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RELATION BETWEEN EXCESSIVE CO_2 EXPIRATION AND PERFORMANCE IN HIGH-INTENSITY EXERCISE

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Abstract. This study examined the relation between excessive CO_2 expiration and exercise performance in exercise exhausted for 1-2 min. First, the Wingate test was conducted to determine the exercise intensity of the performance test. The exercise intensity was set at 80% of the power exerted during the last 5 s of the Wingate test so that subjects could continue for 1-2 min. Total work, which was the value of the work rate times the performed duration was used as an index of the performance level. The excessive CO_2 expiration rate (‡CO_2excess) was defined as the difference between CO_2 output and O_2 uptake. Integration of ‡CO_2excess from the start of exercise to the zero level of ‡CO_2excess observed at around 10 min in recovery was defined as the amount of excessive CO_2 expiration (CO_2excess). The performance was associated with the peak value of blood lactate (peak La) obtained after exercise (r=0.744). The performance was also associated with CO_2excess (r=0.848). It is likely that CO_2excess is a better physiological index for assessing the performance in exercise exhausted for 1-2 min than peak La.

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Key words: CO_2 excess-intensive exercise-blood lactate-performance

Introduction

Performance in exercise exhausted for 1-2 min is associated with peak blood lactate after exercise [4,7]. This was interpreted as relating to the amount of energy derived from lactic formation (lactic capacity). However, a recent study demonstrated that a peak blood lactate after exercise does not reflect the amount of lactate formation [8]. Therefore, the relation between the performance and amount of lactate formation has not been verified.

The lactic acid formed in exercise is hydrolyzed. Within the physiological range

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in pH, the produced hydrogen ion is equivalent to the formed lactate ion but is buffered in the body. Therefore, the actual increase of hydrogen ion is very slight. This buffer action in the active muscle is mainly made by a non-bicarbonate buffer system and absorption of hydrogen ion accompanied by the resolution of phosphocreatine (CrP) [2]. After stopping exercise, lactic acid is spread throughout the entire body and consequently is buffered by the entire body rather than active muscle. Since CrP is recovered after exercise, hydrogen ion absorbed by the resolution of CrP is released. Therefore, the action of CrP is invalid when buffer action from exercise to recovery considered. As the buffer action in the extracellular fluid is mainly owing to the bicarbonate buffer system, buffer action after exercise is mainly dependent on the bicarbonate buffer system, which can be observed as excessive CO_2 expiration [9]. Thus, through the shift of lactic acid from the muscle in exercise to the entire body during recovery, buffer action in exercise could be assessed by the excessive CO_2 expiration (CO_2excess).

Lactic acid produced during intense exercise is involved in the development of fatigue [3]. A high ability of the active muscles to release lactic acid and the high buffer capacity of muscle could prevent muscle functionality and delay the onset of fatigue. Lactic acid released from the active muscle in exercise induces CO_2excess. Lactic acid accumulated in the muscle in exercise gradually spreads throughout the entire body during recovery. This also causes the CO_2 excess. As the buffer capacity of active muscle and the release ability of lactic acid from active muscle have an advantage in the performance in exercise exhausted for 1-2 min, the amount of CO_2excess in exercise and during recovery could be used to assess performance.

In the present study, therefore, we examined whether the performance in the exercise exhausted for 1-2 min is associated with the amount of CO_2 excess.

Material and Methods

Table 1
Characteristics of the subjects and results of the Wingate test

	Age (years)	Height (cm)	Weight (kg)	Peak power (watts)	Average power (watts)
Mean	21	172	62	695	538
SD	0.9	4.1	6.5	130	82

Thirteen men belonging to a sports club in university participated in the present experiments. Physical characteristics of the subjects are listed in Table 1. Each subject was informed of the purpose of the study, the experimental procedure, and the risks associated with the experiments before consent was obtained. Furthermore, they were instructed to abstain from heavy training on the day before the experiments and rest for one hour prior to the start of the experiments.

A cycle ergometer (Powermax-VII, Combi) was used in the experiments. On the first day, each subject performed a 30-s maximal exercise test at a load (kp) corresponding to 7.5% x body weight (Wingate test). During the Wingate test, the power output (watts) was calculated from the imposed load and cycling rate (rpm) averaged for every 5 s. A few days later, a short-term intensive exercise test (SIET) was performed at a work load corresponding to 80% of the power exerted during the last 5 s of the Wingate test [11]. Each subject, after resting on the cycle ergometer for 5 min, performed SIET until he could no longer maintain 90 rpm. After the end of SIET, each subject sat on a chair for a 30-min recovery period.

Ventilation (‡E), O_2 uptake (‡O_2), CO_2 output (‡CO_2) and end tidal CO_2 pressure ($PETCO_2$) were measured breath-by-breath using a respiratory gas analyzer (Aeromonitor AE-280S, Minato) throughout the SIET. These data were averaged and were outputted for each 20-s period. ‡E was measured by a hot-wire flow meter that was fixed to the front of the subject's gas mask. The flow meter was calibrated with a syringe of known volume (2.0 l). Sample gas was drawn continuously from the front of the gas mask (220 $ml \cdot min^{-1}$) for determination of the fractional concentrations of O_2 and CO_2 by a zirconium sensor and an infrared absorption analyzer, respectively. The gas analyzer was calibrated by set standard gases. Twenty-five microliters of blood were sampled from a fingertip using a capillary tube at rest before exercise, at 0, 3, 6, 10, 20, and 30 min after the end of exercise. The lactate concentration (La) in the sampled blood was determined by an automatic lactate analyzer (1500 sport, YSI). The analyzer was calibrated with a standard liquid (5 mM of lactate, YSI).

Excessive CO_2 expiration per unit time (‡CO_2excess) was calculated by subtracting the ‡O_2 values from the ‡CO_2 values during the SIET [10,11]. The ‡CO_2excess was integrated from the start of exercise to the zero level of ‡O_2excess., and this was defined as the total excessive CO_2 expiration (CO_2excess).

The strength of the relation between dependent and independent variables was expressed by a single correlation coefficient of Pearson. A P value of 0.05 or less was significant. Results were expressed as mean ± standard deviation (SD).

Results

Table 2
Blood lactate (mM) at rest and during recovery

	Rest	Recovery					
		0 min	3 min	6 min	10 min	20 min	30 min
Mean	0.97	8.03	9.69	9.55	8.46	6.54	4.69
SD	0.22	1.96	1.55	1.77	1.82	1.78	1.56

Table 2 shows La at rest and during recovery. La immediately after the SIET increased by 77% of peak La. The peak La was observed at 3 or 6 min after SIET. Then La decreased. The work rate given for the performance test of SIET ranged from 279 watts to 453 watts. The duration of SIET was almost the same (70 to 110 s). Total work, which was obtained by multiplying the duration and a given work rate for SIET ranged from 22320 to 37510 joule. There was a significant relation between total work and peak La after SIET (Fig. 1, r=0.744).

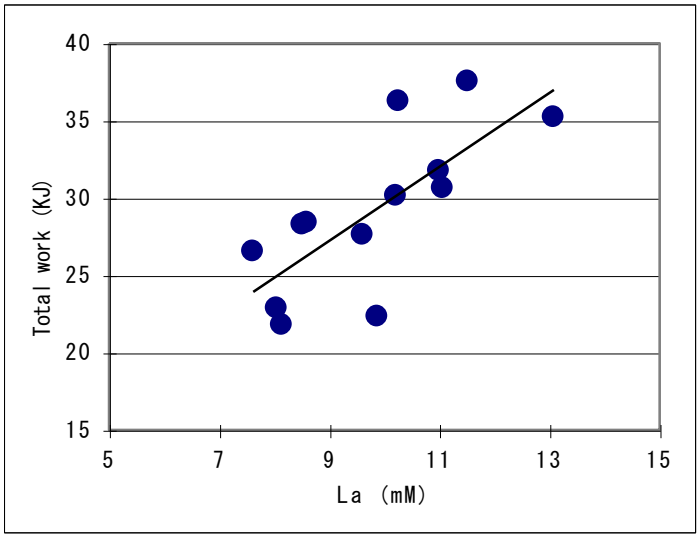

Fig. 1
Relation between peak blood lactate (La) after exercise and total work performed in exercise

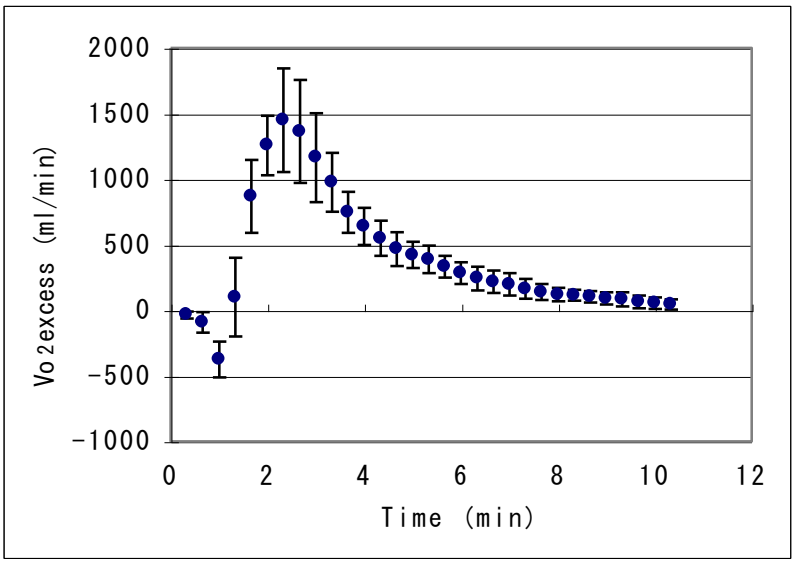

Fig. 2
Relation between amount of excessive CO_2 expiration (CO_2excess) and total work performed in exercise

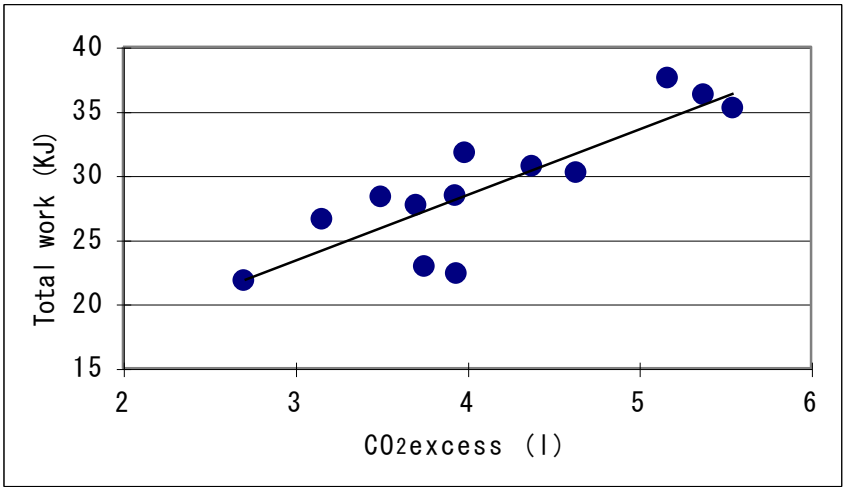

Fig. 3
Change in excessive CO2 expiration per unit time (‡CO_2excess) from the start of

exercise to recovery phase

There was a significant relation between total work and CO_2excess (Fig. 2, r=0.848). As shown in Fig. 3, ‡CO_2excess did not increase but decreased at the onset of SIET. Then ‡CO_2excess started to increase before the end of SIET and showed peak at around at 1.5 min after SIET. ‡CO_2excess became zero level at around 10 min. $PETCO_2$ increased in SIET. $PETCO_2$ started to decrease during recovery and dropped below resting level (Fig. 4).

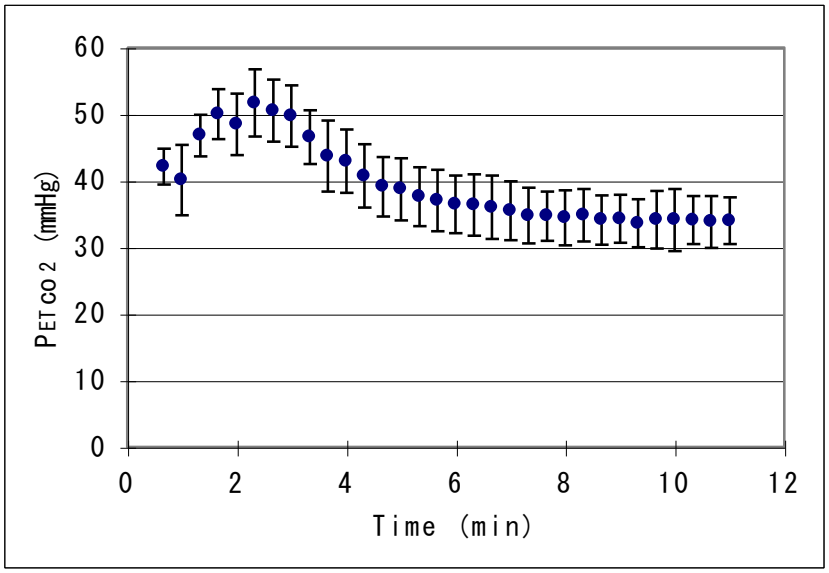

Fig. 4
Change in end tidal CO_2 pressure ($PETCO_2$) from the start of exercise to recovery phase

Discussion

‡O_2excess showed negative values in SIET. This could be due to the rise of CO_2 pressure. As venous CO_2 pressure increases in intensive exercise [6], this would result in the increase of bicarbonate ion not only in the active muscle but also in venous blood. As a result, ‡CO_2excess can be depressed. At the end of exercise, lactic acid is thought to move from the muscle to extra cellular fluid, since the La immediately after SIET increased. This suggests an increase in ‡CO_2excess.

However, the rise of CO_2 pressure could mask the effect of the lactic acid shift. The mask was released after exercise due to the decrease in CO_2 pressure. Therefore, even if lactate is not shifted into the extra cellular fluid, ‡CO_2excess is increased. Afterwards, $PETCO_2$ reached below the resting level. As this is induced by the hyperventilation, ‡ CO_2excess continues. However, the hyperventilation leads to overestimation of CO_2excess.

CrP is synthesized during recovery. This causes the increase of hydrogen ion in the muscle. Although lactic acid is spread throughout the entire body after exercise, this does not always mean that all hydrogen ion spreads throughout the entire body. However, there is a mechanism to exchange Na^+ and H^+ [2,5]. This may well work to help the buffer action in the muscle by spreading the hydrogen throughout the entire body.

Entire body buffer action consists of a non-bicarbonate buffer system and bicarbonate buffer system [2,9]. The former system does not induce CO_2excess. This leads to underestimation of the amount of lactate formation by CO_2excess. However, this underestimation may be counteracted by the overestimation due to hyperventilation.

As mentioned in Introduction, peak La is an index of lactic capacity. This index was associated with the performance. Since it is considered that the performance exhausted for 1-2 min is mainly determined of lactic capacity, the obtained relation between the performance and peak La is well understandable. We postulated that CO_2excess should be a new physiological index for assessing the performance exhausted for 1-2 min. The CO_2excess was also associated with the performance. Furthermore, correlation coefficient of this relation was higher than that obtained in peak La. Although there is a limitation in the use of the CO_2exess, it is likely that CO_2excess is a better physiological index for assessing the performance in exercise exhausted for 1-2 min than peak La.

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