Kinetics of Mixed Venous CO₂ Pressure in Constant-Load Exercise

Tokuo Yano¹⁾, Yasuhito Kumazaki²⁾ and Katsumi Asano²⁾

1) Laboratory of Human Movement Science, Faculty of Education, Hokkaido University

2) Institute of Health and Sports Science, Tsukuba University

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We have studied the kinetics of mixed venous CO₂ pressure ($\bar{P}\text{vco}_2$) at constant work rates of 300, 450 and 600 kpm/min (Yano, 1986). The $\bar{P}\text{vco}_2$ increased at the onset of exercise, and reached a steady state at 300 and 450 kpm/min. However, at 600 kpm/min, the $\bar{P}\text{vco}_2$ indicated a relatively high steady state in comparison with those at other work rates, and decreased after 5 min of exercise. This overshoot may be related to an early increase of lactate at the onset of exercise, since this phenomenon was only observed at the highest exercise intensity of the study. However, lactate was not measured in this study.

The purpose of the present study is, therefore, to examine if $\bar{P}\text{vco}_2$ overshoots because of lactate increase during exercise.

Methods

Four healthy male subjects were studied (age 25 ± 3.1 years, height 173 ± 8.75 cm, body weight 69 ± 1.5 kg, and maximal oxygen uptake 3.08 ± 0.298 l/min). All subjects were informed of the purpose of the experiment, the procedure, and the risks before their consent was obtained.

The exercise test was performed using a Monark bicycle ergometer with a pedaling rate of 50 rpm. On the first day, the exercise was performed incrementally to obtain maximal oxygen uptake. On the second day, the constant-load exercise with 750 kpm/min for 12 min was repeated four times at intervals of over two hours. Oxygen uptake and carbon dioxide output were determined by the Douglas bag method with exercise performed incrementally until exhaustion and at the first run of constant-load exercise. The end tidal CO₂ fraction was measured and blood was taken from a finger tip for lactate analysis at the first run of constant-load exercise. CO₂ rebreathing was carried out at 5-6 and 11-12 min on the second run, 3-4 and 9-10 min on the third run, and 1-2 and 7-8 min on the fourth run.

O₂ and CO₂ were measured by a mass spectrometer MGA-1100 (Perkin Elmer). Blood lactate was measured by lactate analyzer Model 23 L (YSI). Mixed venous CO₂ pressure ($\bar{P}\text{vco}_2$) was determined by the Defares method(1961). Arterial CO₂ pressure ($P\text{aco}_2$) was calculated from the data of tidal volume (V_T) and end tidal CO₂ pressure (P_{ETCO_2}) using the following regression line (Jones et al., 1979).

$$P\text{aco}_2 = 5.5 + 0.90 \cdot P_{\text{ETCO}_2} - 2.1 \cdot V_T$$

Results and Discussion

As shown in Fig. 1, $\bar{P}\text{vco}_2$ increased until 3-4 min and seemed to decrease. However, $\bar{P}\text{vco}_2$ after 3 min of exercise was not significantly related to time lapse ($r = -0.284$, NS, $n=16$). $P\text{aco}_2$ during exercise did not radically change. The respiratory gas exchange ratio (R) indicated temporal low values at the onset of exercise and increased until 2-3 min. Afterwards the rate of increase lessened and indicated a peak value at 5 min. Then R decreased to reach a steady state value. Blood lactate increased until 5-6 min and reached a fairly steady state.

CO₂ production from energy metabolism increases at the onset of exercise. The CO₂ is partly stored and the rest is expired out of the body. Since an increase of stored CO₂ can bring a rise of $\bar{P}\text{vco}_2$ (Yano, 1986), the CO₂ pressure gradient from the body to ambient air increases at the onset of exercise. As a result, CO₂ expiration increases. When this CO₂ expiration increases up to the level of CO₂ production after the onset of exercise, the CO₂ stores and $\bar{P}\text{vco}_2$ could reach a steady state.

Since, as mentioned above, the CO₂ expiration is less than the CO₂ production at the onset of exercise, a temporal decrease of R can be observed (Yano, 1986). R can also be affected by lactate formation (Wasserman and Casaburi, 1991). Therefore, this temporal decrease of R should include the effects of lactate. Also, the slow

increase of R above the later steady state value in the present study is considered to be mainly affected by lactate formation.

Lactic acid produced in exercise is partly buffered by a bicarbonate system (Hultman and Shalin, 1980). In this chemical reaction ($\text{CO}_2 + \text{H}_2\text{O} \rightleftharpoons \text{HCO}_3^- + \text{H}^+$), HCO_3^- can shift towards CO_2 . The shifted CO_2 remains in the closed system, while in the open system the shifted CO_2 is eliminated from the system. Although the human body is basically an open system, a resistance factor between the body and ambient air in CO_2 pressure gradient can act as a closed system. Therefore, CO_2 is not immediately expired from human body. This could cause a temporal CO_2 rise.

Thus, the effect of lactate formation could overlap with the $\text{P}\bar{\text{V}}\text{CO}_2$ kinetics due to stored CO_2 during exercise. Therefore, $\text{P}\bar{\text{V}}\text{CO}_2$ may happen to overshoot as former studies have reported (Davies et al., 1972; Yano, 1986). However, the present study could not confirm the overshoot of $\text{P}\bar{\text{V}}\text{CO}_2$.

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Correspondence to: Tokuo Yano, Faculty of Education, Hokkaido University, Sapporo 060, Japan

Fig. 1 Means and SD on mixed venous CO_2 pressure ($\text{P}\bar{\text{V}}\text{CO}_2$), arterial CO_2 pressure (Paco_2), blood lactate (La) and respiratory gas exchange ratio (R) in constant-load exercise. * represents significance of differences between the last and another means in each parameter. The significance of differences was assessed with Student's paired t test ($p < 0.05$).