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VENTILATION AND BLOOD LACTATE LEVELS AFTER RECOVERY FROM SINGLE AND MULTIPLE SPRINT EXERCISE

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ABSTRACT: The purpose of this study was to examine whether ventilation kinetics are related to blood lactate level after 5 min in recovery after a sprint. Subjects performed two tests, one test consisting of one sprint with maximal effort and the other test consisting of five repeated sprints with time intervals of 6 min. The recovery period from the last sprint was 30 min. Oxygen uptake during recovery from one sprint was the same as that during recovery from five repeated sprints. Ventilation after 50 sec in recovery after one sprint was the same as that in recovery after five repeated sprints despite the significantly higher blood lactate levels during recovery from five repeated sprints than that from a single sprint. There was an exponential relationship between ventilation and blood lactate after 5 min in recovery. The curve shifted to the right in the case of five repeated sprints. End tidal CO_2 pressure after one sprint was higher than that after five repeated sprints during recovery. From these results, it seems that ventilation control related to blood lactate level is modified by end tidal CO_2 pressure.

KEY WORDS: blood lactate, end tidal CO_2 pressure, ventilation, recovery, sprint

INTRODUCTION

Since lactic acid can produced hydrogen ion (H^+) and H^+ can stimulate ventilation, lactic acid is known to be an important factor for control of ventilation ($\dot{V}E$), not only during exercise but also during recovery. An increase in CO_2 pressure in arterial blood ($Paco_2$) can also stimulate $\dot{V}E$ through peripheral chemoreceptors and central chemoreceptors [2]. Exercise-induced metabolic acidosis can stimulate peripheral chemoreceptors as well [11,13-16]. Exercise-induced metabolic acidosis during recovery can induce a lower $Paco_2$ level than the resting value [9,18]. This is known as respiratory compensation. The lower $Paco_2$ may decrease the stimulation of ventilation. Therefore, it is thought that ventilation kinetics are not simply determined by exercise-induced metabolic acidosis. Nevertheless, it has been reported that ventilation increases with an increase in end tidal CO_2 pressure (P_{ETCO_2}) in a rebreathing test but that ventilation is constant below a given level of P_{ETCO_2} [4]. In other words, there is a threshold in the ventilation and P_{ETCO_2} relation. Therefore, the effect of metabolic acidosis on ventilation kinetics may directly appear during recovery in the case of P_{ETCO_2} being lower than the threshold. However, there have been few studies on ventilation control during recovery from exercise, especially from high-intensity exercise for a short duration [2,3,18].

It is known that exercise of a very short duration with maximal effort (sprint) can induce production of lactate [6]. When a sprint is repeated with a given time interval, blood lactate (La) can increase at the end of the repetition more than after one sprint without changing the power output of the sprints [8]. Therefore, if the ventilation kinetics during recovery are affected only by the La level, the difference in La should cause a difference in $\dot{V}E$ kinetics between a sprint and repeated sprints.

However, during the first 5-6 min of recovery from high-intensity exercise, $\dot{V}E$ decreases despite an increase in La [9]. It is also known that arterial potassium, which could affect $\dot{V}E$, increases during exercise and reaches the resting value during recovery at around 5 min [17]. Furthermore, there is the remaining effect of the central nervous system (CNS) in recovery (afterdischarge) [5]. Thus, $\dot{V}E$ control for the first 5-6 min of recovery would be complex due to remaining effects of exercise.

The purpose of the present study was therefore to examine whether $\dot{V}E$ kinetics are related to La level after 5 min in recovery after a sprint.

MATERIALS AND METHODS

Subjects. Six healthy male undergraduate and graduate students participated in the present study. The subjects' mean age, height and body weight were 20.7 ± 1.2 (SD) yr, 178.2 ± 4.1 cm and 68.8 ± 6.8 kg, respectively. 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

Before test days, each subject did a sprint trial to exert maximal effort with a cycle ergometer. Each subject attended our laboratory for two tests. The time interval between two consecutive tests was at least 2 days, and all tests were completed within 3 weeks. Body mass (BM) was used to determine the loads of the cycling sprint. Each subject was instructed to refrain from intense physical exercise, drinking alcohol, and taking caffeine for 24 h prior to each visit.

Experimental protocol

Each subject performed two sprint tests with maximal effort on separate days, one test consisting of one cycling sprint for 10 s (one sprint) and the other test consisting of a 10-s sprint repeated five times with time intervals of 6 min with resting status (five sprints). Each subject came to the laboratory one hour before the start of the test. Immediately after coming to the laboratory, the subjects emptied their bladders and were weighed. Then experimental instruments were attached to the subjects before the experiment.

All sprints were performed with a resistive load [newtons, N] of $0.075 \cdot BM \cdot 9.81^{-1}$ [1]. Subjects were instructed to pedal as many revolutions as possible during cycling sprints. For all tests, subjects were in the seated position during exercise and recovery.

Sprints

All exercise tests were carried out on a cycle ergometer (POWERMAX-VII, Combi, Tokyo, Japan). The duration and resistive load were adjusted by a built-in computer. The computer also calculated peak rpm (rpmpeak) for a given exercise and displayed the results. Time series behaviour in rpm during each cycling sprint was recorded by an online computer at a rate of 10 Hz. Each subject's feet were strapped to the pedals to prevent them from slipping. The seat height was adjusted so that there was a slight bend in the knee joint when the foot pedal was at its lowest position. The results of rpm were averaged over 0.5-s time intervals. Load (F) was $0.075 \cdot BM \cdot 9.81^{-1}$ [1]. Peak power output (PPO: watts (W)) was calculated as follows:

$$\text{Power output} = \text{rpmpeak} \cdot 6 \cdot F \cdot 0.624^{-1},$$

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 W units [$Nm \cdot min^{-1} \cdot W^{-1}$]. The maximum product of rpm and load, during each sprint, is referred to as PPO.

Blood lactate concentration(La): Blood samples (25 μL) were collected from fingertips using capillary tubes and analysed using a lactate analyser (YSI 1500 SPORT, YSI, OH, USA) to measure blood lactate concentration. The lactate analyser was calibrated with a standard lactate solution of 5 mM before each test. Blood was sampled at rest, immediately after one sprint or the last sprint in five sprints, and at 5 min, 10 min, 20 min and 30 min during recovery.

Respiration gas

Data on respiration gas exchange were obtained breath-by-breath using a respiratory gas analyser (AE-280S, Minato Medical Science, Osaka, Japan). Ventilation ($\dot{V}E$) was measured with 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 with a zirconium sensor and infrared absorption analyser, respectively. The gas analyser was calibrated with known standard gas (O_2: 15.17%, CO_2: 4.92%). Respiration gas exchange was measured continuously during rest, exercise, and recovery periods. For each 10-s interval, the averages of $\dot{V}E$, oxygen uptake ($\dot{V}O_2$) and end tidal CO_2 pressure (P_{ETCO_2}) were determined on the basis of breath-by-breath data.

Statistical analysis

Results are presented as means ± standard deviations (SD). A paired t-test was used to examine significant differences between the two tests and between values obtained with different time in the test. A value of $P < 0.05$ was regarded as statistically significant.

RESULTS

Peak power output was 666 ± 85.3 W in one sprint. There was no significant difference between PPO in one sprint and that in five sprints. There were no significant differences between PPOs in the five sprints.

Figure 1 shows $\dot{V}O_2$ kinetics during recovery. Until 30 s, $\dot{V}O_2$ increased and then exponentially decreased. There was no significant difference between $\dot{V}O_2$ during recovery from one sprint and that during recovery from five sprints.

Figure 2 shows La levels at rest and after one sprint and five sprints. La level significantly increased after one sprint, peaked at 5 min after one sprint and five sprints, and then decreased. La level during recovery after five sprints at each time was significantly higher than that during recovery from one sprint.

Figure 3 shows $\dot{V}E$ after one sprint and five sprints. $\dot{V}E$ increased until 50 s and then decreased during recovery from one sprint. $\dot{V}E$ during recovery from five sprints showed a slight decrease until one minute and then rapidly decreased. $\dot{V}E$ after one minute in recovery from one sprint was not significantly different from that after one minute in recovery from five sprints. $\dot{V}E$ until 50s during recovery after one sprint was significantly lower than that after repeated sprints.

Figure 4 shows P_{ETCO_2} after one sprint and five sprints. The peak value of P_{ETCO_2} (52.0 ± 3.9 Torr) appeared at 30 s during recovery from one sprint and was significantly higher than P_{ETCO_2}

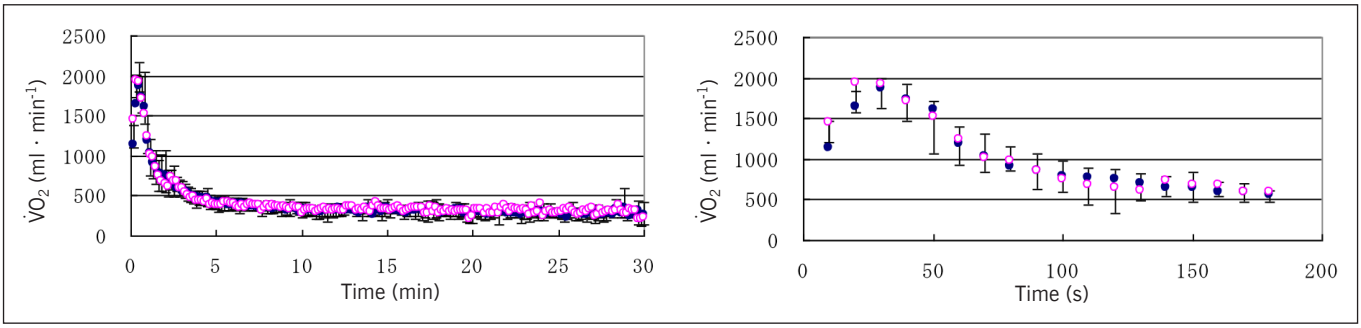

FIG. 1. LEFT PANEL SHOWS OXYGEN UPTAKE ($\dot{V}O_2$) DURING RECOVERY FROM ONE SPRINT (CLOSED CIRCLES) AND DURING RECOVERY FROM FIVE REPEATED SPRINTS (OPEN CIRCLES). RIGHT PANEL SHOWS OXYGEN UPTAKE FOR THE FIRST 3 MIN OF RECOVERY FROM ONE SPRINT (CLOSED CIRCLES) AND THAT FOR THE FIRST 3 MIN OF RECOVERY FROM FIVE REPEATED SPRINTS (OPEN CIRCLES).

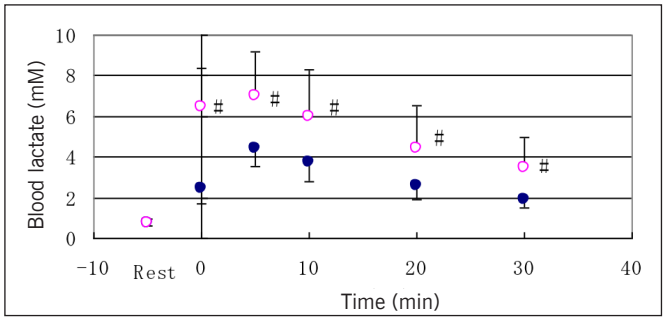

FIG. 2. BLOOD LACTATE LEVELS AT REST AND DURING RECOVERY FROM ONE SPRINT (CLOSED CIRCLES) AND FIVE REPEATED SPRINTS (OPEN CIRCLES).
Note: # Significant difference between blood lactate level during recovery from one sprint and that during recovery from five repeated sprints.

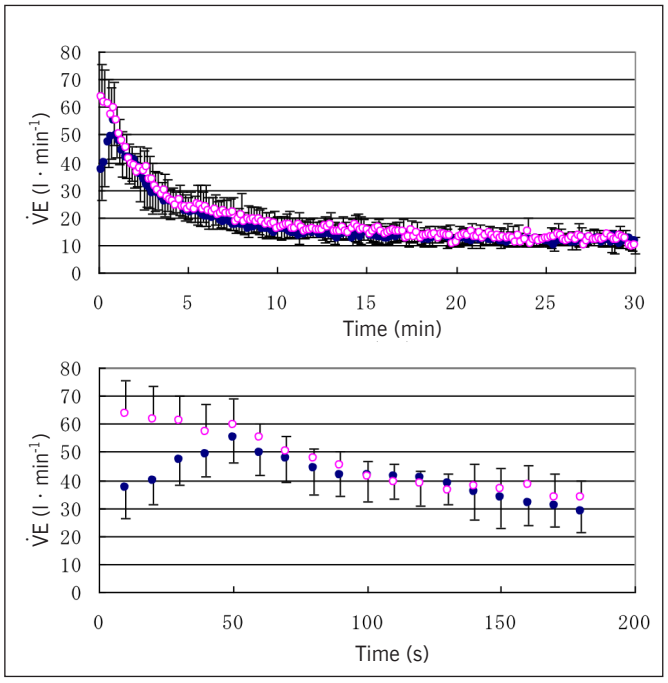

FIG. 3. UPPER PANEL SHOWS VENTILATION DURING RECOVERY FROM ONE SPRINT (CLOSED CIRCLES) AND DURING RECOVERY FROM FIVE REPEATED SPRINTS (OPEN CIRCLES). LOWER PANEL SHOWS VENTILATION FOR THE FIRST 3 MIN OF RECOVERY FROM ONE SPRINT (CLOSED CIRCLES) AND THAT FOR THE FIRST 3 MIN OF RECOVERY FROM FIVE REPEATED SPRINTS (OPEN CIRCLES).

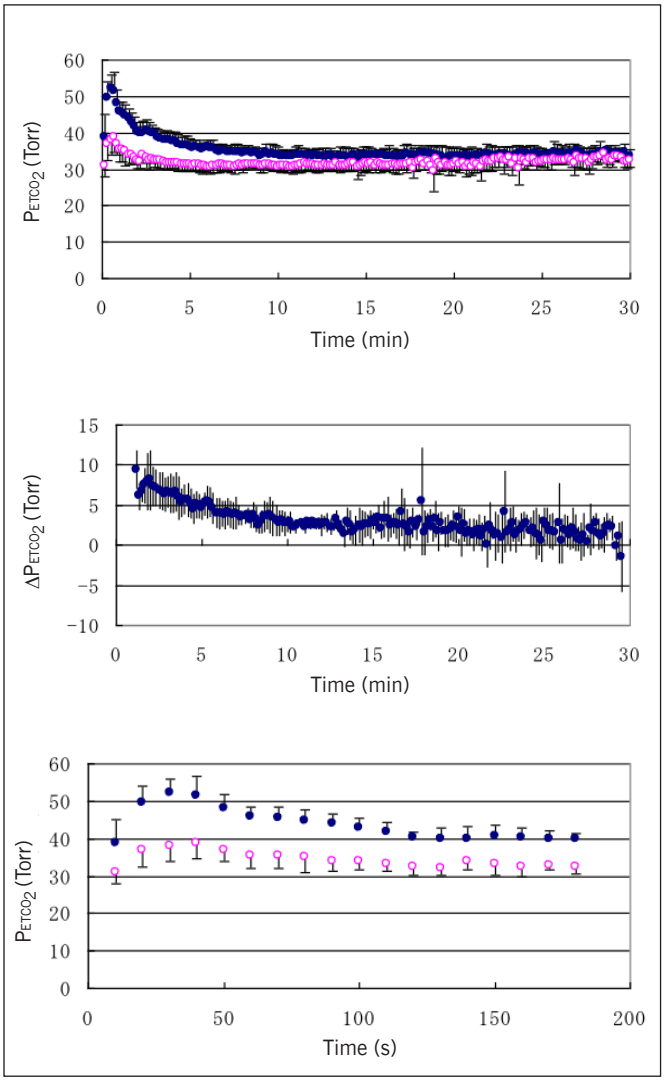

FIG. 4. END TIDAL CO_2 PRESSURE (P_{ETCO_2}) (UPPER PANEL) DURING RECOVERY FROM ONE SPRINT (CLOSED CIRCLES) AND DURING RECOVERY FROM FIVE REPEATED SPRINTS (OPEN CIRCLES). DIFFERENCE BETWEEN END TIDAL PRESSURE AFTER ONE SPRINT AND THAT AFTER FIVE REPEATED SPRINTS (ΔP_{ETCO_2}) (MIDDLE PANEL). P_{ETCO_2} FOR THE FIRST 3 MIN OF RECOVERY FROM ONE SPRINT (CLOSED CIRCLES) AND THAT FOR THE FIRST 3 MIN OF RECOVERY FROM FIVE REPEATED SPRINTS (OPEN CIRCLES) (LOWER PANEL).

at rest (38.8 ± 2.77 Torr). The minimum value appeared at 21 min during recovery from one sprint (32.3 ± 1.68 Torr) and was significantly lower than the resting value. The final value (34.0 ± 2.04 Torr) was significantly higher than the minimum value after one sprint. The minimum value appeared at 19 min during recovery from five sprints (29.2 ± 5.27 Torr) and was significantly lower than the resting value. The final value (32.5 ± 3.93 Torr) was significantly higher than the minimum value after five sprints. The difference between P_{ETCO_2} after one sprint and that after five sprints decreased with time but did not become zero until 30 min of recovery. There was a significant difference between P_{ETCO_2} at each time during recovery after one sprint and after five sprints until 21 min.

There were exponential relationships between $\dot{V}E$ and La from 5 min to 30 min of recovery (Figure 5). Significant correlation coefficients were obtained for one sprint (r=0.780) and for five sprints (r=0.736). At the same La level, ventilation after one sprint was higher than that after five sprints. However, the difference became small when the La level was low.

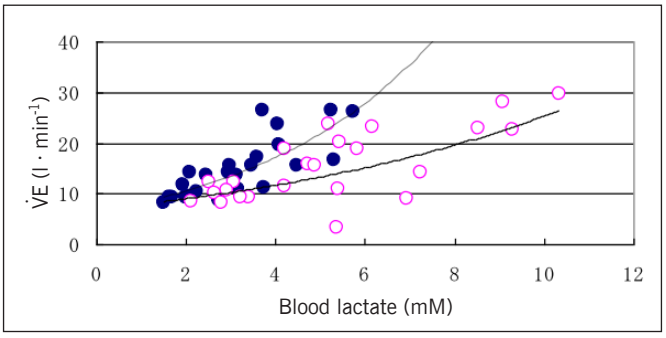

FIG. 5. RELATIONSHIPS BETWEEN BLOOD LACTATE AND VENTILATION (VE) AFTER 5 MIN IN RECOVERY. THE RELATIONSHIPS IN ALL SUBJECTS WHO PARTICIPATED IN ONE SPRINT (CLOSED CIRCLES) AND IN FIVE REPEATED SPRINTS (OPEN CIRCLES) ARE SHOWN.

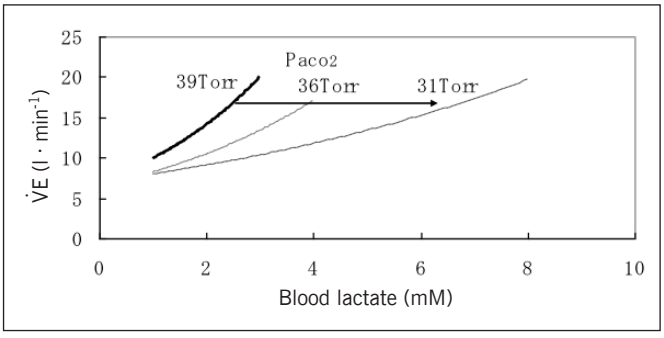

FIG. 6. SCHEMATIC EXPLANATION OF THE RELATIONSHIP BETWEEN BLOOD LACTATE AND VENTILATION ($\dot{V}E$) DURING RECOVERY. THE THICK LINE SHOWS THE RELATIONSHIP BETWEEN BLOOD LACTATE AND VENTILATION WHEN ARTERIAL CO_2 PRESSURE (P_{aco_2}) IS AT THE RESTING LEVEL. THIS LINE IS IMAGINARY. IT CAN BE SHIFTED IN RESPONSE TO CHANGE IN P_{aco_2}. SINCE P_{aco_2} IS DECREASED BELOW THE RESTING LEVEL IN BOTH SPRINTS, THE LINE REPRESENTS THE RELATIONSHIPS IN ONE SPRINT (DOTTED CURVE) AND IN FIVE REPEATED SPRINTS (SOLID CURVE) IN RESPONSE TO THE DECREASED VALUES OF P_{aco_2}.

DISCUSSION

The purpose of the present study was to examine whether $\dot{V}E$ kinetics are related to La level after 5 min in recovery after a sprint. There was an exponential relationship between $\dot{V}E$ and La after 5 min in recovery. The curve shifted to the right in the case of recovery from five repeated sprints. Although $\dot{V}E$ after 50s in recovery from five repeated sprints was the same as that from 50 s in recovery from one sprint, La during recovery from one sprint was significantly lower than that during recovery from five repeated sprints. The difference between P_{ETCO_2} after one sprint and that after five repeated sprints decreased with time but did not become zero until 30 min of recovery.

It has been observed in constant-load exercise that $\dot{V}E$ abruptly increases (phase I), shows an exponential rise (phase II) and then shows a steady state in moderate exercise (phase III) [16]. Haouzi et al. [7] have also reported that $\dot{V}E$ and P_{ETCO_2} in impulse exercise show a peak and then increase again during recovery until 40-50 s. This increase during recovery involves the time delay effect produced by impulse exercise. In the present study, La as well as P_{ETCO_2} increased during this period. La and P_{ETCO_2} can affect the increase in ventilation during recovery. After this increase, $\dot{V}E$ decreased, while La increased until 5 min of recovery in the present study, indicating that there may be factors other than La affecting ventilation. Thus, factors for ventilation control during the early period of recovery from a sprint could not be identified in this experiment.

$\dot{V}E$ after 5 min in recovery was exponentially associated with La level. Since an increase in La can result in a decrease in pH [10] and an increase in H^+ [12], La can stimulate ventilation. The La and ventilation curve obtained after repeated sprints was located on the right side compared with that obtained after one sprint. It has been reported that there is an unknown controlling factor that is not related to $Paco_2$ and pH in $\dot{V}E$ during recovery from intense exercise [3]. That factor may be derived from some effect of the CNS such as afterdischarge [5]. However, the time constant that characterises the decreasing rate of afterdischarge is too short to explain the $\dot{V}E$ level at 30 min.

In the present study, it is likely that the difference in La level was counteracted by the difference in P_{ETCO_2} level and consequently $\dot{V}E$ kinetics attained the same level in both sprints. However, as mentioned in the introduction, there is a threshold in the ventilation and P_{ETCO_2} relation [4]. Although this threshold is around the resting value, it has not been tested whether this threshold is affected by metabolic acidosis. Therefore, it is uncertain whether the threshold determined in the test without metabolic acidosis is valid. Furthermore, the decrease in P_{ETCO_2} can inhibit the increase in H^+ by exercise-induced metabolic acidosis. Therefore, P_{ETCO_2} may have an effect through change in H^+.

The actual relationship between La and $\dot{V}E$ is always affected by $Paco_2$, and the relationship in an isocapnic status is therefore not actually observed but can be imagined as a solid line as shown

in Figure 6. The imaginary relationship can be shifted to the right by changes in the values of $Paco_2$. When La reaches the resting level, P_{ETCO_2} also reaches the resting level. In this case, all curves converge on one point in the relationship between La and $\dot{V}E$.

CONCLUSIONS

Ventilation kinetics during recovery from one sprint were the same as the kinetics during recovery from five repeated sprints. This may be because ventilation control related to blood lactate level is modified by end tidal CO_2 pressure.

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