

POST-EXERCISE DEFENCE OF THERMAL HOMEOSTASIS

James Thoden, Glen Kenny. and Sydney Livingstone**

Faculty of Health Sciences, University of Ottawa,
Faculty of Medicine, University of Ottawa. and
Defence and Civil Institute of Environmental Medicine-

INTRODUCTION

Acute metabolic or environmental changes which displace core temperature normally invoke thermal reflexes in an attempt to defend homeostasis within narrow limits. Why then have we seen an elevated (0.5°C) esophageal temperature (T_{es}), following moderate exercise which is maintained without significant change for up to 90 minutes of resting recovery (1)? While it has been proposed that muscular activity may "reset" the temperature that the hypothalamus attempts to maintain, the majority of evidence supports that temperature elevation during exercise simply reflects an uncompensated thermal error which is reversed when muscular demand for blood flow is terminated (2). Our data showed that Oxygen Consumption (VO_2) had returned to resting levels within 10 min after moderate intensity running, and could therefore not be contributing to the T_{es} elevation. It has been reported that the temperature threshold for skin vessel dilation is elevated during exercise, probably through neural influences (3). However, no references have been made to a persistent post-exercise (PoEx) T_{es} elevation when the primary neural events of movement have ceased. We have also reported that the PoEx T_{es} is quantitatively similar to the T_{es} at which skin vessels dilate during exercise (T_{dil}), thus suggesting a link between the PoEx observations and thermal reflex activity. These observations lead to the hypothesis that there was some residual influence related to exercise that retained the modulation of thermal reflex thresholds during recovery. Testing the hypothesis was begun with experiments to establish if: 1. The PoEx T_{es} was related to pre-exercise (PrEx) temperature, 2. Exogenous thermal loading would produce the same post treatment elevation, and 3. Promoting more rapid cooling would affect the elevated plateau.

METHODS

Body temperatures were represented by an esophageal catheter at the level of the left atrium, a rectal catheter and skin thermistors taped to the forehead, chest, forearm, finger tip and thigh (T_{he} , T_{ch} , T_{fa} , T_{th}). Heart rate was recorded every 5 seconds. All experiments were done in the morning following a physical and mental stress free 24 hr, 1-2 hr habituation in a thermal room (29°C , 50% humidity) and at least 48 hr between experiments. Subjects were allowed about 0.25 l water each PrEx waking hour. On day 1, subjects ($n=9$) repeated three cycles of 15 min treadmill running and 30 min standing recovery. On day 2, subjects ($n=9$) were immersed to the neck in 44°C water until T_{es} equaled that temperature produced by previous exercises, removed, dried and allowed to recover at 29°C . On day 3, subjects ($n=4$) exercised in air at 24°C , stood for 15 min and entered 29°C water to complete recovery.

RESULTS

Repeated running-recovery cycles (fig. 1) produced patterns of rise and then fall of T_{es} to an elevated PoEx plateau that was equal to T_{dil} . This was similar to previous results except that the second and third exercises were begun at an elevated T_{es} and produced further elevations of PoEx T_{es} . PoEx elevations were equal to the rise in T_{dil} with each successive exercise and linearly related to the increases of PrEx T_{es} . Tre failed to recover to PrEx levels in the time measured. Non-acral skin (except thigh) temperatures (T_{fa} shown is representative of T_{he} and T_{ch}) approximated PrEx levels by the end of recovery. T_{fi} remained elevated while T_{th} fell continuously as did heart rate (not shown). Immersion in 44°C water (fig. 2) produced increases in core temperature similar to exercise but no PoEx elevated plateau of T_{es} . Immersion in 29°C water after exercise (fig. 3) prevented the PoEx T_{es} elevation and increased the rates of recovery.

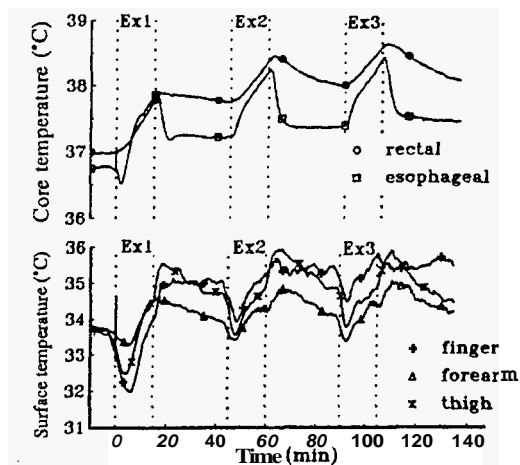


Fig. 1 Intermittent exercise (15 min) and recovery (30 min) at 29°C

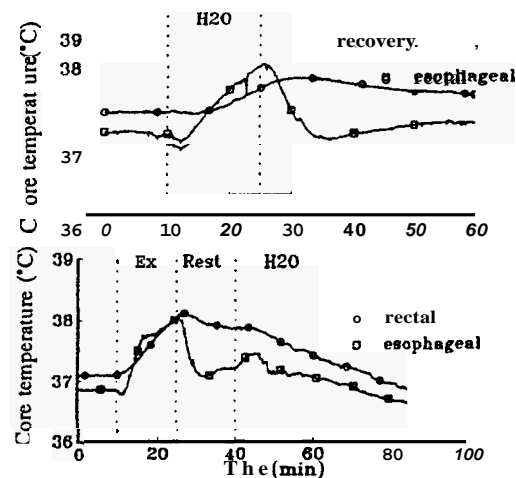


Fig. 2 Water immersion at 44°C (top)
Fig. 3 Exercise (room 24°C) followed by recovery in water at 29°C (bottom)

DISCUSSION

It is plausible that the plateau was maintained because venous return from the exercised muscle and pelvic tissues was distributed to skin only in sufficient volume to provide enough cool blood returning from the skin to reduce Atrial blood to the T_{dil} threshold. Thus, the "thermostat" appeared satisfied even though a significant load error remained in pelvic and leg tissue. This speculation is supported: by the slowly decaying temperature at the thigh and rectal sites which probably acted as heat sources to keep T_{es} up; by heat loss from the skin as indicated by the rapidly falling temperature at other non-acral sites; and by the persisting high temperature at the finger where vasodilatory innervation does not exist and can therefore not be reduced. In addition to indications that skin vessel dilation, invoked by active vasodilation and influenced by baroreceptor activity, is attenuated in direct relation to exercise intensity (3), there may have been thermal or other components involved in the dilation threshold as the effect appeared to accumulate with successive exercises. Whatever was involved in the attenuation during exercise was apparently still present in recovery if one accepts the explanation that the elevated plateau was caused by reduced vasodilation at the same temperature as dilation had occurred during exercise. However, it was certainly not an effect exclusive to heat load alone as it was not produced by immersion in 44°C water. Nor was the effect present if a more rapid heat reduction was promoted by immersion in 29°C water. Other possible sources for explanation of the effect include changes in plasma volume, osmolarity and hormone concentrations.

CONCLUSIONS

1. PrEx T_{es} , T_{dil} and PoEx T_{es} were linearly related in their increases during successive exercise-recovery cycles,
2. Elevated PoEx T_{es} plateaus were not reproduced by immersion in 44°C water,
3. Elevated PoEx T_{es} plateaus were prevented by immersing the subject in water at 29°C.

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