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Relationship between Blood Lactate Concentration and Excessive CO₂ Expiration During and after Ramp Exercise

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Abstract

YUNOKI, T., OGATA, H. and YANO, T., Relationship between Blood Lactate Concentration and Excessive CO₂ Expiration During and after Ramp Exercise. Adv. Exerc. Sports Physiol., Vol.9, No.3 pp.97-103, 2003. The purpose of this study was to examine the kinetics of excessively expired CO₂ and the blood lactate concentration ([La⁻]) during and after ramp exercise. Six healthy males performed three different ramp exercise tests in which the work rate was increased as a ramp function at 10 W/min (R10), 20 W/min (R20) and 40 W/min (R40). Excessive CO₂ expiration during exercise (CO₂ excess) significantly correlated with the [La⁻] increase during exercise (Δ [La⁻]). Excessive CO₂ expiration during recovery (post CO₂ excess) significantly correlated with the [La⁻] increase during recovery (post Δ [La⁻]). However, CO₂ excess in R40 was significantly lower compared with R20 and R10, despite there being no significant difference in Δ [La⁻] among the three tests. In addition, post CO₂ excess showed a positive value even when post Δ [La⁻] was zero. End-tidal CO₂ pressure (PETCO₂) decreased from the onset of respiratory compensation to about 10 min post-exercise in all tests. PETCO₂ decrement during exercise tended to be greater in R10 than in R40, and PETCO₂ decrement during post-exercise tended to be greater in R40 than in R10. PETCO₂ was significantly related to [La⁻] during both at exhaustion and at 10 min post-exercise. However, PETCO₂ at the same [La⁻] was lower at 10 min post-exercise than at exhaustion. Furthermore, alactic O₂ debt, which was calculated from O₂ uptake kinetics during recovery, was significantly higher in R40 than in the other tests. These results suggest that there is a case in which the blood lactate is not always the cause of excessive CO₂ expiration in response to ramp exercise. The decrease in PETCO₂ and the H⁺ uptake-release by breakdown-resynthesis of phosphocreatine were thought to be main factors influencing the excessive CO₂ expiration as well as the [La⁻] increase.

Keywords: Ramp exercise, Lactic acid, Carbon dioxide, End-tidal CO₂ pressure, Phosphocreatine

Introduction

During exercise above certain levels, lactic acid production becomes greater than lactic acid removal, and lactic acid accumulates in the body. Since a large part of accumulated lactic acid is ionized, accumulation of lactic acid is thought to be the main factor yielding metabolic acidosis (decreasing pH) during and after intense exercise. It is well known that the body restrains the fall of pH by bicarbonate buffering ($H^+ + HCO_3^- \rightleftharpoons H_2O + CO_2$) of the hydrogen ion (H⁺) dissociated from lactic acid, and that the buffering effect does not increase unless excessively generated CO₂ in the buffering process is removed by breathing. Therefore, expiring the excessively generated CO₂ is important for maintaining a constant pH value during and after exercise.

In order to estimate the excessive CO₂ expiration related to the bicarbonate buffering of lactic acid during exercise, an incremental exercise test has mainly been used. In incremental exercise, it has been demonstrated that an increase in the lactic acid involves a decrease in the bicarbonate ion (HCO₃⁻), inducing excessive CO₂ expiration due to bicarbonate buffering of lactic acid (2, 6, 24). Furthermore, it has recently been reported that the kinetics of excessively expired CO₂ and lactate ion (La⁻) increase or HCO₃⁻ decrease in the blood are similar during incremental exercise (11, 19). These results suggest that in order to induce excessive CO₂ expiration, lactic acid needs to be diffused from active muscle to the blood and to be buffered within the blood.

Generally, when doing incremental exercise tests (i.e., the exercise time to exhaustion being 5-30 minutes), the peak value of blood La⁻ concentration is normally observed several minutes after exhaustion (15). If the kinetics of excessively expired CO₂ and La⁻ increase or HCO₃⁻ decrease in the blood are similar, as reported by previous studies (11, 19), excessive CO₂ expiration must be induced even after incremental exercise. The relationship between La⁻ and excessive CO₂ expiration during recovery has not been previously examined, but examination of this relationship in recovery may contribute to an understanding of the factors influencing CO₂ elimination during exercise as well

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as during recovery.

The lactic acid produced during exercise is partly diffused from active muscle to the blood, and La^- concentration will reach equilibrium between active muscle and the blood. Nevertheless, complete equilibration should not be expected even during quite low intensity exercise. In incremental exercise tests, the equilibration of La^- would not be seen during exercise, but instead it is assumed it would be seen several minutes after exercise (13). This implies that the extent of the equilibration of La^- is affected by the exercise time to exhaustion. For example, if the work rate were increased rapidly, La^- would not have enough time to diffuse from active muscle into the blood, since the exercise time to exhaustion would decrease. In this case, it is likely that La^- diffusion would increase during recovery. On the other hand, if the work rate were increased slowly, exercise time to exhaustion would increase. Because of this, La^- diffusion would increase during exercise, but decrease during recovery. In the present study, we therefore examined the kinetics of the excessive CO_2 expiration and the blood La^- concentration during and after ramp exercise by changing the incrementation rate.

Materials and methods

Six healthy male volunteers were used as subjects for this study. The mean age, height and weight of the subjects were 21.3 ± 2.6 yrs, 170.3 ± 6.4 cm and 59.6 ± 5.9 kg, respectively. Written voluntary consent to participate in this study was obtained from all subjects after informing them of the purpose, procedure, and possible risks of the experiment, and the study was carried out by referring to the ethical standard of ethics committee at our university and the Helsinki Declaration.

A bicycle ergometer in which the load can be adjusted by a computer (Combi 232C) was used. After a 5-min rest period, each subject completed 3 different ramp exercise tests in randomized order on separate days. The tests consisted of a 10 W/min ramp slope (R10), a 20 W/min ramp slope (R20) and a 40 W/min ramp slope (R40). After 4 min of unloaded pedalling (about 6 W) at 60 rpm, the work rate was increased in ramp mode until the subject could no longer maintain the rotation rate of 60 rpm. After exhaustion, each subject remained in a resting state for 30 min.

Ventilation and gas exchange responses were measured from the resting period (5 min) before unloaded pedalling to 30 min after exhaustion by an on-line computerized breath-by-breath method (AE-280S, Minato Medical Science). Inspired and expired gas volumes were measured using a hot-wire respiratory flow system. Flow signals were electrically integrated for the duration of each breath to calculate minute ventilation (\dot{V}_E). A 2-liter syringe was used to calibrate the system, which was linear throughout a flow range of 0-600 l/min of \dot{V}_E . The expired fraction of O_2 and CO_2 were analyzed using a zirconium solid electrolyte oxy-

gen analyzer and an infrared carbon dioxide analyzer, respectively. O_2 uptake ($\dot{V}\text{O}_2$), CO_2 output ($\dot{V}\text{CO}_2$), end-tidal O_2 pressure (PETO_2) and end-tidal CO_2 pressure (PETCO_2) were then calculated for each 20-sec interval. The gas analyzer was calibrated by known standard gases. Blood samples (25 μl) were collected from fingertips using capillary tubes and analyzed using a lactate analyzer (YSI-1500 sport, YSI) to measure the blood La^- concentration ($[\text{La}^-]$). Blood was sampled at rest, exhaustion, and 3, 5, 10, 20, 30 min post-exercise. The lactate analyzer was calibrated by a standard lactate solution of 5 mM before each exercise test. The subject's hand was pre-warmed in water of about 45 $^\circ\text{C}$ to improve circulation of the blood-sampling site. The increase in $[\text{La}^-]$ from rest to exhaustion was expressed as $[\text{La}^-]$ increase during exercise ($\Delta[\text{La}^-]$) and the increase in $[\text{La}^-]$ observed after exhaustion was expressed as $[\text{La}^-]$ increase post-exercise (Post $\Delta[\text{La}^-]$).

Anaerobic threshold (AT) was estimated using the following gas exchange criteria: (i) an increase in $\dot{V}\text{CO}_2$ relative to $\dot{V}\text{O}_2$, (ii) an increase in $\dot{V}E/\dot{V}\text{O}_2$ without an increase in $\dot{V}E/\dot{V}\text{CO}_2$ and (iii) an increase in PETO_2 without a decrease in PETCO_2 (17, 21). In addition, the onset of respiratory compensation (RCP) was determined by (i) an increase in $\dot{V}E$ relative to $\dot{V}\text{CO}_2$ and (ii) an increase in ventilatory equivalent for $\dot{V}\text{CO}_2$ ($\dot{V}E/\dot{V}\text{CO}_2$) (1, 12, 22).

Excessive expiration of CO_2 during exercise was calculated according to previous studies (5, 6, 8, 24, 25). Aerobically produced $\dot{V}\text{CO}_2$ was estimated by extrapolating the $\dot{V}\text{CO}_2$ - $\dot{V}\text{O}_2$ regression line ($\dot{V}\text{CO}_2 = S_1 \cdot \dot{V}\text{O}_2 - a$) below AT up to the end of exercise. The difference between the actual $\dot{V}\text{CO}_2$ and aerobically produced $\dot{V}\text{CO}_2$ was integrated from AT to the end of exercise, and this was defined as CO_2 excess:

$$\text{CO}_2 \text{ excess} = \int (\dot{V}\text{CO}_2 - (S_1 \cdot \dot{V}\text{O}_2 - a)) dt$$

Furthermore, excessive expiration of CO_2 during recovery was calculated (26) according to the method of Cerretelli and Di Prampero (4). By assuming that the respiratory quotient (RQ) is equal to 1.0, the difference between $\dot{V}\text{CO}_2$ and $\dot{V}\text{O}_2$ was integrated from the end of exercise to the time (on average, at 7 min post-exercise) at which the difference became zero [respiratory exchange ratio (R)=1.0], and this was defined as post CO_2 excess:

$$\text{Post CO}_2 \text{ excess} = (\dot{V}\text{CO}_2 - \dot{V}\text{O}_2 \cdot \text{RQ}) dt$$

A double exponential model was fitted to $\dot{V}\text{O}_2$ data during recovery (16). Model parameters were determined by least-squares nonlinear regression, in which the closest fit was defined by minimization of the chi-square:

$$\Delta\dot{V}\text{O}_2(t) = \alpha \cdot \exp(-t/\tau_1) + \beta \cdot \exp(-t/\tau_2)$$

where $\Delta\dot{V}\text{O}_2(t)$ is the $\dot{V}\text{O}_2$ above the resting value at t -sec after the end of exercise, α and β are the asymptotic values, and τ_1 and τ_2 are the time constants for each exponential term. To estimate the magnitude of phosphocreatine (PCr) resynthesis during recovery, alactic O_2 debt was cal-

culated from the product of α and τ_1 :

$$\text{alactic O}_2 \text{ debt} = \alpha \cdot \tau_1$$

Repeated-measures ANOVA was used to evaluate possible differences in the variables obtained in the 3 ramp exercise tests. The Tukey multiple comparison procedure was used to extract pairs whose mean values were different. The strength of the relationship between 2 variables was expressed by the correlation coefficient of Pearson. Differences were considered significant at $P < 0.05$. Results were expressed as means \pm SD.

Results

Peak $\dot{V}O_2$ ($\dot{V}O_{2\text{peak}}$) was similar in all tests in spite of the significant differences in time to exhaustion and maximum work rate (WR_{max}) (Table 1). There was no significant difference in percentage (%) of the $\dot{V}O_2$ at AT ($\dot{V}O_{2\text{AT}}$) to the $\dot{V}O_{2\text{peak}}$ among the 3 tests (R10, $52.7 \pm 3.7\%$; R20, $51.8 \pm 9.2\%$; R40, $54.5 \pm 4.7\%$). On the other hand, % of the $\dot{V}O_2$ at RCP ($\dot{V}O_{2\text{RCP}}$) to the $\dot{V}O_{2\text{peak}}$ was significantly higher ($P < 0.05$) in R40 ($91.9 \pm 4.6\%$) than in the R20 ($81.5 \pm 6.1\%$) and in the R10 ($79.4 \pm 5.7\%$). The time from AT to RCP and the time from AT to exhaustion significantly decreased with the increasing incrementation rate (Table 1). Peak $\dot{V}E$ ($\dot{V}E_{\text{peak}}$) was similar in all

tests (R10, 89.3 ± 17.3 l; R20, 97.7 ± 23.3 l; R40, 92.5 ± 14.1 l) but peak $\dot{V}CO_2$ ($\dot{V}CO_{2\text{peak}}$) was significantly different ($P < 0.05$) between R10 and R40 (R10, 2.87 ± 0.24 l; R20, 3.18 ± 0.29 l; R40, 3.27 ± 0.26 l).

As shown in table 2, $[La^-]$ peaked until 5 min post-exercise from exhaustion, and subsequently decreased gradually. A significant difference in $[La^-]$ among the 3 tests was not observed at any measurement time. In addition, there was no significant difference in peak value for $[La^-]$ ($[La^-]_{\text{peak}}$) among the 3 tests. However, the time at which $[La^-]$ peaked in R10 was different from that in the other tests. That is, $[La^-]_{\text{peak}}$ was observed at exhaustion in R10 (in 5 of 6 subjects) but at 3-5 min post-exercise in R20 and R40 (in 5 of 6 subjects). Because of this, the increase in $[La^-]$ during recovery (post $\Delta[La^-]$), calculated as the difference between $[La^-]$ at exhaustion ($[La^-]_{\text{exhaustion}}$) and $[La^-]_{\text{peak}}$, increased with the increasing incrementation rate, resulting in a significant difference ($P < 0.05$) in post $\Delta[La^-]$ between R40 (0.80 ± 0.54 mM) and R10 (0.04 ± 0.10 mM).

To estimate the magnitude of PCr resynthesis, a double exponential function was fitted to $\dot{V}O_2$ data during recovery. Goodness of fit was good judging from the coefficient of determination (r^2), which was between 0.949

Table 1 Exercise performance and oxygen uptake at exhaustion, anaerobic threshold (AT), and onset of respiratory compensation (RCP).

Ramp	R10	R20	R40
WRmax (watts)	222 \pm 37	249 \pm 35 ^a	282 \pm 29 ^{b,c}
Time to exhaustion (min)	22.4 \pm 3.6	12.6 \pm 1.7 ^a	7.2 \pm 0.7 ^{b,c}
AT-Exhaustion (min)	11.4 \pm 2.4	6.7 \pm 2.0 ^a	3.6 \pm 0.5 ^{b,c}
AT-RCP (min)	6.6 \pm 1.7	4.1 \pm 1.4	2.9 \pm 0.4 ^{b,c}
$\dot{V}O_{2\text{peak}}$ (l/min)	2.67 \pm 0.31	2.73 \pm 0.31	2.67 \pm 0.27
$\dot{V}O_{2\text{AT}}$ (l/min)	1.39 \pm 0.16	1.41 \pm 0.25	1.45 \pm 0.18
$\dot{V}O_{2\text{RCP}}$ (l/min)	2.11 \pm 0.20	2.18 \pm 0.32	2.46 \pm 0.35 ^b

Values are means \pm SD of 6 subjects. WRmax, work rate at exhaustion; Time to exhaustion, time from onset of ramp exercise to exhaustion; AT-Exhaustion, time from AT to exhaustion; AT-RCP, time from AT to RCP; $\dot{V}O_{2\text{peak}}$, oxygen uptake at exhaustion; $\dot{V}O_{2\text{AT}}$, oxygen uptake at AT; $\dot{V}O_{2\text{RCP}}$, oxygen uptake at RCP; a, significantly different from R10 ($p < 0.05$); b, significantly different from R10 ($p < 0.05$); c, significantly different from R20 ($p < 0.05$).

Table 2 Blood lactate concentrations (mM) in three ramp exercise tests.

Ramp	Rest	Exhaustion	Recovery			Peak
			3 min	5 min	10 min	
R10	0.94 \pm 0.18	7.40 \pm 1.73	6.63 \pm 1.67	6.26 \pm 1.57	5.17 \pm 1.42	7.44 \pm 1.68
R20	0.83 \pm 0.20	7.65 \pm 2.10	7.71 \pm 2.17	7.48 \pm 2.67	6.27 \pm 2.33	8.00 \pm 2.25
R40	0.98 \pm 0.25	7.11 \pm 1.42	7.73 \pm 1.93	7.51 \pm 1.94	6.55 \pm 1.82	7.91 \pm 1.65

Values are means \pm SD of 6 subjects.
Peak, means \pm SD of peak values obtained by each subject.

and 0.986. The τ_1 in R40 (54.3 ± 7.0 s) was significantly higher ($P < 0.05$) compared with R20 (43.1 ± 5.5 s) and R10 (48.1 ± 6.4 s). Similarly, alactic O_2 debt was significantly higher ($P < 0.05$) in R40 (2.15 ± 0.56 l) than in R20 (1.56 ± 0.34 l) and R10 (1.76 ± 0.27 l). The alactic O_2 debt significantly correlated ($r = 0.685$, $p < 0.01$) to the maximum work rate (WR_{max}), suggesting that PCr breakdown grew larger as the work rate at exhaustion became higher.

CO_2 excess significantly correlated with $\Delta [La^-]$ ($[La^-]_{exhaustion} - [La^-]_{rest}$) ($r = 0.871$, $p < 0.001$, Fig. 1), suggesting that, overall, CO_2 excess is dependent on the increase in $\Delta [La^-]$. However, looking at the results of the 3 tests separately, CO_2 excess significantly lower when the incrementation rate was higher ($P < 0.05$, Table 3), in spite of there being no significant difference in $\Delta [La^-]$ among the 3 tests. As shown in Table 3, the slope (S_1) of the $\dot{V}CO_2 - \dot{V}O_2$ relationship below AT was significantly lower in R40 than in the other tests ($P < 0.05$, Table 3). In contrast, the slope (S_2) above AT was significantly higher in R40 than in the other two tests ($P < 0.05$, Table 3). Consequently, the higher the incrementation rate, the greater the CO_2 excess per unit time. However, because time to exhaustion shortened as the incrementation rate increased, CO_2 excess was

significantly greater when the incrementation rate was lower. On the other hand, post CO_2 excess was greater when the incrementation rate was higher. As a result, the post CO_2 excess obtained in R40 was significantly higher ($P < 0.05$) compared with R10 (Table 3). Similarly, the post $\Delta [La^-]$ was significantly higher ($P < 0.05$) in R40 than in R10. Because of this, post CO_2 excess was significantly correlated with post $\Delta [La^-]$ ($r = 0.765$, $p < 0.001$, Fig. 2). Nevertheless, as shown in Figure 2, post CO_2 excess was about 1.0 l when post $\Delta [La^-]$ was zero.

Table 4 shows $PETCO_2$ during exercise and subsequent recovery. $PETCO_2$ rose from the start of incremental exercise to AT, plateaued from AT to RCP, and fell from RCP to at about 10 min post-exercise in all tests. There was no significant difference in the $PETCO_2$ values among the 3 tests at any measurement time. However, $PETCO_2$ at exhaustion tended to be higher during higher incrementation rate exercise. Therefore, the fall in $PETCO_2$ from AT to exhaustion tended to be greater in R10 (-7.4 ± 6.9 torr) than in R40 (-2.6 ± 3.9 torr), and the fall in $PETCO_2$ from exhaustion to 10 min post-exercise tended to be greater in R40 (-12.4 ± 3.0 torr) than in R10 (-7.5 ± 5.7 torr). Figure 3 shows the $PETCO_2 - [La^-]$ relationships at exhaus-

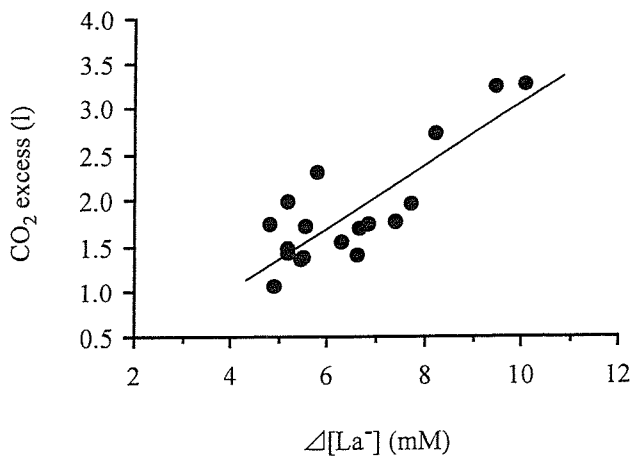


Fig. 1 Relationship between the amounts of excessive CO_2 expiration (CO_2 excess) and the increase in blood lactate concentration ($\Delta [La^-]$) during exercise.

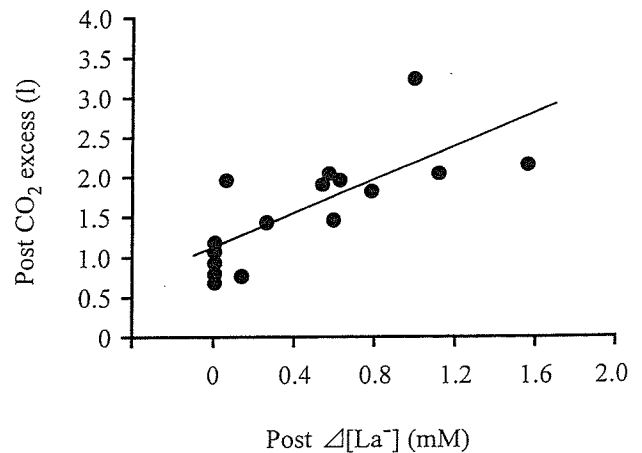


Fig. 2 Relationship between the amounts of excessive CO_2 expiration (post CO_2 excess) and the increase in blood lactate concentration (post $\Delta [La^-]$) post-exercise.

Table 3 Slopes of $\dot{V}CO_2 - \dot{V}O_2$ relationship and amounts of excessive CO_2 expiration.

Ramp	R10	R20	R40
S_1	1.00 ± 0.04	0.99 ± 0.07	$0.88 \pm 0.05^{b,c}$
S_2	1.23 ± 0.11	1.40 ± 0.13^a	$1.58 \pm 0.19^{b,c}$
CO_2 excess (l)	2.04 ± 0.66	1.98 ± 0.67	$1.66 \pm 0.57^{b,c}$
Post CO_2 excess (l)	1.03 ± 0.27	1.61 ± 0.50	1.99 ± 0.77^b

Values are means \pm SD of 6 subjects. S_1 and S_2 , slopes of the $\dot{V}CO_2 - \dot{V}O_2$ relationship below and above anaerobic threshold, respectively; CO_2 excess and Post CO_2 excess, amounts of excessive CO_2 expiration during exercise and recovery, respectively; a, significantly different from R10 ($p < 0.05$); b, significantly different from R10 ($p < 0.05$); c, significantly different from R20 ($p < 0.05$).

tion and at 10 min post-exercise, respectively. Both at exhaustion ($r = -0.579, p < 0.01$) and at 10 min post-exercise ($r = -0.744, p < 0.001$), there were significant correlations in which PETCO₂ decreased with an increase in [La⁻]. However, when compared at the same [La⁻], PETCO₂ during recovery was lower than that at exhaustion (Fig. 3).

Discussion

In this study, three ramp exercises were performed in order to examine the kinetics of the excessive CO₂ expiration and blood lactate concentration during and after exercise. The important findings in this study are as follows; 1) Although excessive CO₂ expiration during exercise (CO₂ excess) was significantly related to the increase in [La⁻] during exercise ($\Delta[La^-]$), the value of CO₂ excess was significantly different due to the difference in incrementation rate in spite of the fact that $\Delta[La^-]$ was similar in the 3 tests. 2) Although excessive CO₂ expiration during recovery (post CO₂ excess) was significantly related to the increase in [La⁻] during recovery (post $\Delta[La^-]$), the post CO₂ excess showed a positive value even when post $\Delta[La^-]$ was zero. These results suggest that there is a case

in which the blood lactate is not always the cause of excessive CO₂ expiration in response to ramp exercise.

A. Calculation of excessive CO₂ expiration

Excessive CO₂ expiration during exercise and post-exercise was calculated by different methods with regard to estimating RQ, respectively. RQ progressively becomes larger as work rate increases with the change of metabolic substrate used by active muscle (20). Therefore, the $\dot{V}CO_2 - \dot{V}O_2$ relationship ($\dot{V}CO_2 = S_1 \cdot \dot{V}O_2 - a$) below AT was used for the calculation of excessive CO₂ expiration during exercise (CO₂ excess). The quotient $[S_1 - (a/\dot{V}O_2)]$ obtained from dividing this $\dot{V}CO_2 - \dot{V}O_2$ relationship by $\dot{V}O_2$ corresponds to RQ. This value $[S_1 - (a/\dot{V}O_2)]$ increases with an increase in work load (increase in $\dot{V}O_2$) and then approaches the value of S_1 . On the other hand, since $\dot{V}O_2$ during recovery is lower than that during exercise, if the $\dot{V}CO_2 - \dot{V}O_2$ relationship during exercise is applied to the recovery phase, the value of RQ is lower. However, since increased La⁻ after exercise inhibits the mobilization of fatty acids (9, 10), RQ does not decrease too much. Thus, RQ is thought to be very nearly 1.0, at least for 7 min after exercise in which post CO₂ excess is observed. Therefore, by assuming that RQ in the recovery phase is equal to 1.0, excessive CO₂ expiration during post-exercise (post CO₂ excess) can be calculated.

The slope (S_1) of the $\dot{V}CO_2 - \dot{V}O_2$ relationship below AT was lowest in R40. Ward and Whipp (23) observed that R and S_1 decreased when work rate was incremented rapidly, and this phenomenon was interpreted as the effect of the metabolically produced CO₂ being stored in the body rather than being excreted by the lungs. Furthermore, it has been demonstrated that S_1 decreases if CO₂ stores are reduced by hyperventilation before exercise (17). This decrease in S_1 is thought to be because CO₂ is stored in the body in order to retake the previously reduced CO₂ stores. Therefore, under these conditions, PETCO₂ increases abruptly compared with the conditions without prior hyperventilation (17). In the present study, CO₂ stores were not effected because an abrupt increase in PETCO₂ was not observed during the period from the start of ramp exercise to AT in R40.

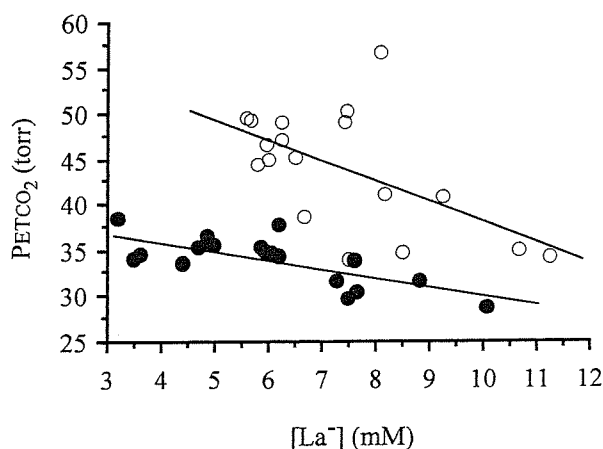


Fig. 3 Relationships between PETCO₂ and blood lactate concentration ([La⁻]) at exhaustion (○) and at 10 min post-exercise (●).

Table 4 PETCO₂ (torr) in three ramp exercise tests.

Ramp	Exercise					Recovery		
	Rest	Unloaded	AT	RCP	Exhaustion	3min	5min	10min
R10	38.5±3.2	40.4±2.4	49.3±4.4	48.4±5.1	41.9±6.9	36.6±3.0	34.6±2.3	34.4±2.1
R20	39.6±3.3	41.1±3.2	50.8±2.8	50.9±4.8	44.0±8.6	38.5±5.0	35.2±4.3	34.4±4.1
R40	37.7±2.3	38.8±1.9	48.5±2.4	49.9±4.4	45.9±4.1	38.0±4.7	34.7±2.2	33.5±1.3

Values are means ± SD of 6 subjects.

Unloaded, unloaded pedalling (about 6 W).

B. Excessive CO₂ expiration during exercise

PETCO₂ correlated with [La⁻] during exercise. However, because there was no significant difference in [La⁻] increase during exercise (Δ [La⁻]) among the 3 tests, the difference in PETCO₂ reduction at exhaustion among the 3 tests is attributable to the difference in time to exhaustion. It has been suggested that when the lactic acidosis is more prolonged, with time, a secondary role of the central respiratory chemoreceptor is added to the dominant role of the peripheral respiratory chemoreceptor (carotid body) in order to compensate for the continuing metabolic acidosis, through a slow influx of H⁺ into the brain extracellular fluid (18). Although in the present study it cannot be confirmed that the above mechanism occurred, it seems likely that the magnitude of respiratory compensation increased with an increase in exercise time since the magnitude of PETCO₂ reduction at exhaustion tended to be greater in lower incrementation rate exercise. PETCO₂ reduction helps to widen the venoarterial CO₂ difference, leading to an increase in the elimination of CO₂ from venous blood as well as tissues. Thus, the difference in the PETCO₂ reduction will lead to a difference in excessive CO₂ expiration resulting from respiratory compensation. It is likely, therefore, that this is a factor that lead to the difference in CO₂ excess despite there being no significant difference in Δ [La⁻] among the 3 tests.

WR_{max} was the highest in R40 although $\dot{V}O_{2peak}$ and [La⁻]_{peak} were similar among the 3 tests. This indicates the possibility that there was a difference in the contribution of a high-energy phosphate system to muscle metabolism during each test although the contributions of the aerobic system and the lactic system were similar among the 3 tests. It is reported that PCr decreases linearly with an increase in work rate during incremental exercise (3). In the present study, it was found that alactic O₂ debt significantly correlated with WR_{max}. Therefore, the difference in alactic O₂ debt will reflect the difference in the magnitude of breakdown or resynthesis of PCr.

It is known that PCr breakdown is an important buffering action (PCr + H⁺ + ADP → ATP + Cr) (7, 14). Thus, a part of the increased H⁺ due to lactic acid production is consumed by the action. In the present study, since Δ [La⁻] in the 3 tests were similar, it was thought that there was no significant difference in the increase in H⁺ related to lactic acid. However, judging from the alactic O₂ debt, H⁺ consumed by the PCr breakdown would be higher in R40 compared with the other ramp exercise tests. Therefore, in R40, the percentage of H⁺ buffered by HCO₃⁻ will decrease, resulting in a decrease in CO₂ excess.

C. Excessive CO₂ expiration after exercise

Although there was a correlation between PETCO₂ and [La⁻] post-exercise as well as during exercise,

PETCO₂ at the same [La⁻] value was lower post-exercise than during exercise. This suggests that respiratory compensation will continue after exercise, resulting in excessive CO₂ expiration related not only to buffering, but also to respiratory compensation. Furthermore, after exercise, PCr will be resynthesized and H⁺ released. It has been suggested that the released H⁺ due to PCr resynthesis is rapidly diffused from muscle to the blood (14). Since a dominant buffering system in extracellular fluid is the bicarbonate system, diffused H⁺ will be buffered by HCO₃⁻ and then excessive CO₂ expiration will be induced. Therefore, post CO₂ excess will show a positive value even when post Δ [La⁻] is zero. Post CO₂ excess will then increase with higher ramp exercise since H⁺ released by the PCr resynthesis would be higher in R40 compared with the other ramp slopes as indicated by the alactic O₂ debt.

From the results of the present study, it is suggested that there is a case in which the blood lactate is not always the cause of excessive CO₂ expiration in response to ramp exercise. The respiratory compensation and the H⁺ uptake-release by PCr breakdown-resynthesis were thought to be main factors influencing the excessive CO₂ expiration as well as the [La⁻] increase.

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