HUMAN MINUTE VENTILATION RESPONSES TO CARBON DIOXIDE LEVELS PRIOR TO AND FOLLOWING AN EXERCISE INDUCED HYPERTHERMIA

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The control of ventilation during exercise is poorly understood. Minute ventilation (V_E) can increase by as much as fifteen times during exercise, while normal metabolic mediators of ventilation are at or below resting levels. Elevated core temperature has been shown as a potential mediator of a hyperpnea in both resting and exercising humans. For resting subjects is not clear, however, if the ventilatory response to carbon dioxide changes with an actively induced hyperthermia, as it is known to occur for passively induce hyperthermia. The purpose of this study was to compare V_E responses to progressively elevated inspired carbon dioxide tensions between pre-exercise resting normothermic and post-exercise resting hyperthermic subjects. Six subjects (mean ± SE, 27.5 ± 1.3 years of age; body weight 72.8 ± 1.6 kg; heights 1.8 ± 0.8 m) performed a modified Read rebreathing test. During rebreathing the initial inspired carbon dioxide was 7%, oxygen was 43% with the balance from nitrogen. Each rebreathing was preceded by 1-2 min of hyperventilation. The rebreathing tests were conducted both before and after performing seated cycle ergometer exercise. Cycle ergometer workloads were increased from rest by 20W/2 min to steady state of 196.6 ± 3.9 W. The highest workloads employed were 73.3 ± 0.3 % of the subjects' previously obtained maximal attainable ergometer workload. The pre-exercise esophageal temperature of 36.62±0.02°C was significantly increased both at the end of exercise by 1.51±0.03°C ($p<0.05$) and during the rebreathing by 0.84±0.05°C ($p<0.05$). The threshold point and slopes of the relationship of V_E plotted as a function of end-tidal carbon dioxide tension ($P_{ET}CO_2$) were compared between the two conditions. From the normothermic to the hyperthermic condition, the $P_{ET}CO_2$ threshold for V_E significantly decreased ($p<0.05$) from 6.95 ± 0.13 kPa to 6.15 ± 0.10 kPa. The slope of the V_E vs. $P_{ET}CO_2$ significantly increased ($p<0.05$) from 15.63 ± 1.28 L/min•kPa for normothermic subjects to 22.48 ± 0.91 L/min•kPa for hyperthermic subjects. In addition, at a given $P_{ET}CO_{2,}$ there was an elevated V_E in the hyperthermic versus normothermic condition. In conclusion, for resting humans previously rendered hyperthermic by exercise, there appears to be both a greater sensitivity to inspired carbon dioxide and a greater level of ventilation. The data supports the hypothesis that respiratory control centre output is increased for resting human subjects previously rendered hyperthermic by exercise.

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