kenny, G.P., Giesbrecht, G.G. and Thoden, J.S. **1996,** A comparison of human thermoregulatory response following dynamic exercise and **warm-**water immersion, European Journal of Applied Physiology, **74,336-341.**

4. Kenny, G.P., Giesbrecht, G.G. and Thoden, J.S. **1996,** Post-exercise thermal homeostasis as a function of changes in pre-exercise core temperature, European Journal of Applied Physiology, **74,258-263.**

5. Kenny G.P., Chen, A.A., Nurbakhsh, B.A., Denis, P.M., Proulx, C.E. and Giesbrecht, G.G. **1998,** Moderate exercise increases the post-exercise threshold for vasoconstriction and shivering, Journal of Applied Physiology. Forthcoming.

6. Kenny, G.P., Nurbakhsh, B.A., Denis, P.M., Proulx, C.E. and Giesbrecht, G.G. **1998,** Moderate exercise increases the post-exercise threshold for vasodilation and sweating, Journal of Applied Physiology. Forthcoming.

7. Sawka, **M.N.** and Wenger, C.B. **1988,** Physiological responses to acute exercise-heat stress, in K.B. Pandolf, M.N. Sawka and R.R. Gonzalez (eds), Human Performance Physiology and Environmental Medicine at Terrestrial Extremes. (New York Benchmark), **97-152.**

8. Thoden, J.S., Kenny, G.P., Reardon, F., Jette, M. and Livingstone, S. **1994,** Disturbance of thermal homeostasis during post-exercise hyperthermia, European Journal of Applied Physiology, **68, 170-176.**