

**Relationship between Hyperventilation and Excessive
CO₂ Output during Recovery from Repeated Cycling
Sprints**

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Summary

The purpose of the present study was to examine whether excessive CO₂ output ($\dot{V}_{CO_2\text{excess}}$) is dominantly attributable to hyperventilation during the period of recovery from repeated cycling sprints. A series of four 10-sec cycling sprints with 30-sec passive recovery periods was performed two times. The 1st series and 2nd series of cycle sprints (SCS) were followed by 360-sec passive recovery periods (1st recovery and 2nd recovery). Increases in blood lactate (ΔLa) were 11.17 ± 2.57 mM from rest to 5.5 min during 1st recovery and 2.07 ± 1.23 mM from the start of the 2nd SCS to 5.5 min during 2nd recovery. CO₂ output (\dot{V}_{CO_2}) was significantly higher than O₂ uptake (\dot{V}_{O_2}) during both recovery periods. This difference was defined as $\dot{V}_{CO_2\text{excess}}$. $\dot{V}_{CO_2\text{excess}}$ was significantly higher during 1st recovery than during 2nd recovery. $\dot{V}_{CO_2\text{excess}}$ was added from rest to the end of 1st recovery and from the start of the 2nd SCS to the end of 2nd recovery (CO_2excess). ΔLa was significantly related to CO_2excess ($r=0.845$). However, ventilation during 1st recovery was the same as that during 2nd recovery. End-tidal CO₂ pressure (PETco_2) significantly decreased from the resting level during the recovery periods, indicating hyperventilation. PETco_2 during 1st recovery was significantly higher than that during 2nd recovery. It is concluded that $\dot{V}_{CO_2\text{excess}}$ is not simply determined by ventilation during recovery from repeated cycle sprints.

Key words: blood lactate, ventilation, excessive CO₂ output, recovery period, cycling sprint.

Introduction

The following findings indicate that excessive CO₂ output ($\dot{V}_{CO_2\text{excess}}$) might be attributable to hyperventilation. Firstly, volitional hyperventilation causes excessive CO₂ expiration (Jones and Jurkowski, 1979). Volitional hyperventilation decreases arterial CO₂ pressure (Paco₂) and consequently increases arterial-venous CO₂ pressure difference. This increase results in excessive removal of CO₂ from tissues. At the same time, since arterial-venous CO₂ difference is increased at the lung level, CO₂ is excessively expired. Secondly, hyperventilation starts when $\dot{V}_{CO_2\text{excess}}$ occurs above the ventilatory threshold (VT) in incremental exercise (Wasserman et al., 1973; Beaver et al., 1986b). During incremental exercise, blood lactate is progressively increased above the VT. This is buffered by the bicarbonate system. This results in progressive reduction of blood bicarbonate ion (Beaver et al., 1986a) and metabolic acidosis. In order to improve this metabolic acidosis, ventilation is driven and becomes hyperventilation above the VT in incremental exercise. As a result, $\dot{V}_{CO_2\text{excess}}$ is progressively increased above the VT.

A short-term cycling sprint with maximal effort results in an increase in blood lactate during recovery. When a cycling sprint is repeated with intervals (interval being a recovery period for the body), blood lactate is summed from the preceding recovery period to the following recovery period (Gaitanos et al., 1993; Matsuura et al., 2006, 2007). Therefore, metabolic acidosis during preceding recovery can become greater than that during following recovery. This greater metabolic acidosis during following recovery may result in greater ventilation and consequently greater $\dot{V}_{CO_2\text{excess}}$ as it does in incremental exercise.

On the other hand, some studies have shown a direct relationship between an increase in blood lactate (ΔLa) and CO₂excess (sum of $\dot{V}_{CO_2\text{excess}}$ during exercise or during exercise and recovery) during exercise (Yano, 1987; Hirakoba et al., 1993; Yano, 1998; Yano et al., 2002) and recovery (Yunoki et al., 1999; Yunoki et al., 2003). When ΔLa is the changed value per min, CO₂excess is equivalent to $\dot{V}_{CO_2\text{excess}}$. Therefore, it has been shown in these studies that ΔLa per min is associated with $\dot{V}_{CO_2\text{excess}}$. However, it is generally likely that hyperventilation is attributable to $\dot{V}_{CO_2\text{excess}}$, especially during incremental exercise. Also, Yunoki et al. (1999) have confirmed from experimental results during and after short intensive exercise that the time course of $\dot{V}_{CO_2\text{excess}}$ is affected by hyperventilation.

The purpose of the present study was, therefore, to examine whether $\dot{V}_{CO_2\text{excess}}$ is dominantly attributable to hyperventilation during the period of recovery from repeated cycling sprints.

Methods

Subjects

Eight healthy male undergraduate students participated in this study. The subjects' mean age, height and body weight were 20.8 ± 2.1 (SD) yr, 173.4 ± 10.0 cm and 66.0 ± 9.2 kg, respectively. They were participating in regular training programs. Each subject signed a statement of informed consent following a full explanation regarding the nature of the experiment. The Ethics Committee of Hokkaido University Graduate School of Education approved the present study.

Design

Each subject attended our laboratory for one test. The subjects' body characteristics were measured and each subject performed four cycling sprints of the experimental protocol described below to become familiarized with repeated cycling sprints with maximal effort as a training trial. Body weight (BW) was used to determine the loads of cycling sprint. Each subject was instructed to refrain from intense physical exercise, drinking, and taking caffeine for 24 hours prior to each visit. None of the subjects had a smoking habit.

Experimental protocol

Experimental instruments were fitted to each subject 1 hour before the test. Then, After resting for 3 min on the bicycle seat, four 10-sec cycling sprints with 30-sec passive recovery periods were performed two times. The 1st and 2nd series of cycling sprints (SCS) were followed by 360-sec passive recovery periods (1st recovery and 2nd recovery). All cycling sprints were performed with a load (F) [N] of $0.075 \cdot BW \cdot 9.81^{-1}$ (Ayalon et al., 1974) from a standing start. Subjects were instructed to pedal as many revolutions as possible during cycling sprints.

Measurements and determinations

All exercise tests were carried out on a bicycle ergometer (POWERMAX-VII, Combi, Tokyo, Japan). The duration and load were adjusted by a built-in computer. The computer also calculated peak rpm (Rpm_{peak}) in a given exercise and displayed the results. Time series behavior in rpm during each cycling sprint was recorded by an online computer at a rate of 10 Hz. Peak power output (PPO) during each cycling sprint was calculated by the following equation:

$$PPO [\text{watt}] = Rpm_{peak} \cdot 6 \cdot F \cdot 0.624^{-1},$$

where 6 is the distance calculated by the built-in computer as the flywheel went into a 360-degree roll [m], and 0.624 is the value for transforming Nm units to watt units [$Nm \cdot min^{-1} \cdot watt^{-1}$]. Mean power output (MPO) for 10-sec was calculated from the above

equation using the data of average Rmp.

Blood samples (25 μL) were collected from fingertips using capillary tubes. The samples were analyzed using a lactate analyzer (YSI-1500 sport, YSI, Tokyo, Japan) to measure blood lactate concentration (La). The lactate analyzer was calibrated by a standard lactate solution of 5 $\text{mmol}\cdot\text{L}^{-1}$ before each test. Samples were taken at 5.5 min during 1st recovery and 2nd recovery.

Oxygen uptake ($\dot{V}\text{O}_2$), carbon dioxide output ($\dot{V}\text{CO}_2$) and end-tidal CO_2 pressure (PET CO_2) were obtained breath-by-breath using a respiratory gas analyzer (AE-280S, Minato Medical Science, Osaka, Japan). Ventilation (VE) was measured by a hot-wire flow meter, and the flow meter was calibrated with a syringe of known volume (2.0 L). O_2 and CO_2 concentrations were measured by a zirconium sensor and infrared absorption analyzer, respectively. The gas analyzer was calibrated by known standard gas (O_2 : 15.17%, CO_2 : 4.92%). $\dot{V}\text{O}_2$, $\dot{V}\text{CO}_2$, VE and PET CO_2 were measured continuously during rest, exercise, and recovery periods. For each 10-sec interval, the averages of $\dot{V}\text{O}_2$, $\dot{V}\text{CO}_2$, VE and PET CO_2 were calculated.

CO_2 excess was defined as total of $\dot{V}\text{CO}_2$ excess from the start of the 1st SCS to the end of 1st recovery and from start of the 2nd SCS to the end of 2nd recovery. $\dot{V}\text{CO}_2$ excess is obtained by the difference between $\dot{V}\text{CO}_2$ and $\dot{V}\text{O}_2$ (Yunoki et al., 1999).

Statistical analysis

Results are presented as means \pm standard deviations (SD). Pearson's correlation coefficient was used to express the strength of the relationship between $\dot{V}\text{CO}_2$ and VE. One-way ANOVA for repeated measures was used to examine the time effect. If F ratios were significant, the Tukey-Kramer post-hoc test was used for the comparison. Two-way ANOVA for repeated measurements was used for comparison between 1st and 2nd recovery periods. If a significant interaction was indicated, the paired t-test was used to examine differences between two recovery conditions and time effects. A value of $P < 0.05$ was regarded as statistically significant.

Results

PPO significantly decreased from the 1st cycling sprint (746 ± 119 watts) to the fourth cycling sprint (652 ± 94 watts) in the 1st SCS. PPO in the 1st cycling sprint in the 2nd SCS (747 ± 120 watts) returned to the 1st cycling sprint level in the 1st series. Then PPO significantly decreased (632 ± 113 watts) as it did in the 1st SCS. MPO significantly decreased from the 1st cycling sprint (587 ± 109 watts) to the fourth cycling sprint (495 ± 82 watts) in the 1st SCS. MPO in the 1st cycling sprint in the 2nd

SCS (573 ± 87 watts) returned to the 1st cycling sprint level in the 1st series. Then MPO significantly decreased (477 ± 86 watts) as it did in the 1st SCS. That is, work load was the same level in both series.

Figure 1 shows $\dot{V}O_2$ and $\dot{V}CO_2$ (upper panel) during the test and $\dot{V}CO_{2\text{excess}}$ during the two recovery periods (lower panel). $\dot{V}CO_2$ was significantly higher than $\dot{V}O_2$. This difference during 1st recovery reached almost zero level immediately before the 2nd SCS. $\dot{V}CO_2$ in 1st recovery was significantly higher than that in 2nd recovery for the first two minutes. $\dot{V}O_2$ kinetics during 1st recovery was the same as that during 2nd recovery. $\dot{V}CO_{2\text{excess}}$ during 1st recovery was significantly higher than that during 2nd recovery.

As shown in Figure 2, $\dot{V}E$ during 1st recovery was the same as that during 2nd recovery. $\dot{V}E$ rapidly decreased for the first 2-3 min and its rate of decrease became slow. Figure 3 shows $PETCO_2$ during the test. $PETCO_2$ temporarily increased after the 1st SCS and significantly decreased from 7.8 min to 12 min (1.8-6 min during the 1st recovery period) and from 13.3 min until the end of 2nd recovery. $PETCO_2$ in 1st recovery was significantly higher than that in 2nd recovery.

La was 0.89 ± 0.17 mM at rest. La was determined at 5.5 min during 1st recovery and 2nd recovery. La during 1st recovery (12.1 ± 2.60 mM) was significantly lower than that during 2nd recovery (14.1 ± 2.43 mM). Increase in La (ΔLa) from rest to 1st recovery (11.17 ± 2.57 mM) was significantly greater than that from the start of the 2nd SCS to 2nd recovery (2.07 ± 1.23 mM). $PETCO_2$ at the time point of La determination during 1st recovery (31.8 ± 3.09 Torr) was significantly higher than that at the time point of La determination during 2nd recovery (29.6 ± 2.26 Torr). The higher La became during 2nd recovery, the lower $PETCO_2$ became during 2nd recovery.

Figure 4 shows the relationship between $CO_{2\text{excess}}$ and changed values in blood lactate (ΔLa) from rest to 5.5 min during 1st recovery and from the start of the 2nd cycling sprints to 5.5 min during 2nd recovery. There was a significant correlation between $CO_{2\text{excess}}$ and ΔLa ($r=0.845$). $CO_{2\text{excess}}$ from the start of the 1st SCS to the end of 1st recovery (4.46 ± 0.92 l) was significantly higher than that from the start of 2nd SCS to the end of the 2nd recovery (1.74 ± 0.50 l).

Discussion

Relationship between blood lactate and $\dot{V}E$

Ventilation during 1st recovery was the same as that during 2nd recovery despite the difference in La . This is a new finding. In the present study, pH was not measured. However, La level might strongly affect blood pH level because it is known that pH is

decreased in proportion to an increase in lactate level in the blood after maximal exercise of short duration (Osnes and Hermensen, 1971).

The following findings suggest that hyperventilation in exercise is induced by metabolic acidosis due to an increase in blood lactate detected by peripheral chemoreceptors. Firstly, in subjects who had had both carotid bodies surgically resected, ventilation was the same at a steady state below the VT but less above the VT than that in the normal group (Wasserman et al., 1975). This suggests that metabolic acidosis detected by carotid bodies works for hyperventilation. Secondly, it was found that intravenous infusion of bicarbonate during incremental exercise attenuated the decrease in blood pH above the VT and consequently reduced hyperventilation by 15-30% (Peronnet et al., 2007). However, if this hyperventilation accompanies a decrease in P_{aCO_2} , it would stimulate central chemoreceptors and peripheral receptors via its effect on pH (Clement et al., 1992) and consequently can attenuate the hyperventilation.

We assume in this discussion that ventilation consists of hyperventilation and non-hyperventilation components and that the non-hyperventilation component shows the same kinetics during two recovery periods and inevitably is controlled by factors other than blood lactate and P_{aCO_2} . Clement et al. (1996) suggested that ventilation 30 min after heavy exercise remains stimulated by a process other than post-exercise metabolic acidosis in man. Since ventilation during recovery from exercise below VT gradually decreases while pH and P_{aCO_2} are the resting levels (Stringer et al., 1992), ventilation should be driven by other than humoral factors. Indeed, a study using positron emission tomography in human subjects suggested that motor cortex plays a role in ventilatory control during and after exercise in the humoral phases (Fink et al. 1995).

Thus, hyperventilation during 2nd recovery did not increase despite an increase in blood lactate probably due to lower P_{aCO_2} than that during 1st recovery.

Relationship between blood lactate and V_{CO_2} excess

During recovery, lactate is not produced in muscle. However, lactate is transported from the muscle to blood. The buffering system is primarily a non-bicarbonate system in muscle cells (Hultman and Shalin, 1980) but a bicarbonate system in blood (Yano, 1987; Peronnet and Aguilaniu, 2006). Therefore, transportation of lactate to blood makes it possible to reduce bicarbonate ion without production of lactic acid in the body. As a result, the reduced bicarbonate becomes V_{CO_2} excess by hyperventilation (Yunoki et al. 1999). After the end of heavy, very heavy and cycling sprint, P_{aCO_2} becomes lower than the resting level (Kowalchuk et al., 1988; Stringer et

al., 1992). Therefore, this $\dot{V}_{CO_2\text{excess}}$ during recovery includes respiratory compensation (Yunoki et al., 2003). However, the results of these studies have not provided a sufficient explanation for $\dot{V}_{CO_2\text{excess}}$ during recovery.

A model in which $\dot{V}_{CO_2\text{excess}}$ is derived from the downward shift of the CO_2 dissociation curve due to lactate increase has been proposed on the basis of experimental data obtained in incremental exercise (Figure 5: Yano, 1997). At the active muscle level, lactate is transported from muscle tissue to blood. An increase in blood lactate (ΔLa) can cause a downward shift in the oxygenated CO_2 dissociation curve (Miyamura and Honda, 1978). Mixed venous CO_2 pressure (P_{vCO_2}) determines venous CO_2 content with the shifted CO_2 dissociation curve. Arterial CO_2 content is determined by both P_{aCO_2} and the CO_2 dissociation curve before the shift. At the lung level, there is no shift in the CO_2 dissociation curve since there is no ΔLa . CO_2 content in venous blood is eliminated by pulmonary ventilation. P_{aCO_2} is determined by the ventilation. Since there is no shift in the CO_2 dissociation curve at the lung level, venous-arterial CO_2 difference at the lung level is increased more than that at the muscle level by the shifted value and decrease in P_{aCO_2} (ΔP_{aCO_2}). This difference is associated with $\dot{V}_{CO_2\text{excess}}$ due to ΔLa and ΔP_{aCO_2} . (Even if the effect of oxygenation on the CO_2 dissociation curve (Christensen-Douglas-Holden effect) is taken into consideration, this model is valid.)

Lactate in femoral venous blood increases until 4-5 min of recovery after short intensive exercise and then slightly decreases from 4-5 min of recovery (Kowalchuk et al., 1988). Therefore, the shift in the CO_2 dissociation curve should occur during the early period of recovery. During this phase, this shift should help CO_2 elimination from blood to the lungs and the eliminated CO_2 should be expired from the lungs to air by ventilation. If P_{aCO_2} is decreased by ventilation, the expired CO_2 will include $\dot{V}_{CO_2\text{excess}}$ due to hyperventilation as volitional hyperventilation. Thus, it is likely that the shift in the CO_2 dissociation curve functions as facilitation for CO_2 expiration by ventilation.

Since $\dot{V}_{CO_2\text{excess}}$ reached almost zero at the end of the 1st recovery period in the present study, ΔLa around this end point is judged to be almost zero. In this stage, the 2nd SCS was started. Therefore, La produced in the 2nd SCS should be added to the La level at 1st recovery. However, the La level during 2nd recovery did not become twice the blood lactate level at 1st recovery. This smaller ΔLa can reduce the degree of shift in the CO_2 dissociation curve, resulting in less $\dot{V}_{CO_2\text{excess}}$ during 2nd recovery.

Conclusion

Ventilation during the two recovery periods was similar despite different levels of blood lactate. This is probably due to the difference in P_{aCO_2} . $\dot{V}_{CO_2\text{excess}}$ during the 2nd recovery period was lower than that during the 1st recovery period despite the fact that there was no change in ventilation. An increase in blood lactate was directly related with $CO_2\text{excess}$ than ventilation. It is therefore concluded that $\dot{V}_{CO_2\text{excess}}$ is not simply determined by ventilation during recovery from repeated cycle sprints.

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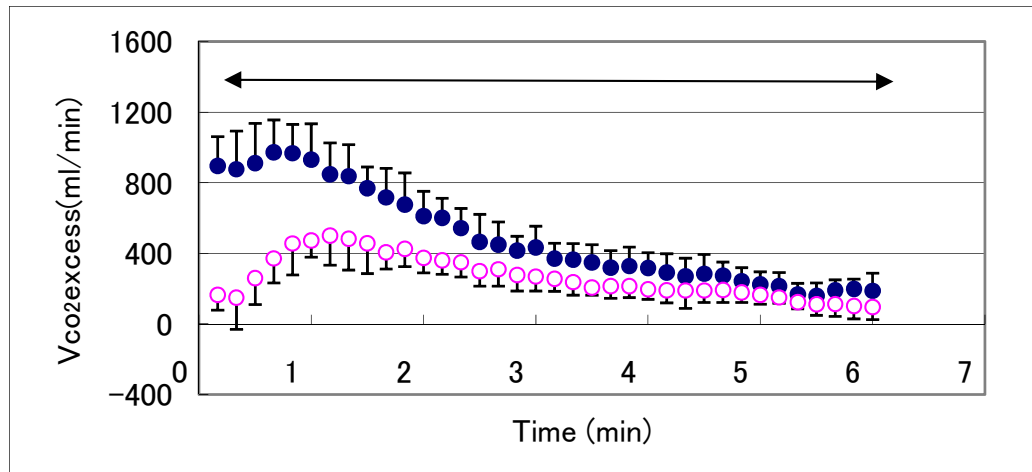
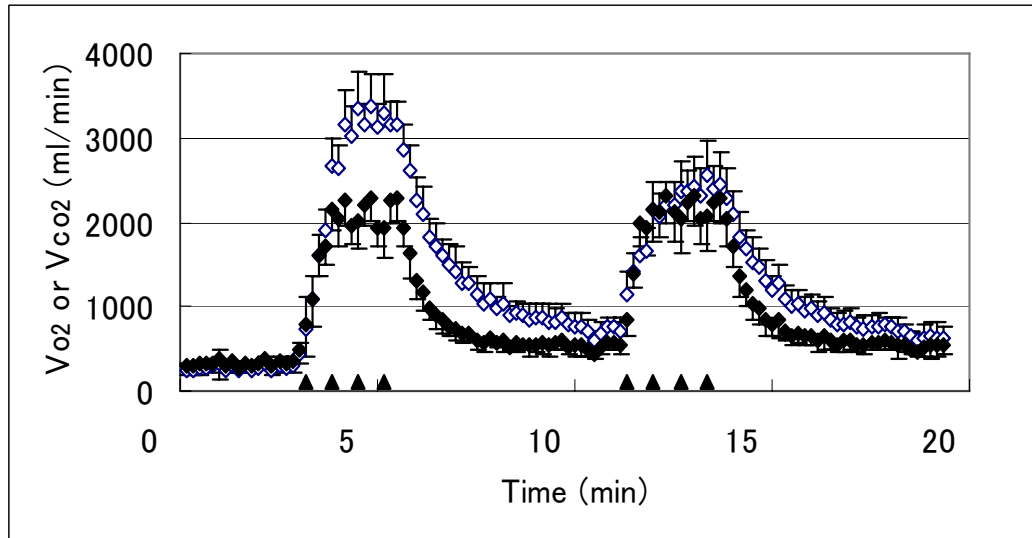


Fig. 1. O_2 uptake (\blacklozenge) and CO_2 output (\blacklozenge) in repeated cycling sprints (upper panel). A series of four cycling sprints was performed two times (\blacktriangle). Excessive CO_2 output ($V_{CO_2\text{excess}}$) after 1st recovery (\bullet) and 2nd recovery (\circ) (lower panel). Arrow shows significant difference between 1st recovery and 2nd recovery.

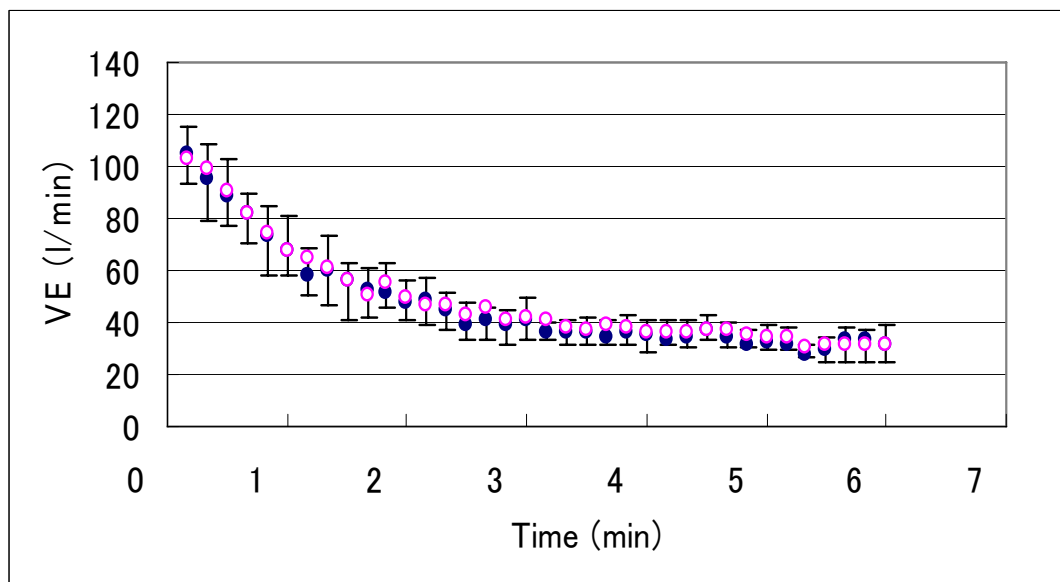
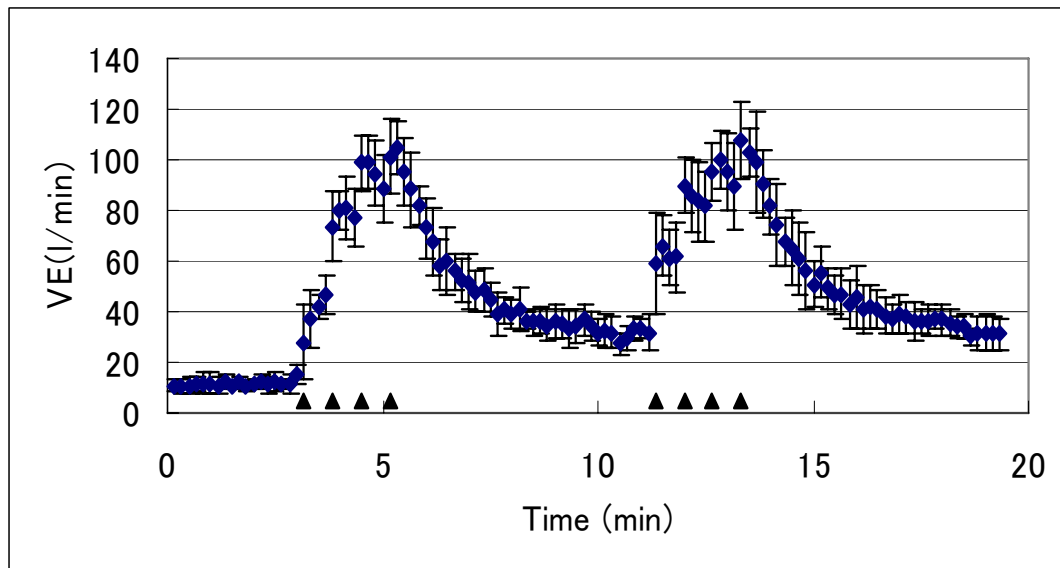


Fig. 2. Ventilation (◆) in repeated cycling sprints (upper panel). A series of four cycling sprints was performed two times (▲). Ventilation after 1st recovery (●) and 2nd recovery (○) (lower panel).

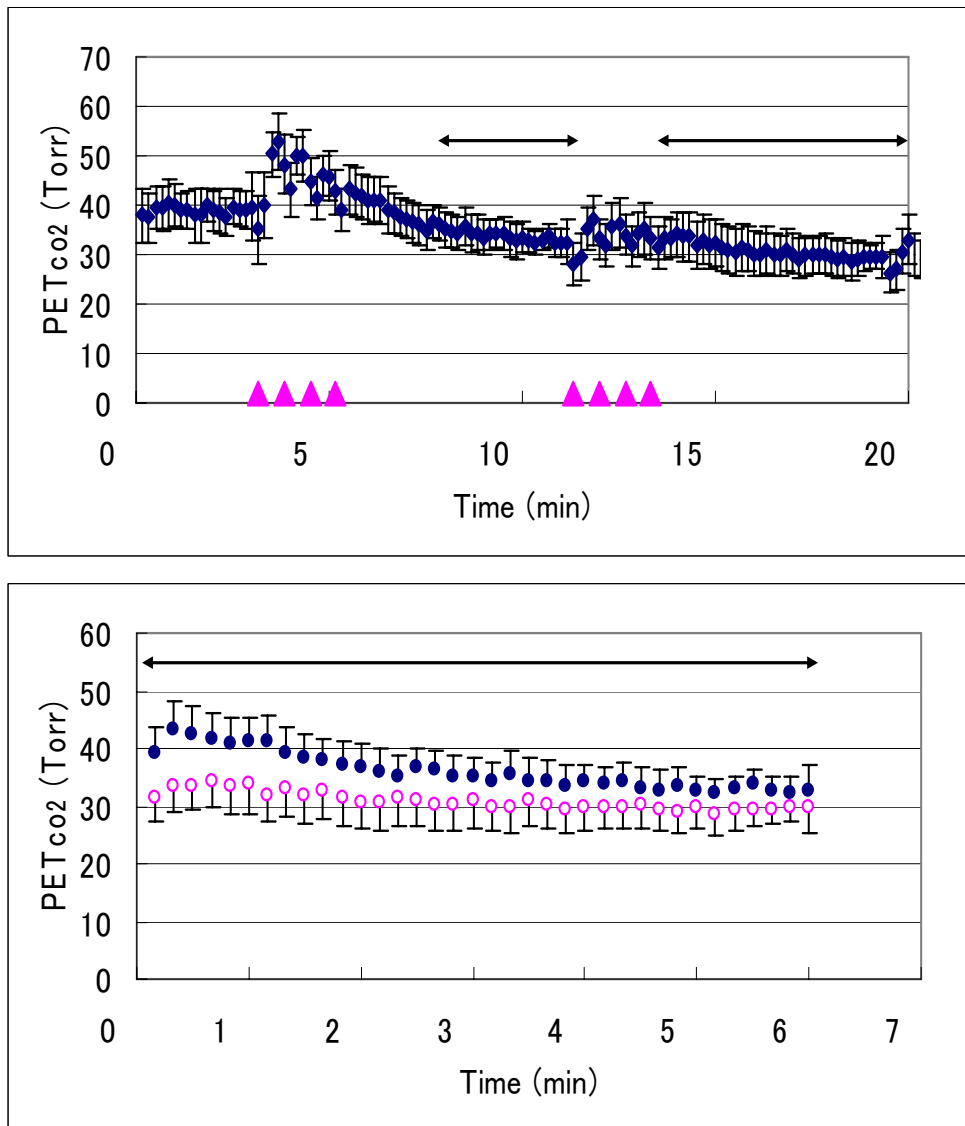


Fig. 3. End-tidal CO₂ pressure (PET_{co2}) (◆) in repeated cycling sprints (upper panel). A series of four cycling sprints was performed two times (▲). Arrows show significant difference between PET_{co2} at rest and after cycling sprints (upper panel) and significant difference between 1st recovery and 2nd recovery (lower panel). End-tidal CO₂ pressure after 1st recovery (●) and 2nd recovery (○) (lower panel). Arrow shows significant difference between 1st recovery and 2nd recovery.

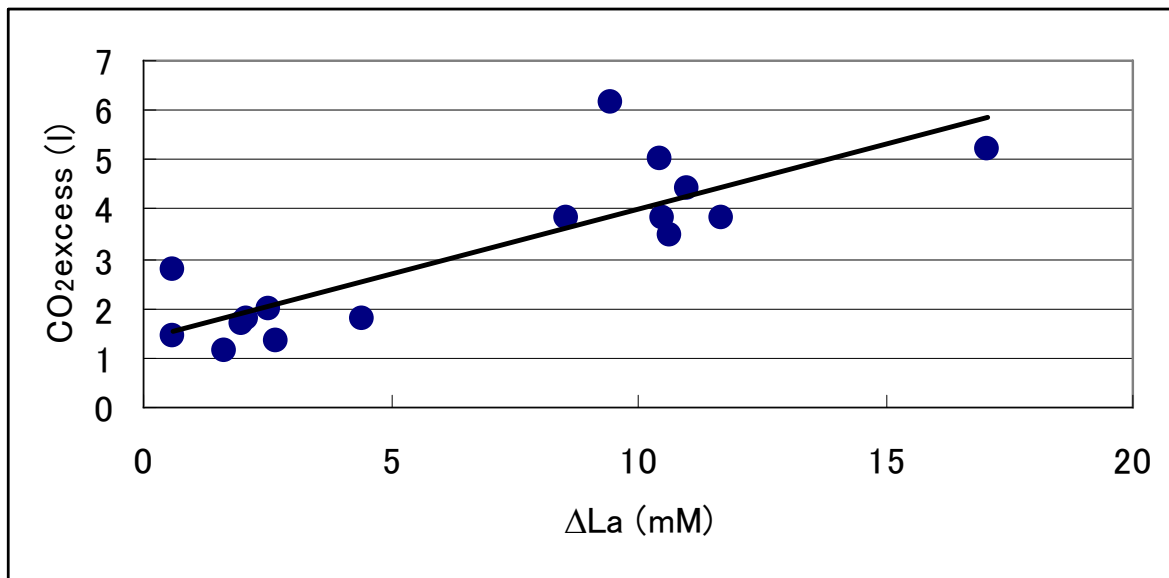


Fig. 4. Relationship between changed value in blood lactate concentration (ΔLA) and CO_2 excess.

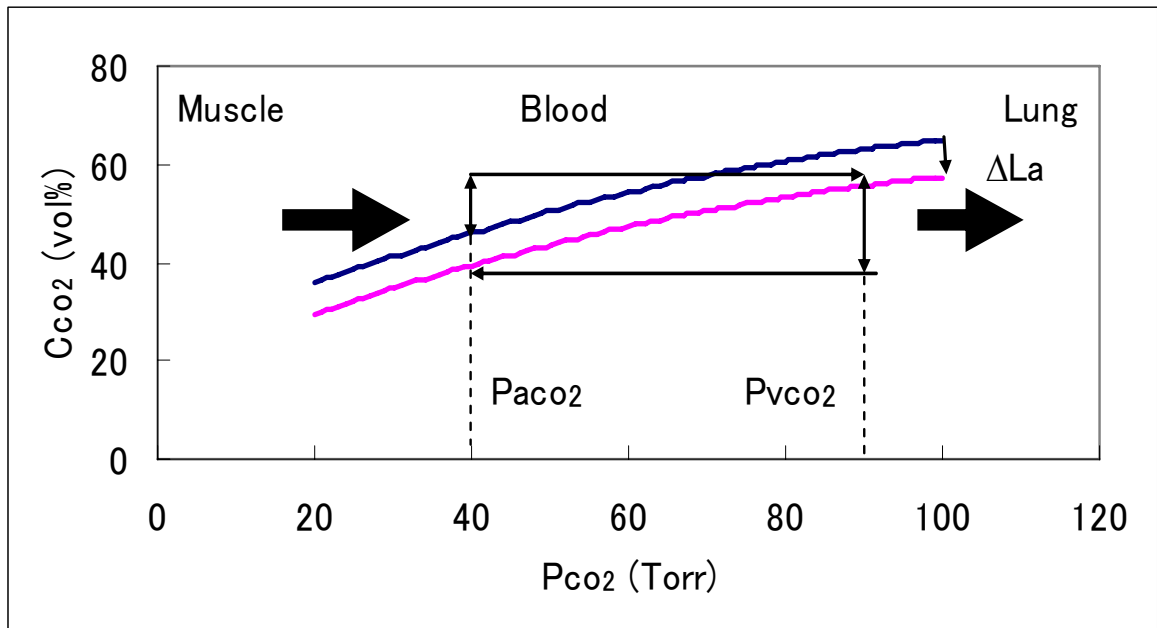


Fig. 5. Model of excessive CO₂ output ($\dot{V}_{CO_2\text{excess}}$). CO₂ dissociation curve is shifted downward due to lactate increase at the muscle level but is unchanged at the lung level due to no change in lactate. This shift in the CO₂ dissociation curve causes the difference in arterial-venous CO₂ content at the lung level and muscle level. If arterial CO₂ pressure (P_{ACO₂}) is decreased by ventilation, then $\dot{V}_{CO_2\text{excess}}$ due to ventilation is added. P_{VCO₂}: mixed venous CO₂ pressure