DYNAMICS OF CARDIORESPIRATORY RESPONSES TO EXERCISE IN HEAT

R. Grucza, Y. Miyamoto and Y. Nakazono

Department of Applied Physiology, Medical Research Centre, PASci, Warsaw, Poland; Department of Information Engineering, Yamagata University, Yonezawa, Japan

INTRODUCTION

At the onset of dynamic exercise blood flow to the exercising muscles increases rapidly due to activation of the sympathetic nervous system and to elevated venous return by muscle pumping action (1). However, exercise and hyperthermia represent conflicting challenges to the cardiovascular system. There is competition between the non-thermally induced vasoconstriction in inactive tissue, in the face of massive vasodilatation in active muscle, and simultaneous attenuation of active vasodilatation by the effect of relaxing homeothermic defence (2). Increases in internal and skin temperatures promote peripheral vascular pooling and transcapillary filtration reducing central venous pressure and thus impairing cardiac filling. The pooled blood may, therefore, affect the kinetics of the cardiovascular responses to dynamic exercise and have some influence on kinetics of the respiratory system (3). The aim of the present work was to test to what extent an increased ambient temperature affects cardiorespiratory dynamics in exercising subjects.

MATERIALS and METHODS

Nine male university students with a mean age of 21.8 years (SD 0.7), height 169 cm (SD 6), and body mass 60.4 kg (SD 8.5) volunteered for this study.

The exercise was performed on a bicycle ergometer in the upright position at an intensity of 50 % VO$_2$ max for 10 min, first at ambient temperature ($T_a$) of 28 °C and, on the next day, at $T_a$ of 37 °C.

Auditory canal temperature ($T_d$) was measured with a probe closely located to the tympanic membrane. The $T_d$ as well as mean skin temperature ($T_s$) on chest, arm and thigh were measured by a thermistor, with an accuracy of 0.1 °C.

Stroke volume (SV), heart rate (HR) and cardiac output (Q) were continuously determined by an automated measuring system based on impedance cardiography (4). Respiratory frequency (f), tidal volume (V$_T$) and minute ventilation (V$_E$) were measured automatically breath-by-breath by a second computer system.
RESULTS

The higher ambient temperature caused greater increases in $T_d$ (0.50 vs 0.40 °C, $p<0.05$) and in $T_e$ (0.70 vs 0.22 °C, $p<0.01$) in the exercising subjects (Fig. 1).

*Ventilatory responses*
Exercise in both ambient temperatures caused similar increases for each of the ventilatory variables. However, $f$ was significantly greater during recovery from the exercise performed at 37 °C than at 28 °C. Acceleration of $f$ during exercise at 37 °C was 3.6 times greater than deceleration from the exercise (0.29 vs 0.08 breaths.s$^{-1}$, respectively). Time constants of $V_T$ and $V_E$ did not differ in the two ambient temperatures but were shorter for ON than for OFF response during exercise at 37 °C (50.1 vs 82.2 s, $p<0.05$ and 55.4 vs 77.2 s, $p<0.05$, respectively) (see Fig. 2).

*Cardiac responses*
No steady-state for HR was observed. The further increase in HR, after 5 min of the exercise, could reflect changes in SV, which after a sudden increase at the beginning of exercise, showed a constant decrease during exercise at 28 °C and 37 °C. As a result $Q$ was almost stable throughout both exercises (see Fig. 3).

The time constant of SV for ON response was faster during exercise at 37 °C than at 28 °C (11.2 vs 15.2 s, $p<0.05$). For all measured cardiac variables deceleration was significantly slower than acceleration. These data indicate that a longer time was necessary for recovery when exercise of the same intensity was performed at higher ambient temperature.

DISCUSSION

The present study showed that increased ambient temperature did not influence the kinetics of the cardiorespiratory response at the beginning of dynamic exercise but the higher ambient temperature significantly increased the time necessary for recovery from the exercise.

The decrease in stroke volume, after a sudden increase at the beginning of exercise, is usually observed when the exercise is performed in the upright position (4). The decrease in SV observed in the present work might be an effect of impaired venous return caused by thermally induced peripheral vasodilatation. The decreased SV was compensated by increased HR resulting in an almost stable value of $Q$ necessary to fulfil metabolic demand during the short time moderate exercise.

The lack of temperature related differences in ventilatory responses during exercise is in agreement with previous data indicating that an increment of about 0.5 °C in body temperatures during moderate exercise does not constitute an extra stimulus to the respiratory system (5). However, greater $f$ observed after exercise performed at 37 °C would suggest that also in humans a rudimentary thermoregulatory reaction in the form
Fig. 1: Increases in mean skin temperature ($T_s$) and deep body temperature ($T_d$) in response to exercise performed at ambient temperature of 28°C (shaded columns) and 37°C (white columns).

Fig. 2: Respiratory frequency, tidal volume, and minute ventilation during dynamic exercises performed with the same relative intensity at two different ambient temperatures. Values are means and SD; *$p<0.05$, paired t-test.
Heart rate, stroke volume, and cardiac output during the dynamic exercises performed at different ambient temperatures. Values are means and SD; *p<0.05, paired t-test.

Fig. 3: Heart rate, stroke volume, and cardiac output during the dynamic exercises performed at different ambient temperatures. Values are means and SD; *p<0.05, paired t-test.

of panting could be triggered in order to increase body heat loss under specific thermal conditions.

REFERENCES