COMPARISON OF CORE TEMPERATURE THRESHOLDS FOR VENTILATION TO A POINT OF INCREASED BLOOD LACTATE ACCUMULATION DURING INCREMENTAL EXERCISE IN HUMANS

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During progressive exercise blood lactate levels begin to increase as the intensity of exercise increases. At higher levels of incremental exercise one view supports that increase in ventilation, relative to metabolic need, is a respiratory compensation for the lactic acidosis. Employing the terminology of Skinner-McLellan-Kinderman this respiratory compensation is initiated at the first ventilation threshold (VT_1) at approximately 30-50% of maximal oxygen consumption. Core temperature thresholds for ventilation during incremental exercise have also been identified (White & Cabanac 1996) and core temperature is suggested to be an additional stimulus to ventilation at levels of exercise greater than about 70% of maximal oxygen consumption. It is not yet known how these core temperature thresholds for ventilation compare to the point of elevated blood lactates during incremental exercise. The purpose of this study was to compare the level of oxygen consumption (VO_2) at the core temperature threshold for ventilation to that at the point of blood lactate accumulation during incremental exercise. Six fit male college-aged subjects pedaled a cycle ergometer on 2 occasions in incremental exercise tests until the point of exhaustion. In one session the power output was increased by 20W/2 min (slow ramp) and in the other by 40W/2 min (fast ramp). Subjects were instrumented for esophageal temperature (T_{es}), skin temperatures and their expired gases were collected to assess VO_2, carbon dioxide production (VCO_2) and minute ventilation (V_E). At each workload two arterialized blood samples were taken from the tip of the finger and subsequently analysed for blood lactate concentrations. In both exercise sessions, ventilatory equivalents for oxygen consumption (V_E/VO_2) and carbon dioxide production (V_E/VCO_2) were plotted as a function of both T_{es} and VO_2. From 2 independent observations of these plots (or by 3 if a discrepancy was evident) the VO_2 at the T_{es} threshold for ventilation in each exercise session was assessed. In addition, visual inspection of scatterplots of blood lactate levels versus VO_2 allowed determination of the VO_2 at the 2-mmol blood lactate point. In the slow ramp session the VO_2 of 1.51±0.14 l/min at the 2-mmol point was significantly less than both the VO_2 of 2.85±0.21 l/min ($p<0.001$) at the T_{es} threshold for V_E/VO_2 and the VO_2 of 2.44± 0.19 l/min at the T_{es} threshold V_E/VCO_2 ($p<0.001$). This was also evident in the fast ramp session when the VO_2 of 0.86 ± 0.12 l/min at the 2mmol point of lactate accumulation was significantly less than the VO_2 at the T_{es} thresholds for V_E/VO_2 ($p <0.001$) and for V_E/VCO_2 ($p <0.001$). In conclusion, the VO_2 at the 2-mmol point of blood lactate accumulation is significantly less than the VO_2 observed at the esophageal temperature threshold for ventilation during incremental exercise to maximal attainable work-rates. The results support that the accumulation of blood lactate and the T_{es} thresholds for ventilation are separate events with potentially different metabolic and/or neural origins.

White, M.D. & Cabanac, M. (1996) Exercise Hyperpnea and hyperthermia in human. *J. Appl. Physiol.* 81, 1249-1254.

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