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Effect of arterial carbon dioxide on ventilation during recovery from impulse exercises of various intensities

Authors

Roghayyeh Afroundeh[1)], Takuma Arimitsu[1)], Ryo Yamanaka[1)] Chang-shun Lian[1)] Takahiro Yunoki[2)], Tokuo Yano[2)]

Affiliations

[1)] Graduate School of Education, Hokkaido University, Sapporo, Japan

[2)] Department of Human Developmental Sciences, Faculty of Education, Hokkaido University, Sapporo, Japan

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Corresponding Author: Tokuo Yano Ph.D.

Address: Department of Human Development Science, Faculty of Education, Hokkaido University, Kita-11, Nishi-7, Kita-ku, Sapporo 060-0811, Japan

E-mail: **yano@edu.hokudai.ac.jp**

Tel & Fax: +81-11-706-5090

Abstract

To determine that whether arterial carbon dioxide ($PaCO_2$) affects ventilation ($\dot{V}E$) during recovery from impulse-like exercises of various intensities, subjects performed four impulse-like tests with different workloads. Each test consisted of a 20-sec impulse-like exercise at 80 rpm and 60-min recovery. Blood samples were collected at rest and during recovery to measure blood ions and gases. $\dot{V}E$ was measured continuously during rest, exercise and recovery periods. A significant curvilinear relationship was observed between $\dot{V}E$ and pH during recovery from the 300 and 400 watts tests in all subjects. $\dot{V}E$ was elevated during recovery from the 100 watts test despite no change in any of the humoral factors. Arterialized carbon dioxide ($PaCO_2$) kinetics showed fluctuation, being increased at 1 min and decreased at 5 min during recovery, and this fluctuation was more enhanced with increase in exercise intensity. There was a significant relationship between $\dot{V}E$ and $PaCO_2$ during recovery from the 300 and 400 watts tests in all subjects. The results of the present study demonstrate that pH and neural factors drive $\dot{V}E$ during recovery from impulse-like exercise and that fluctuation in $PaCO_2$ controls $\dot{V}E$ as a feedback loop and this feedback function is more enhanced as the work intensity increases.

Keywords: arterial CO_2 pressure, blood pH, impulse-like exercise, recovery, ventilation

Introduction

Carbon dioxide pressure in arterial plasma ($PaCO_2$) at rest is the factor that has the greatest influence on regulation of ventilation ($\dot{V}E$), and there is a linear relationship between $\dot{V}E$ and $PaCO_2$ known as Oxford model (5). This linear relationship between $PaCO_2$ and $\dot{V}E$ has also been introduced as a chemoreflex model that describes a ventilatory recruitment threshold that is the $PaCO_2$ level below which $\dot{V}E$ is unaffected by $PaCO_2$ and above which $\dot{V}E$ is linearly related to $PaCO_2$ (7, 8). However, in light-to-moderate exercise, $\dot{V}E$ closely couples with O_2 consumption and CO_2 production, and $PaCO_2$ generally remains at a constant level under these conditions (18, 29, 30). In addition, during strenuous exercise with a relatively large anaerobic component, lactic acidosis and subsequent H^+ concentration provide an additional ventilatory stimulus (hyperventilation) that reduces $PaCO_2$ (16, 25). Thus, it is thought that the Oxford model cannot be simply adopted for $\dot{V}E$ kinetics in exercise.

The mechanisms subserving $\dot{V}E$ control during exercise and recovery have been studied widely, and many factors have been suggested to drive $\dot{V}E$ during exercise and recovery (6) including metabolic acidosis or increase in plasma hydrogen ion [H^+] (3, 17, 20, 25, 27, 28, 30), central command (4, 32) and arterial potassium (K^+) (33). These factors may conspire to control $\dot{V}E$ independently of $PaCO_2$ during exercise (21). However, the results of our previous study revealed that $\dot{V}E$ during recovery from one impulse-like exercise was not different from $\dot{V}E$ during recovery from five repeated impulse-like exercises despite different pH levels, and the similarity of $\dot{V}E$ could be explained by $PaCO_2$ kinetics suggesting that $PaCO_2$ itself has an effect on $\dot{V}E$ (1). These findings indicate that the role of $PaCO_2$ in $\dot{V}E$ control is controversial and needs to be investigated more. Therefore, the purpose of the present study was to investigate the effect of $PaCO_2$ on $\dot{V}E$ response during recovery from impulse-like exercises of different work

intensities. We were interested in impulse-like exercise because the byproducts of metabolic acidosis in muscles enter plasma at the end of exercise (during recovery) when there is no muscle contraction to stimulate $\dot{V}E$, and the rise in $\dot{V}E$ triggered by this impulse forcing occurs well after cessation of the contractions (14, 26).

Methods

Subjects

Seven healthy active males participated in this study. The subjects' mean age, height and body weight were 21.2 ± 1.7 (SD) yr, 173.7 ± 7.4 cm and 66.2 ± 8.1 kg, respectively. Each subject signed a statement of informed consent following a full explanation regarding the nature of the examination. The Ethics Committee of Hokkaido University Graduate School of Education approved the present study.

Design

Each subject attended our laboratory for four tests. The time interval between two consecutive tests was at least 2 days, and all tests were completed within one month. Each subject was instructed to refrain from intense physical exercise, drinking alcohol and taking caffeine for 24 h prior to the tests. None of the subjects had a smoking habit.

Examination protocol

Each subject performed four tests consisting of one impulse-like exercise for 20 sec on separate days. Tests were performed with resistive loads of 100, 200, 300 and 400 watts at 80 rpm by an ergometer (POWERMAX-V_{II}, Combi, Tokyo, Japan). Each subject came to the laboratory 1 hour before the start of the test. Examination instruments were attached to the

subjects before the examination. Subjects performed impulse-like tests after resting for 3 min on a bicycle seat. The duration and load were adjusted by a built-in computer.

Measurements and determinations

Blood samples (125 µl) were collected from fingertips using a capillary tube. Each subject's hand was pre-warmed in 40-45^{0}C water prior to each test in order to arterialize capillary blood. It has been shown that such blood samples might not accurately reflect arterial O_2 pressure but can closely reflect arterial CO_2 and pH (34). Twenty five-µl samples were analyzed using a lactate analyzer (YSI-1500 sport, YSI, Ohio, USA) to measure blood lactate concentration (La^-), and 100-µl samples were analyzed using a blood gas analyzer (i-STAT, i-STAT Corporation, Abbott Park, IL, USA) to measure O_2 partial pressure (PaO_2), $PaCO_2$, potassium concentration (K^+) and pH. HCO_3^- concentration [(HCO_3^-)] was calculated from pH and PCO_2 by using the Henderson-Hasselbalch equation. The lactate analyzer was calibrated by a standard lactate solution of 5 mmol.l^{-1} and the blood gas analyzer was calibrated by known calibration liquid (pH: 7.43, PCO_2: 30 Torr, PO_2: 160 Torr, [Na^+]: 140 mEq.l^{-1}, [K^+]: 4 mEq.l^{-1}) before each test. Blood was sampled at rest and after 1 min, 5 min, 10 min, 15 min, 30 min and 60 min during the recovery period.

$\dot{V}E$ 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: 4.9%). $\dot{V}E$ was measured continuously during rest, exercise and recovery periods. For each 30-sec interval, the averages of $\dot{V}E$ were calculated.

Statistical analysis

Results are presented as means ± standard deviations (SD). One-way ANOVA for repeated measures was used to examine the time effect. If F ratios were significant, the Bonferroni post-hoc test was used for comparison. Two-way ANOVA for repeated measurements was used for comparison between tests. If a significant interaction was indicated, one-way ANOVA was used to examine differences between the four tests. A value of $p < 0.05$ was regarded as statistically significant.

Results

pH, La^- and $PaCO_2$ levels are shown in Figure 1. pH values were decreased significantly at 1 min during recovery from all tests except for the 100 watts test, for which there was no significant difference at any time point. There were significant differences in pH values between the 100 and 300 watts tests at 1 min during recovery and between the 100 and 400 watts tests at 1 min and 5 min during recovery ($p < 0.05$). There was no significant difference between the 100 and 200 watts tests ($p > 0.05$).

La^- level significantly increased and peaked at 5 min during recovery from the 400 watts test (3.57±0.63 mmol/l) and at 1 min during recovery from the 300 watts and 200 watts tests (2.49±0.31 mmol/l and 1.71±0.41 mmol/l, respectively) and then decreased to the resting values (0.83±0.11 mmol/l, .081±0.23 mmol/l and 0.86±0.24 mmol/l, respectively). It did not change during recovery from the 100 watts test. There were significant differences in La^- values between the 100 and 300 watts tests at 1 min, 5 min, 10 min and 15 min during recovery and between the 100 and 400 watts tests at 1 min, 5 min, 10 min, 15 min and 30 min during recovery ($p<0.05$).

The results showed that there was fluctuation in $PaCO_2$ level during recovery from the 300 and 400 watts tests. In the 400 watts test, $PaCO_2$ was increased significantly at 1 min during recovery versus the rest level (45 ± 3.2 mmHg and 38.7 ± 1.2 mmHg, respectively). Although it was not statistically significant ($p>0.05$), the level of $PaCO_2$ was higher than the rest value at 1 min during recovery from the 300 watts test (42.9 ± 2.6 mmHg and 39 ± 2.2 mmHg, respectively). After that it fell below the resting level at 5 min and was significantly different compared with the level of 1 min in both tests ($p<0.05$).

The higher the work intensity was, the higher was arterialized $PaCO_2$ at 1 min during recovery and the lower was arterialized $PaCO_2$ at 5 min during recovery. There was a significant relationship between work intensity and arterialized $PaCO_2$ at 1 min, 5 min and 10 min during recovery (Figure 2).

No significant change was observed in PaO_2 levels during recovery at any time point in all tests ($p>0.05$). Mean values for pH, $PaCO_2$ and PaO_2 are presented in Table I. There was no significant differences between the four tests in $[K^+]$ during recovery at any time point ($p>0.05$). Mean values for $[K^+]$ and La^- are presented in Table II.

As can be seen in Figure 3, $\dot{V}E$ was elevated during recovery from the 100 watts test and it reached the rest value after about 4 min. There was no significant difference in $\dot{V}E$ during recovery between the 100 and 200 watts tests. $\dot{V}E$ was significantly different during recovery from the 100 and 300 watts tests until 270 sec ($p<0.05$). A significant difference was also found during recovery until 390 sec between the 100 and 400 watts tests ($p<0.05$).

We obtained the relationship between $\dot{V}E$ and pH during recovery from the 300 watts and 400 watts tests using data for all subjects, and the relationship was exponential (Figure 4). High correlation coefficients were obtained for the 300 watts test ($r = 0.742$; $p<0.05$) and 400 watts test

($r = 0.852$; $p<0.05$). There was also a significant relationship between $\dot{V}E$ and $PaCO_2$ during recovery from the 300 watts and 400 watts tests using data for all subjects (Figure 5). Significant correlation coefficients were obtained for the 300 watts test ($r = 0.605$; $p<0.05$) and 400 watts test ($r = 0.706$; $p<0.05$).

Discussion

The subjects in the present study performed four impulse-like tests with different work intensities and duration of 20 sec. A significant relationship was observed between $\dot{V}E$ and pH during recovery from the 300 and 400 watts tests for all subjects, but this relationship was curvilinear. $\dot{V}E$ was elevated during recovery from the 100 watts test despite no change in any of the humoral factors. $PaCO_2$ kinetics showed fluctuation, being increased at 1 min and decreased at 5 min during recovery, and this fluctuation was more enhanced with increase in exercise intensity.

High correlation coefficients obtained for the pH – $\dot{V}E$ relationships during recovery from the 300 and 400 watts tests suggest that pH has an effect on $\dot{V}E$ during recovery from impulse-like exercise. It has been reported that carotid bodies are responsible for respiratory compensation for the metabolic acidosis of exercise (23, 31), and these chemoreceptors are stimulated with H^+ ions (3) and as a consequence contribute to hyperventilation. The results of the 100 watts test showed that $\dot{V}E$ was elevated during recovery for approximately 4 min. None of the humoral factors measured in this study changed during recovery from the 100 watts test. Therefore, neural factors might have an effect on $\dot{V}E$ during recovery from impulse-like exercise. One of the possible neural mechanisms driving $\dot{V}E$ during recovery is after-discharge, or short-term potentiation of ventilatory drive that sustains hyperpnoea even after a stimulus is withdrawn (12). Although the

time constant for after-discharge has been reported to range from 51 to 57 sec in anesthetized cats (10, 11) the results in unanesthetized animals showed a longer time constant with a period of over 5 min (9). The other possible mechanism can be thin fiber afferents. Although thin fiber afferents (i.e., groups III and IV) are thought to respond to metabolic stimuli (15), it has been reported that these afferents may also play an important role in increasing ventilation at low levels of dynamic exercise when the metabolic demand of working muscles is not large (2). Therefore, it is possible that these afferents play a role in stimulating ventilation during recovery from impulse exercise, even in the 100 watts test in the present study.

The main findings of the present study were that $PaCO_2$ was increased at 1 min during recovery from all tests except for the 100 watts test and that it was increased more in higher than lower work intensity tests. After this augmentation, $PaCO_2$ dropped significantly at 5 min versus 1 min during recovery, and it was declined more in higher than lower work intensity tests (Figure 1). These results are novel and different from results of previous studies showing a constant or decreased level of $PaCO_2$ during exercise (16, 18, 25, 29, 30). Since changes in $PaCO_2$ are dependent on the $\dot{V}E$ response to exercise (19), this increase in $PaCO_2$ at 1 min suggests that $\dot{V}E$ is not sufficient to expire CO_2 adequately, and since this response is enhanced with increase in work intensity, it seems that work intensity is a hindrance for $\dot{V}E$ response. The fluctuation of $PaCO_2$ observed in the present study indicates that it acts as a feedback loop in the process of $\dot{V}E$ control. Thus, when the ventilatory response to main stimulatory factors (pH and neural factors) is not adequate due to some hindrance (work intensity in the present study), $PaCO_2$ increases at 1 min and stimulates $\dot{V}E$, and consequently more CO_2 is expired from the lungs, resulting in a decrease in the level of $PaCO_2$ at 5 min. Therefore, the high level of $PaCO_2$

is reduced via a feedback mechanism and this feedback function is more enhanced as the work intensity increases (Figure 2).

Although there was a significant relationship between pH and $\dot{V}E$ during recovery from the 300 and 400 watts tests, but this relationship was curvilinear, indicating that other factors in addition to pH might be involved in $\dot{V}E$ control. In support of our results, it has been shown that the increase in plasma H^+ concentration is responsible for only ~30% of the hyperventilation during exercise and that a delayed hyperventilatory response was observed when pH was maintained by bicarbonate infusion, suggesting that other control mechanisms of hyperventilation are involved (17, 20). K^+, which has been reported to be another humoral factor to possibly be capable of stimulating chemoreceptors to induce exercise hyperpnoea during exercise and recovery (24, 33), was not significantly different compared with rest values at any work load in the present study. Thus, we cannot ascribe $\dot{V}E$ kinetics to K^+. However, a high correlation coefficient was obtained for the relationship between $\dot{V}E$ and $PaCO_2$ in both the 300 and 400 watts tests (Figure 5), indicating that $PaCO_2$ also has an effect on $\dot{V}E$. It is believed that the blood-brain barrier is relatively impermeable to H^+ but permeable to CO_2, and consequently central chemoreceptors would be stimulated by hypercapnia more than by acute metabolic acidosis of arterial blood (3). Carotid bodies are also known to respond rapidly to hypercapnia (13).This is consistent with the feedback concept of $PaCO_2$ explained in the previous paragraph.

Although the subjects' fitness level (e.g., maximal oxygen uptake) was not assessed in the present study, it is speculated from the subjects' daily physical activity that our subjects were active. It has been reported that ventilatory response to exercise is different dependding on the physical fitness level of subjects (22). However, it was confirmed in that study (22) that change

in breathing pattern with increase in exercise intensity was the same irrespective of the level of physical fitness. Therefore, our findings can be generalized to a broad range of the population.

In conclusion, the results of the present study demonstrate that pH and neural factors drive $\dot{V}E$ during recovery from impulse-like exercise and that fluctuation in $PaCO_2$ controls $\dot{V}E$ as a feedback loop and this feedback function is more enhanced as work intensity increases.

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Table I. Mean values ± SD of arterialized blood pH, Oxygen (PaO_2) and Carbon dioxide ($PaCO_2$) in the four tests

			Recovery					
		Rest	1 min	5 min	10 min	15 min	30 min	60 min
pH	100 watt	7.41±0.02	7.40±0.02	7.41±0.02	7.39±0.01	7.42±0.02	7.41±0.01	7.41±0.02
	200 watt	7.42±0.02	7.38±0.02*	7.41±0.01	7.40±0.02	7.42±0.01	7.41±0.02	7.41±0.02
	300 watt	7.42±0.01	7.35±0.02*†	7.38±0.02*	7.39±0.01$^{\#}$	7.42±0.01	7.41±0.01	7.41±0.01
	400 watt	7.42±0.01	7.31±0.02*†	7.36±0.02$^{*\dagger\#}$	7.38±0.02$^{*\#}$	7.39±0.02	7.39±0.01	7.41±0.02
PaO_2 (mmHg)	100 watt	86.1±5.9	87.3±6.8	89.9±4.2	86.3±4.1	90.0±9.5	84.3±3.7	84±5.3
	200 watt	88.7±6.4	89.4±10.9	91.6±10.1	90.0±8.8	85.8±6.6	84.0±6.7	79.6±4.7
	300 watt	88.9±5.4	94.9±8.3	96.3±7.8	89.1±6.4	88.3±7.2	81.6±5.2	82.1±9.6
	400 watt	86.7±10.3	93.7±7.5	99.1±5.6	92.1±8.4	88.0±7.3	83.9±7.4	81.3±3.9
$PaCO_2$ (mmHg)	100 watt	38.4±2.8	39.6±2.5	39.7±2.2	40.4±2.2	39.3±2.2	40.2±2.2	40.0±2.1
	200 watt	38.8±2.5	41.1±2.1	38.3±1.2	39.5±1.6	39.9±1.5	40.4±2.1	40.8±1.9
	300 watt	39.0±2.2	42.9±2.6	38.2±3.1$^{\#}$	38.7±2.6$^{\#}$	38.3±1.9	40.1±2.6	40.5±2.6
	400 watt	38.7±1.2	45.0±2.9*†	37.5±1.9$^{\#}$	37.5±3.0$^{\#}$	38.2±2.1	39.9±1.4	40.3±1.8

Values represent means ± SD (N= 7) for each time point. *significant difference compared with rest value in all tests; $^{\#}$ significant difference compared with 1 min in all tests; †significant difference compared with 100 watts test; $p < 0.05$.

Table II. Mean values ± SD of arterialized blood lactate (La^-) and potassium (K^+) in the four tests

			Recovery					
		Rest	1 min	5 min	10 min	15 min	30 min	60 min
La (mmol.l^{-1})	100 watt	1.00±0.21	1.12±0.23	1.00±0.18	0.99±0.18	0.97±0.13	0.88±0.17	0.83±0.27
	200 watt	0.86±0.24	1.71±0.41*†	1.44±0.43	1.31±0.31	1.12±0.43	1.05±0.39	0.93±0.49
	300 watt	0.81±0.23	2.49±0.31*†	2.46±0.52*†	2.07±0.50*†	1.61±0.38*†	1.14±0.29	1.04±0.44
	400 watt	0.83±0.1	2.70±0.44*†	3.57±0.63$^{*\dagger\#}$	2.84±0.68*†	2.21±0.54*†	1.40±0.35*†	0.87±0.19
K^+ (mmol.l^{-1})	100 watt	3.90±0.24	3.94±0.19	3.97±0.25	3.81±0.20	3.93±0.18	3.86±0.18	4.07±0.19
	200 watt	3.90±0.24	3.94±0.26	3.86±0.19	3.94±0.24	3.96±0.25	3.79±0.23	3.93±0.17
	300 watt	3.96±0.21	4.07±0.18	3.81±0.17	3.84±0.15	3.89±0.19	3.80±0.14	4.01±0.23
	400 watt	4.00±0.28	4.10±0.18	3.90±0.26	3.94±0.29	3.84±0.26	3.84±0.24	4.00±0.25

Values represent means ± SD (N= 7) for each time point. *significant difference compared with rest value in all tests; # significant difference compared with 1 min in all tests; †significant difference compared with 100 watts test; $p < 0.05$.

Legends of figures

Fig. 1 Changes in arterialized blood lactate (La^-), carbon dioxide ($PaCO_2$), and pH during recovery from 100 watts (open circles), 200 watts (closed circles), 300 watts (open triangles), and 400 watts (closed triangles) tests. Data presented are means ± SD. *Significantly different compared with rest values ($p<0.05$). $^\#$Significantly different compared with values at 1 min ($p<0.05$). †Significantly different compared with 100 watts ($p<0.05$).

Fig. 2 Relationships between arterialized carbon dioxide ($PaCO_2$) and work intensities (watts) at 1 min (upper panel, r = 0.995), 5 min (middle panel, r = 0.919) and 10 min during recovery from impulse-like exercise (Lower panel, r = 0.996).

Fig. 3 Differences in ventilation ($\dot{V}E$) during recovery between 100 watts and 200 watts tests (upper panel), between 100 watts and 300 watts tests (middle panel), and between 100 watts and 400 watts tests (lower panel). Data presented are means ± SD. †Significantly different compared with 100 watts ($p<0.05$).

Fig. 4 Relationships between arterialized pH and ventilation ($\dot{V}E$) during recovery from the 300 watts test (open triangles; r = 0.742, $p<0.05$) and the 400 watts test (closed triangles; r = 0.852, $p<0.05$). Data presented are data for all subjects.

Fig. 5 Relationships between arterialized carbon dioxide ($PaCO_2$) and ventilation ($\dot{V}E$) during recovery from the 300 watts test (open triangles; $r = 0.605$, $p<0.05$) and the 400 watts test (closed triangles; $r = 0.706$, $p<0.05$). Data presented are data for all subjects.

Figure 1

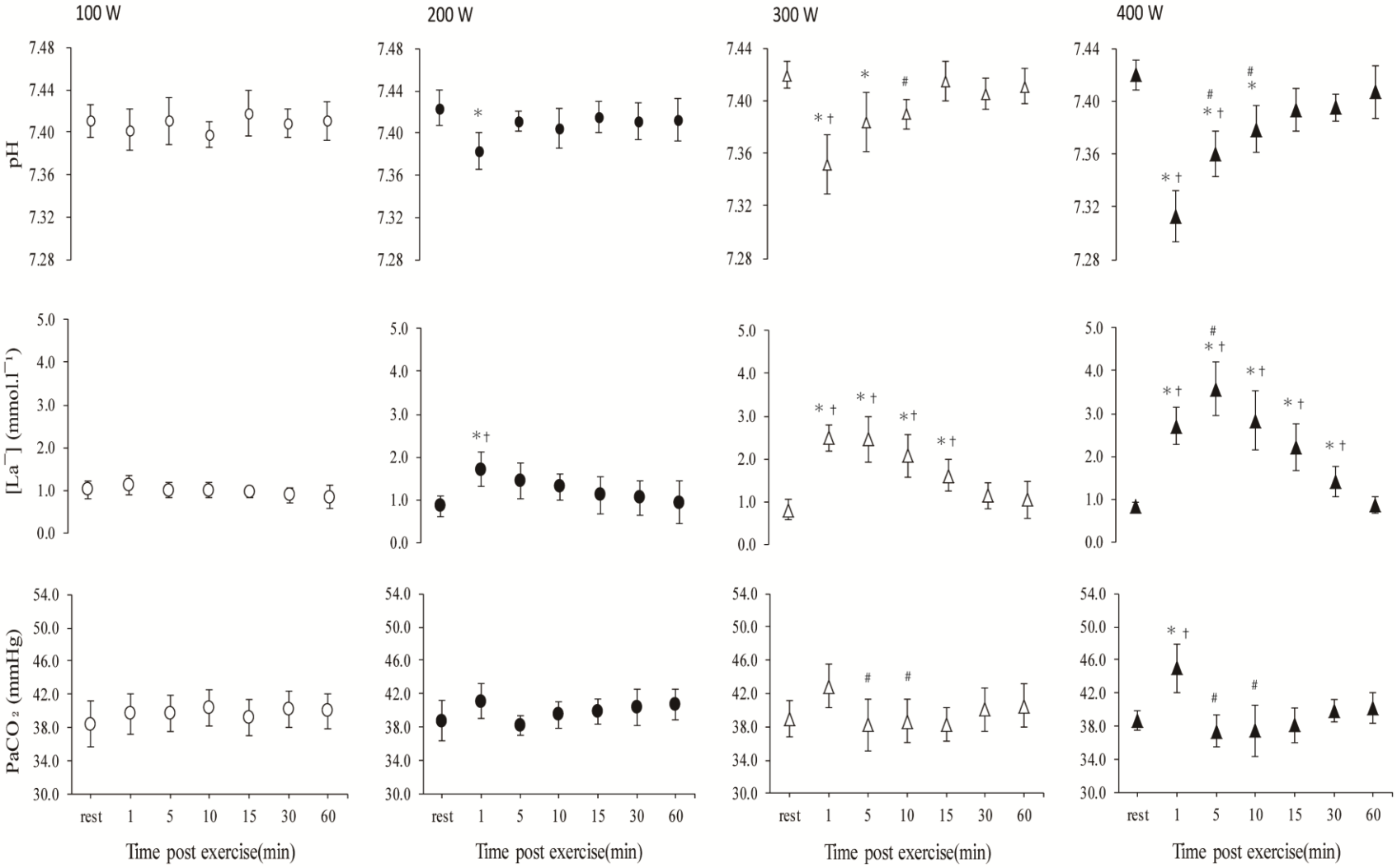
100 W
200 W
300 W
400 W
pH
[La⁻] (mmol.l⁻¹)
PaCO₂ (mmHg)
rest
1
5
10
15
30
60
Time post exercise(min)

Figure 2

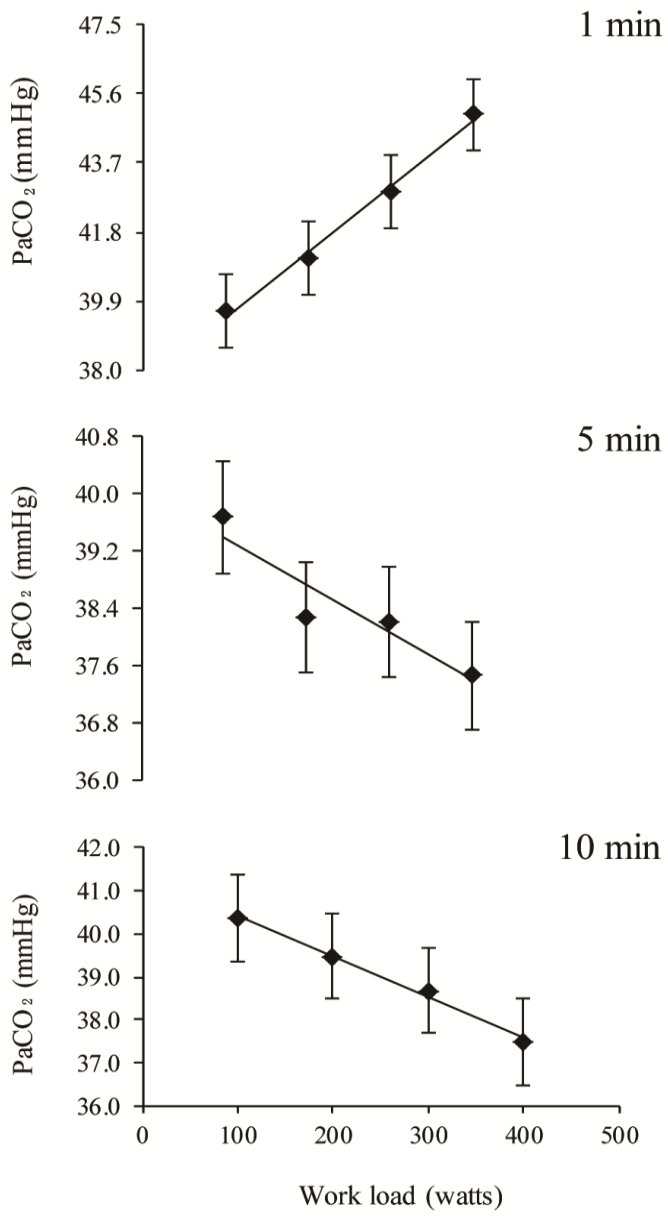
1 min
5 min
10 min
PaCO₂ (mmHg)
47.5
45.6
43.7
41.8
39.9
38.0
40.8
40.0
39.2
38.4
37.6
36.8
36.0
42.0
41.0
40.0
39.0
38.0
37.0
36.0
0
100
200
300
400
500
Work load (watts)

Figure 3

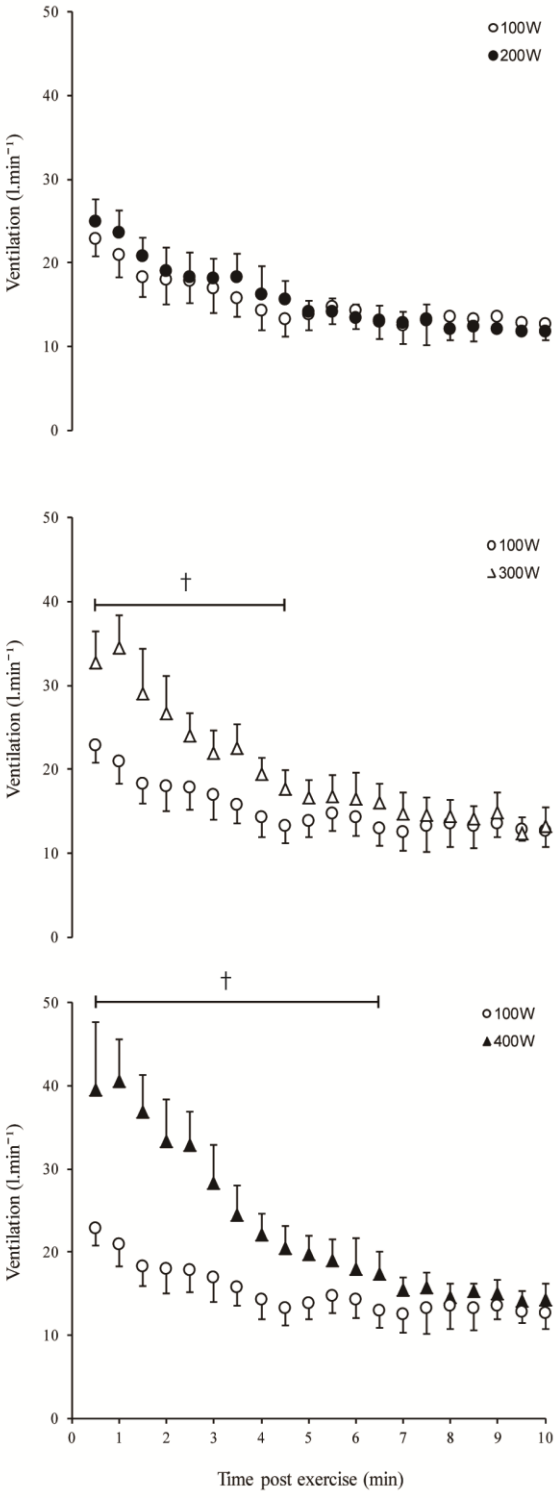
100W
200W
Ventilation (l.min⁻¹)
100W
300W
†
100W
400W
†
Time post exercise (min)

Figure 4

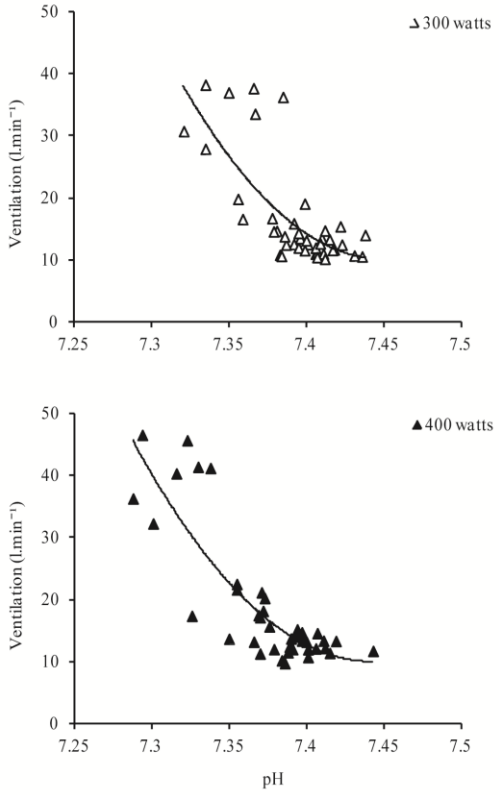
300 watts
400 watts
Ventilation (Lmin⁻¹)
pH
50
40
30
20
10
0
7.25
7.3
7.35
7.4
7.45
7.5

Figure 5