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Relationship between Hyperventilation and Excessive CO_2 Output during Recovery from Repeated Cycling Sprints

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Summary

The purpose of the present study was to examine whether excessive CO_2 output ($\dot{V}co_2$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 first series and second series of cycle sprints (SCS) were followed by 360-sec passive recovery periods (first recovery and second recovery). Increases in blood lactate (ΔLa) were 11.17±2.57 mM from rest to 5.5 min during first recovery and 2.07±1.23 mM from the start of the second SCS to 5.5 min during second recovery. CO_2 output ($\dot{V}co_2$) was significantly higher than O_2 uptake ($\dot{V}o_2$) during both recovery periods. This difference was defined as $\dot{V}co_2$excess. $\dot{V}co_2$excess was significantly higher during first recovery than during second recovery. $\dot{V}co_2$excess was added from rest to the end of first recovery and from the start of the second SCS to the end of second recovery (CO_2excess). ΔLa was significantly related to CO_2excess (r=0.845). However, ventilation during first recovery was the same as that during second recovery. End-tidal CO_2 pressure ($PETco_2$) significantly decreased from the resting level during the recovery periods, indicating hyperventilation. $PETco_2$ during first recovery was significantly higher than that during second recovery. It is concluded that $\dot{V}co_2$excess is not simply determined by ventilation during recovery from repeated cycle sprints.

Key words

Blood lactate • Ventilation • Excessive CO_2 output • Recovery period • Cycling sprint

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Introduction

The following findings indicate that excessive CO_2 output ($\dot{V}co_2$excess) might be attributable to hyperventilation. Firstly, volitional hyperventilation causes excessive CO_2 expiration (Jones and Jurkowski, 1979). Volitional hyperventilation decreases arterial CO_2 pressure ($Paco_2$) and consequently increases arterial-venous CO_2 pressure difference. This increase results in excessive removal of CO_2 from tissues. At the same time, since arterial-venous CO_2 difference is increased at the lung level, CO_2 is excessively expired. Secondly, hyperventilation starts when $\dot{V}co_2$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, $\dot{V}co_2$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

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in greater ventilation and consequently greater $\dot{V}co_2$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 CO_2excess (sum of $\dot{V}co_2$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, CO_2excess is equivalent to $\dot{V}co_2$excess. Therefore, it has been shown in these studies that ΔLa per min is associated with $\dot{V}co_2$excess. However, it is generally likely that hyperventilation is attributable to $\dot{V}co_2$excess, especially during incremental exercise. Yunoki *et al.* (1999) have confirmed from experimental results during and after short intensive exercise that the time course of $\dot{V}co_2$excess is affected by hyperventilation.

The purpose of the present study was, therefore, to examine whether $\dot{V}co_2$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) years, 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 h 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 first and second series of cycling sprints (SCS) were followed by 360-sec passive recovery periods (first recovery and second 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-V_{II}, 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:

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

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 mmol/l before each test. Samples were taken at 5.5 min during first recovery and second recovery.

Oxygen uptake ($\dot{V}o_2$), carbon dioxide output ($\dot{V}co_2$) and end-tidal CO_2 pressure ($PETco_2$) were obtained breath-by-breath using a respiratory gas analyzer (AE-280S, Minato Medical Science, Osaka, Japan). Ventilation ($\dot{V}E$) was measured by a hot-wire flow meter, and the flow meter was calibrated with a syringe of known volume (2 liters). 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 %). $\dot{V}o_2$, $\dot{V}co_2$, $\dot{V}E$ and $PETco_2$ were measured continuously during rest, exercise, and recovery periods. For each 10-sec interval, the averages of $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$ and $PETCO_2$ were calculated.

CO_2excess was defined as total of $\dot{V}CO_2$excess from the start of the first SCS to the end of first recovery and from start of the second SCS to the end of second recovery. $\dot{V}CO_2$excess is obtained by the difference between $\dot{V}CO_2$ and $\dot{V}O_2$ (Yunoki *et al.* 1999).

Statistical analysis

Results are presented as means ± standard deviations (SD). Pearson's correlation coefficient was used to express the strength of the relationship between $\dot{V}CO_2$ and $\dot{V}E$. 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 first and second 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 first cycling sprint (746±119 watts) to the fourth cycling sprint (652±94 watts) in the first SCS. PPO in the first cycling sprint in the second SCS (747±120 watts) returned to the first cycling sprint level in the first series. Then PPO significantly decreased (632±113 watts) as it did in the first SCS. MPO significantly decreased from the first cycling sprint (587±109 watts) to the fourth cycling sprint (495±82 watts) in the first SCS. MPO in the first cycling sprint in the second SCS (573±87 watts) returned to the first cycling sprint level in the first series. Then MPO significantly decreased (477±86 watts) as it did in the first 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 $\dot{V}CO_2$excess during the two recovery periods (lower panel). $\dot{V}CO_2$ was significantly higher than $\dot{V}O_2$. This difference during first recovery reached almost zero level immediately before the second SCS. $\dot{V}CO_2$ in first recovery was significantly higher than that in second recovery for the first two minutes. $\dot{V}O_2$ kinetics during first recovery was the same at that during second recovery. $\dot{V}CO_2$excess during first recovery was

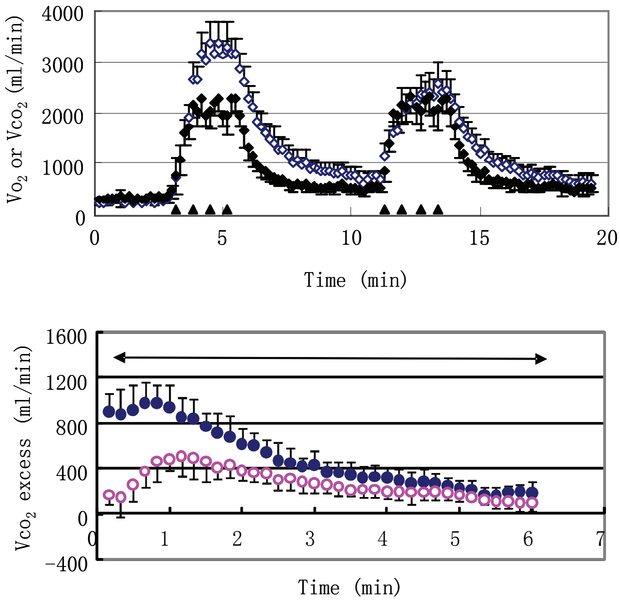

Fig. 1. O_2 uptake (◆) and CO_2 output (◇) in repeated cycling sprints (upper panel). A series of four cycling sprints was performed two times (▲). Excessive CO_2 output ($\dot{V}CO_2$excess) after first recovery (●) and second recovery (○) (lower panel). Arrow shows significant difference between first recovery and second recovery.

significantly higher than that during second recovery.

As shown in Figure 2, $\dot{V}E$ during first recovery was the same as that during second 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 first SCS and significantly decreased from 7.8 min to 12 min (1.8-6 min during the first recovery period) and from 13.3 min until the end of second recovery. $PETCO_2$ in first recovery was significantly higher than that in second recovery.

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

Figure 4 shows the relationship between CO_2excess and changed values in blood lactate (ΔLa) from rest to 5.5 min during first recovery and from the

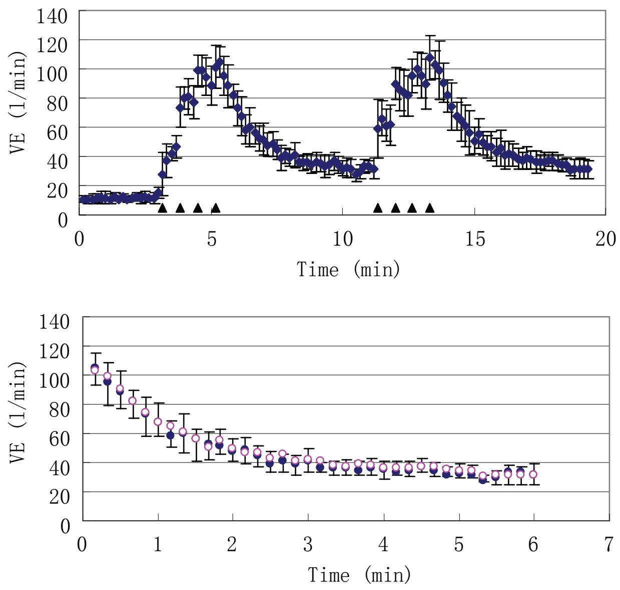

Fig. 2. Ventilation (◆) in repeated cycling sprints (upper panel). A series of four cycling sprints was performed two times (▲). Ventilation after first recovery (●) and second recovery (○) (lower panel).

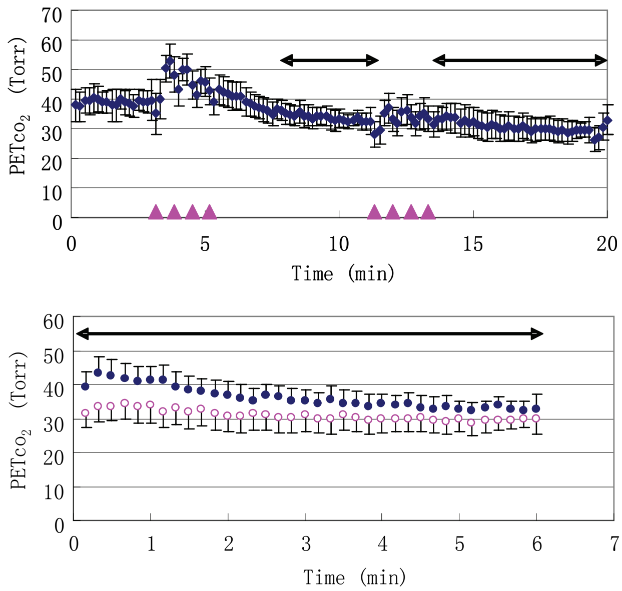

Fig. 3. End-tidal CO_2 pressure ($PETco_2$) (◆) in repeated cycling sprints (upper panel). A series of four cycling sprints was performed two times (▲). Arrows show significant difference between $PETco_2$ at rest and after cycling sprints (upper panel) and significant difference between first recovery and second recovery (lower panel). End-tidal CO_2 pressure after first recovery (●) and second recovery (○) (lower panel). Arrow shows significant difference between first recovery and second recovery.

start of the second cycling sprints to 5.5 min during second recovery. There was a significant correlation between CO_2excess and ΔLa (r = 0.845). CO_2excess from the start of the first SCS to the end of first recovery (4.46±0.92 l) was significantly higher than that from the start of second SCS to the end of the second recovery (1.74±0.50 l).

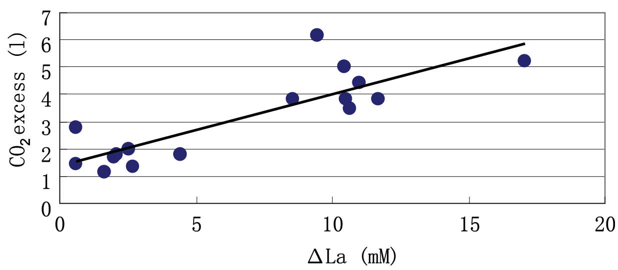

Fig. 4. Relationship between changed value in blood lactate concentration (ΔLA) and CO_2 excess.

Discussion

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

Ventilation during first recovery was the same as that during second 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 $Paco_2$, 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 is inevitably controlled by factors other than blood lactate and $Paco_2$. 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 $Paco_2$ are at 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 venilatory control during and after exercise in the humoral phases (Fink *et al.* 1995).

Thus, hyperventilation during second recovery did not increase despite an increase in blood lactate probably due to lower $Paco_2$ than that during first recovery.

Relationship between blood lactate and $\dot{V}co_2$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 $\dot{V}co_2$excess by hyperventilation (Yunoki *et al.* 1999). After the end of heavy, very heavy and cycling sprint, $Paco_2$ becomes lower than the resting level (Kowalchuk *et al.* 1988, Stringer *et al.* 1992). Therefore, this $\dot{V}co_2$excess during recovery includes respiratory compensation (Yunoki *et al.* 2003). However, the results of these studies have not provided a sufficient explanation for $\dot{V}co_2$excess during recovery.

A model in which $\dot{V}co_2$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 (Fig. 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 ($Pvco_2$) determines venous CO_2 content with the shifted CO_2 dissociation curve. Arterial CO_2 content is determined by both $Paco_2$ 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. $Paco_2$ 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 $Paco_2$ ($\Delta Paco_2$). This difference is associated with $\dot{V}co_2$excess due to ΔLa and $\Delta Paco_2$. Even if the effect of oxygenation on the CO_2 dissociation curve (Christensen-Douglas-Holden effect) is taken into consideration, this model is valid.

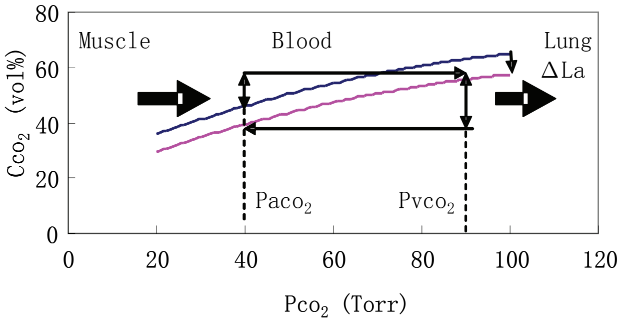

Fig. 5. Model of excessive CO_2 output ($\dot{V}co_2$excess). CO_2 dissociation curve is shifted downward due to lactate increase at the muscle level but is unchanged at the lung level due to no change in lactate. This shift in the CO_2 dissociation curve causes the difference in arterial-venous CO_2 content at the lung level and muscle level. If arterial CO_2 pressure ($Paco_2$) is decreased by ventilation, then $\dot{V}co_2$excess due to ventilation is added. $Pvco_2$: mixed venous CO_2 pressure.

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 $Paco_2$ is decreased by ventilation, the expired CO_2 will include $\dot{V}co_2$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 $\dot{V}co_2$excess reached almost zero at the end of the first recovery period in the present study, ΔLa around this end point is judged to be almost zero. In this stage, the second SCS was started. Therefore, La produced in the second SCS should be added to the La level at first recovery. However, the La level during second recovery did not become twice the blood lactate level at first recovery. This smaller ΔLa can reduce the degree of shift in the CO_2 dissociation curve, resulting in less $\dot{V}co_2$excess during second recovery.

Conclusions

Ventilation during the two recovery periods was similar despite different levels of blood lactate. This is probably due to the difference in $Paco_2$. $\dot{V}co_2$exess during the second recovery period was lower than that during the first recovery period despite the fact that there was no change in ventilation. An increase in blood lactate was directly related with CO_2excess than ventilation. It is therefore concluded that $\dot{V}co_2$excess is not simply determined by ventilation during recovery from repeated cycle sprints.

Conflict of Interest

There is no conflict of interest.

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