The Differences in CO₂ Kinetics during Incremental Exercise among Sprinters, Middle, and Long Distance Runners

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Abstract The purpose of this study was to investigate the differences in kinetics of CO₂ output ($\dot{V}_{CO_2}$) during incremental exercise in sprinters (S), middle (MD), and long distance runners (LD). In the steady state exercise, the $\dot{V}_{CO_2}$ was linearly related to the O₂ uptake ($\dot{V}_{O_2}$). In the incremental exercise below anaerobic threshold (AT), the $\dot{V}_{CO_2}$ was also linearly related to the $\dot{V}_{O_2}$. The difference between the $\dot{V}_{CO_2}$ estimates from the regression lines obtained in steady state and incremental exercise was added from the start of exercise up to a given time. The added values were defined as CO₂ stores. The CO₂ stores per body weight were significantly related to mixed venous CO₂ pressure ($P_{vCO_2}$) determined by the CO₂ rebreathing method. The slopes of the regression lines between $P_{vCO_2}$ and CO₂ stores per body weight were not different among three groups. If $\dot{V}_{CO_2}$ above AT is estimated from the $\dot{V}_{O_2}$ using the regression line obtained in incremental exercise below AT, the estimated $\dot{V}_{CO_2}$ is lower than the measured $\dot{V}_{CO_2}$. The sum of the differences in $\dot{V}_{CO_2}$ up to a given time was defined as CO₂ excess. The CO₂ excess per body weight was significantly related to ΔLA (the difference between blood lactates at 5 min after exercise and at rest). The ratios of CO₂ excess per body weight to ΔLA were 3.30 ± 1.49, 4.16 ± 2.33, and 5.55 ± 2.05 for sprinters, middle, and long distance runners, respectively. This ratio obtained in sprinters was significantly lower than that in long distance runners ($p < 0.01$).

Key words: CO₂ stores, CO₂ excess, mixed venous CO₂ pressure, blood lactate.

It is known that metabolically produced CO₂ is partially stored in the body (CO₂ stores) at the onset of exercise (Linnarsson, 1974). Due to the stored CO₂, it is expected that the CO₂ pressure in the tissue, the venous CO₂ pressure and the mixed venous CO₂ pressure are increased successively. In fact, it has been shown that mixed venous CO₂ pressure is increased in proportion to the increase of CO₂ stores.

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(YANO et al., 1984a; YANO, 1986).

It is well known that blood lactic acid is increased at higher exercise intensities. To buffer the H⁺ dissociated from lactic acid, CO₂ is expired excessively (CO₂ excess). ISSEKUTZ and RODAHL (1961) tried to quantitatively measure the CO₂ excess in untrained subjects. The CO₂ excess at the onset of steady state exercise was, then, estimated from the difference between the CO₂ output and the product of O₂ uptake and metabolic gas exchange ratio (RQ) which was assumed to be 0.75. However, BOHUYS et al. (1966) were unable to reproduce the excellent correlation between blood lactate change and CO₂ excess calculated by the Issekutz method in trained subjects. It can also be pointed out that ISSEKUTZ and RODAHL (1961) neglected the effect of CO₂ stores at the onset of exercise. To ameliorate these problems, CLODE and CAMPBELL (1969) measured the change of mixed venous CO₂ pressure (PvCO₂) as well as of blood lactate (ΔLA) from rest to exercise. At the same time, metabolic CO₂ production was estimated from oxygen uptake by assuming a constant RQ and the difference between CO₂ production and measured CO₂ output was assumed to be derived from the change in body CO₂ storage. However, as this estimate cannot accurately evaluate the independent effects of both lactate and CO₂ storage on expiratory gas, the comparison of ΔLA and CO₂ excess or PvCO₂ and CO₂ stores is insufficient.

In incremental exercise, it is known that blood lactate is increased above a given level of exercise intensity (AT), and CO₂ simultaneously begins to be excessively exhaled (WASSERMAN et al., 1973). This excessive CO₂ elimination can be a tool to estimate the change in CO₂ storage due to lactate production. Furthermore, metabolically produced CO₂ in incremental exercise is partially expired and the rest is stored. Due to this CO₂ stores, CO₂ output becomes lower than that expected from the oxygen uptake. This phenomenon is observed below AT in incremental exercise. If this difference found below AT is extrapolated to the work load above AT, the CO₂ stores can be estimated throughout the work load. These make it possible to separately compare the CO₂ stores–PvCO₂ and CO₂ excess–ΔLA relations (YANO, 1984a).

Both CO₂ pressure increase and lactic acid production that happen in the incremental exercise result in H⁺ production. If one has high buffer values, one's increasing rate of bicarbonate against the CO₂ pressure increase should be high and thereby one can store large quantities of CO₂. Meanwhile, the bicarbonate system shares in the role for buffering lactic acid with the nonbicarbonate system. If one has a high buffer value in the nonbicarbonate system, the role of buffering action by the bicarbonate system can relatively be reduced and eventually one may reduce the CO₂ elimination due to lactate production (CO₂ excess). From the above, the purpose of the present study is to examine if the individual difference of buffer value makes the differences in CO₂ stores–PvCO₂ and CO₂ excess–ΔLA relations. For this purpose sprinters, middle, and long distance runners were employed as subjects in three experimental groups in the present study, since PARKHOUSE et al. (1983) measured muscle buffer value using a biopsy technique and reported that...
sprinters have higher buffer value than marathon runners.

METHODS

The subjects were male college students enrolled in a track and field athletic club and consisted of four sprinters (S) specializing in 100 and 200 m dashes, six middle distance runners (MD) specializing in 400 to 1,500 m races, and six long distance runners specializing in races of 5,000 m or more. Table 1 shows mean and S.D. in age, height, weight, maximal O$_2$ uptake ($V_{O_2}^{max}$), and blood lactate at 5 min after exhaustion ($LA_{max}$) for the three groups.

The subjects exercised at incremental and steady state work loads using a bicycle ergometer (Monark). On the first day, resting values were determined at 5 to 10 min while subjects were resting for 15 min. The incremental exercise whose work load was increased at a rate of 180 kp m/min with 60 rpm every minute, was performed from 180 kp m/min to exhaustion. On the second day, after the same procedure of resting measurement as carried out on the first day, incremental exercise with the same procedure as carried out the first day but only up to 540 kp m/min was started. After 10 min recovery following the incremental exercise, the subjects exercised for 10 min at a steady state work rate of 900 kp m/min and continued at a work rate of 1,080 kp m/min for another 10 min. On the third day, after the same procedure of resting measurement as carried out on the first day, the subjects exercised at a steady state work rate of 360 kp m/min for 10 min and then at 720 kp m/min for another 10 min. After at least 30 min recovery, incremental exercise with the same procedure as carried out on the first day but up to 1,080 kp m/min was started. These three series were performed on separate days within a week (Fig. 1).

At rest in the three series, respiratory gas was collected for 5 min with a Douglas bag. In steady state exercise, O$_2$ uptake ($V_{O_2}$) and CO$_2$ output ($V_{CO_2}$) were

Table 1. Physical characteristics, maximal O$_2$ uptake, and maximal blood lactate in long distance runners (LD), middle distance runners (MD), and sprinters (S).

<table>
<thead>
<tr>
<th></th>
<th>Age (years)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>$V_{O_2}^{max}$ (l/min)</th>
<th>$V_{O_2}^{max}$/weight (ml/(min·kg))</th>
<th>$LA_{max}$ (mm/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LD</td>
<td>19.7±1.80</td>
<td>171.2±4.14</td>
<td>57.5±4.79</td>
<td>3.64±0.34</td>
<td>63.1±5.78</td>
<td>9.74±1.92</td>
</tr>
<tr>
<td>MD</td>
<td>20.3±0.94</td>
<td>172.0±5.16</td>
<td>61.5±4.43</td>
<td>3.61±0.22</td>
<td>59.0±4.66</td>
<td>10.40±2.21</td>
</tr>
<tr>
<td>S</td>
<td>21.8±0.43$^b$</td>
<td>173.0±6.12</td>
<td>67.0±5.92$^{**}$</td>
<td>3.29±0.12$^b$</td>
<td>49.4±2.72$^{**}$</td>
<td>8.80±1.98</td>
</tr>
</tbody>
</table>

Values are the mean ± S.D. $^*$p<0.05, $^{**}$p<0.01, $^{***}$p<0.001. $^a$Differences between LD and S. $^b$Differences between MD and S.
determined at 6 to 8 min, and 8 to 10 min at 360 and 720 kp m/min, and 8 to 9 min and 9 to 10 min at 900 and 1,080 kp m/min during each 10-min exercise. In the three incremental exercises, \( \dot{V}_{O2} \) and \( \dot{V}_{CO2} \) were determined for every 1 min throughout the exercises. The ventilation was measured by a dry gas meter. The \( O2 \) and \( CO2 \) were analyzed by a mass spectrometer (Perkin Elmer MGA-1100).

Blood samples from an antecubital vein were taken immediately before and 5 min after incremental exercise on the first day and the third day. Samples were analyzed by lactate analyzer LA 640 (Roche) to determine the blood lactate (LA).

Mixed venous \( CO2 \) pressure (\( P_{\dot{V}CO2} \)) was determined by the \( CO2 \) rebreathing method (DEFARES et al., 1961). The rebreathing gas containd 5% of \( CO2 \) in \( O2 \) balance. The \( CO2 \) rebreathing was performed immediately after expired gas collections in steady state exercise and incremental exercise. The \( CO2 \) during rebreathing was analyzed by a mass spectrometer (Perkin Elmer MGA-1100). The \( CO2 \) pressure measured during rebreathing was used to estimate \( P_{\dot{V}CO2} \) (detail in YANO et al., 1984b).

Gas exchange parameters used for the determination of anaerobic threshold (AT) consisted primarily of ventilation, \( CO2 \) output, fractional concentration of expired \( CO2 \) and fractional concentration of expired \( O2 \) (WASSERMAN et al., 1973; DAVIS et al., 1976). These criteria were plotted against work rate. These criteria for AT determination can be summarized as follows: a) The point of departure from a linear relationship in both ventilation and \( \dot{V}_{CO2} \) which can be assumed at a certain
b) Abrupt increase in fractional concentration of expired CO₂. c) A systemic increase in ventilation-equivalent for O₂.

The regression line was determined individually in the relationship between $\dot{V}_{O₂}$ and $\dot{V}_{CO₂}$ obtained in four steady state exercises ($\dot{V}_{CO₂} = c\dot{V}_{O₂} + d$). The regression line was also determined individually for the relationship between $\dot{V}_{O₂}$ and $\dot{V}_{CO₂}$ obtained in incremental exercise below AT ($\dot{V}_{CO₂} = a\dot{V}_{O₂} + b$). When $\dot{V}_{O₂}$ determined in incremental exercise was substituted into two regression lines respectively, each predicted $\dot{V}_{CO₂}$ was obtained. The differences between these predicted $\dot{V}_{CO₂}$ were added from a start to a given time of exercise. These added values were defined as CO₂ stores (see Fig. 2).

$$\text{CO₂ stores} = \sum ((c-a)\dot{V}_{O₂} + (d-b)).$$

The equation obtained below AT ($\dot{V}_{CO₂} = a\dot{V}_{O₂} + b$) was extrapolated at the exercise intensity above the AT. The differences between this extrapolated and the measured $\dot{V}_{CO₂}$ were added from the work rate at AT to a given time of exercise. The added values were defined as CO₂ excess.
\[ \text{CO}_2 \text{ excess} = \sum (\dot{V}_{\text{CO}_2} - (a \dot{V}_{\text{O}_2} + b)). \]

The \( \dot{V}_{\text{O}_2} \) and \( \dot{V}_{\text{CO}_2} \) in the incremental exercise were the values averaged for three measurements up to 540 \( \text{kpm/min} \) and thereafter for two measurements up to 1,080 \( \text{kpm/min} \) at each identical work rate.

RESULTS

The coefficient of correlation between \( \dot{V}_{\text{O}_2} \) and \( \dot{V}_{\text{CO}_2} \) in steady state exercise ranged from 0.999 to 0.996 \((p < 0.01)\). In the incremental exercise, \( \dot{V}_{\text{O}_2} \) obtained below AT was also significantly related to the \( \dot{V}_{\text{CO}_2} \) \((r = 0.993-0.999, p < 0.01)\). This obtained regression line \( (\dot{V}_{\text{CO}_2} = a \dot{V}_{\text{O}_2} + b) \) was almost parallel to and lower than the regression line obtained in steady state exercise \( (\dot{V}_{\text{CO}_2} = c \dot{V}_{\text{O}_2} + d) \).

The relationship between \( \text{CO}_2 \) stores per body weight (\( \text{CO}_2 \) stores/w) and \( P\tilde{V}_{\text{CO}_2} \) determined in incremental exercise is shown in Fig. 3. The \( \text{CO}_2 \) stores/w was significantly related to \( P\tilde{V}_{\text{CO}_2} \) in sprinters \((r = 0.681, p < 0.05)\), middle \((r = 0.768, p < 0.001)\), and long distance runners \((r = 0.702, p < 0.01)\). The slopes of the regression lines were 0.447(S), 0.302(MD), and 0.351(LD), respectively. There were no differences in the slopes among three groups.

Figure 4 shows \( \text{CO}_2 \) excess per weight (\( \text{CO}_2 \) excess/w) and \( \Delta LA \) (difference of LA between values at 5 min after exercise and rest). Since six subjects did not attain their AT at 1,080 \( \text{kpm/min} \), \( \text{CO}_2 \) excess at 1,080 \( \text{kpm/min} \) could not be obtained. Therefore, ten data were presented at 1,080 \( \text{kpm/min} \) in the figure. \( \Delta LA \) was significantly related to the \( \text{CO}_2 \) excess/w \((S: r = 0.933, p < 0.001; \text{MD: } r = 0.863,\)

![Fig. 3. Relationship between \( \text{CO}_2 \) stores per body weight and \( P\tilde{V}_{\text{CO}_2} \). Symbols are assigned for sprinters (---), middle distance runners (--○), and long distance runners (--×).](image)
p <0.01; LD: r = 0.804, p < 0.05). The ratio of CO₂ excess/w to ΔLA was calculated in every subject. The averaged values were 3.30 ± 1.49 (n = 8), 4.16 ± 2.32 (n = 10), and 5.55 ± 2.05 (n = 8) for sprinters, middle, and long distance runners, respectively. This ratio obtained in sprinters was significantly lower than that in long distance runners (p < 0.01).

**DISCUSSION**

In the present study, increasing rates of CO₂ stores/w against the increase of \( P_{\text{CO}_2} \) (body CO₂ slope) were measured in sprinters, middle, and long distance runners, respectively. The body CO₂ slopes were not different among the groups and ranged from 0.320 to 0.477 ml/(kg·mmHg).

MiYAMURA and HONDA (1978) examined the slope of blood CO₂ dissociation curve (blood CO₂ slope) by theoretical analysis and experiment, and suggested that change in blood CO₂ slope was influenced by the buffer value of the system. Accordingly when the buffer value is high in the system, the blood CO₂ slope can become high according to their theory. If the result in blood CO₂ dissociation can be applied to the systemic CO₂ dissociation, the body CO₂ slope can be assumed to be affected by the buffer value of the subjects. Since PARKHOUSE et al. (1983) found higher buffer values in sprinters than in marathon runners using biopsy technique, in the present study the \( P_{\text{CO}_2} - \text{CO}_2 \) stores relation was examined in sprinters,
middle, and long distance runners. Clode et al. (1967) also studied the body \( CO_2 \) slope with reducing the \( CO_2 \) stores by hyperventilation or obtaining the \( CO_2 \) stores by continuous rebreathing into a 5-l bag but found no difference in the body \( CO_2 \) slopes between untrained and trained subjects. Thus neither the present study nor previous study confirmed the above mentioned hypothesis.

In calculation of \( CO_2 \) stores in the present study, the \( CO_2 \) metabolically produced in the incremental exercise was assumed to be obtained by the relationships between \( \dot{V}_{O_2} \) and \( \dot{V}_{CO_2} \) in steady state exercise. Furthermore, the expired \( CO_2 \) was estimated from the regression line below the AT. Then this regression line was used to estimate \( \dot{V}_{CO_2} \) also at exercise intensity above the AT. In a previous study, Yano (1986) tried to quantitatively obtain \( CO_2 \) stores in light steady state exercise without blood lactate production. It was then assumed that the metabolic gas exchange ratio (RQ) was obtained from respiratory gas exchange ratio (R) at 5 min of exercise, and \( CO_2 \) stores were obtained from the difference between \( CO_2 \) output and the product of \( O_2 \) uptake and the determined RQ. Although this assumption in calculation of \( CO_2 \) stores was different from that of the present study, the body \( CO_2 \) slope was reported to be 0.330 ml/(kg·mmHg) which was in good agreement with the present results. Thus no quantitative discrepancy was found by adapting the present assumptions.

The coefficient of correlation between \( P_{CO_2} \) and \( CO_2 \) stores/\( w \) ranged from 0.681 to 0.768 in the present study. In the previous study performed to examine the relationship between \( P_{CO_2} \) and \( CO_2 \) stores/\( w \) at the onset of steady state exercise (Yano, 1986), a similar coefficient was reported (\( r = 0.713 \)). Thus a coefficient of around 0.7 may be the inherent value using these methods and indicates that \( P_{CO_2} \) can predict \( CO_2 \) stores/\( w \) with about 50% accuracy from coefficient of determination (\( r^2 \)). This accuracy may, however, have been somewhat low for finding out the difference in slopes among groups.

In the present study the ratios of \( CO_2 \) excess/\( w \) to ALA in sprinters, middle, and long distance runners were 3.30, 4.10, and 5.55 (ml/kg)/(mm/l), respectively. This ratio obtained in sprinters was significantly lower than that in long distance runners.

A buffer value (\( \beta \)) is defined as a ratio of the buffered \( H^+ \) (\( \Delta H^+ \)) and the change of pH (\( \Delta pH \)). This ratio can be expressed in the bicarbonate and nonbicarbonate systems, and the \( \Delta pH \) is a common item in both the systems. When these relations are used,

\[
\Delta pH = \Delta H^+_B/\beta_B = \Delta H^+_N/\beta_N = \Delta H^+_T/(\beta_B + \beta_N),
\]

where subscript \( T \) was expressed for the total of the bicarbonate and nonbicarbonate system, \( B \) for bicarbonate system, \( N \) for nonbicarbonate system. When these equations are rearranged:

\[
\beta_B/(\beta_B + \beta_N) = \Delta H^+_B/\Delta H^+_T.
\]

Lactic acid produced during exercise (\( \Delta ALA \)) is fully dissociated and the released \( H^+ \) (\( \Delta H^+_T \)) is practically equivalent to the produced lactic acid in the physiological
range of pH (Hultman and Sahlin, 1980). To buffer the released H\(^+\), bicarbonate is decreased (\(\Delta HCO_3^-\)) almost in proportion to the degree of buffered H\(^+\) (\(\Delta H^+\)).

\[
\beta_B/(\beta_B + \beta_N) = \Delta HCO_3^-/\Delta LA.
\]

At the same time, the amount of CO\(_2\) nearly equivalent to the decrease of bicarbonate is expired (CO\(_2\) excess).

\[
\beta_B/(\beta_B + \beta_N) = CO_2\text{ excess}/\Delta LA.
\]

According to this equation, the ratio in the left side can be reduced when nonbicarbonate buffer value is augmented without changing bicarbonate buffer value. This reduction in the ratio can result in the decrease of CO\(_2\) excess when comparing the same amount of lactate production. Parkhouse et al. (1983) reported that sprinters possessed higher muscle buffer values due to the carnosine level than marathon runners. These suggested that less CO\(_2\) excess would be expected in sprinters.

There is another possible to explanation for the differences among the groups: It is known that buffering is mainly due to protein in the intracellular fluid and to bicarbonate in the extracellular fluid (Ganong, 1973). If H\(^+\) is buffered in the extracellular fluid more than in the intracellular fluid, the ratio of H\(^+\) buffered by bicarbonate system would become large in the total body fluid. The capillarity in the muscle of endurance-trained men is dense (Saltin et al., 1977) and H\(^+\) dissociated from lactic acid may flow into the extracellular fluid.

The present study clearly indicates that there were differences in CO\(_2\) excess to \(\Delta LA\) relation among the present groups and suggests two possibilities for explaining the differences. It would be apparent in either explanation that these differences derived from those in contributing rate of bicarbonate as buffering system in the whole body fluid.

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REFERENCES


