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Relationship between ventilation and predicted arterial CO_2 pressure during recovery from an impulse-like exercise without metabolic acidosis

Authors

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

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: Roghayyeh Afroundeh

Address: Graduate School of Education, Hokkaido University, Kita-11, Nishi-7, Kita-ku, Sapporo 060-0811, Japan

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

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

Abstract

We investigated ventilation ($\dot{V}E$) control factors during recovery from light impulse-like exercise (100 watts) with a duration of 20 sec. Blood ions and gases were measured at rest and during recovery. $\dot{V}E$, end tidal CO_2 pressure ($PETCO_2$) and respiratory exchange ratio (RER) were measured continuously during rest, exercise and recovery periods. Arterial CO2 pressure ($PaCO_{2\ pre}$) was estimated from $PETCO_2$ and tidal volume (V_T). RER at 20 sec of exercise and until 50 sec during recovery was significantly lower than RER at rest. Despite no change in arterialized blood pH level, $PaCO_{2\ pre}$ was significantly higher in the last 10 sec of exercise and until 70 sec during recovery than the resting value. $\dot{V}E$ increased during exercise and then decreased during recovery; however, it was elevated and was significantly higher than the resting value until 155 sec ($p<0.05$). There was a significant relationship between $\dot{V}E$ and $PaCO_{2\ pre}$ during the first 70 sec of recovery in each subject. The results suggest that $PaCO_2$ drives $\dot{V}E$ during the first 70 sec of recovery after light impulse-like exercise. Elevated $\dot{V}E$ in the interval from 70 sec until 155 sec during recovery might be due to neural factors.

Keywords: arterial CO_2 pressure, impulse-like exercise, recovery, respiratory exchange ratio, ventilation

Introduction

The chemosensory mechanism (feedback control) is one of the important ventilatory control mechanisms in exercise (Dempsey 2006, Waldrop et al. 2006). This mechanism involves stimulation of peripheral chemoreceptors by an increase in hydrogen ion concentration [H^+] (Peronnet et al. 2007, Ward 2007, Wasserman et al. 1975, Whipp 1994) or by arterial potassium (K^+) (Yoshida et al. 1990).

It is known that exercise of a very short duration with maximal effort can induce production of lactic acid (Gaitanos et al. 1993, Osnes and Hermansen 1972) and that lactic acid persists in the blood for a long time after exercise (Knuttgen et al. 1972). During the early period of recovery, a downward shift in the CO_2 dissociation curve may occur due to an increase in blood lactate (ΔLa). 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 ($\dot{V}E$) (Yano et al. 2009).

Arterial carbon dioxide pressure ($PaCO_2$) can also stimulate $\dot{V}E$ through peripheral and central chemoreceptors (Clement et al. 1992). However, $PaCO_2$ remains at constant level during moderate exercise (Oren et al. 1981, Wasserman et al. 1967) and $PaCO_2$ is reduced during strenuous exercise due to hyperventilation (Kowalchuk et al. 1988, Stringer et al. 1992). Likewise, $PaCO_2$ is the major factor controlling [H^+] known as the Stewart model (Duffin 2010, Stewart 1983). Therefore, it is thought that $PaCO_2$ has no effect on $\dot{V}E$ in an exercise condition or that it has an indirect effect via changes in [H^+] (Duffin 2005, Poon 2011). Nevertheless 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 the difference in $PaCO_2$

kinetics, suggesting that pH is not the only humoral factor that drives $\dot{V}E$ and that $PaCO_2$ has a direct effect on $\dot{V}E$ (Afroundeh et al. 2012).

In addition, a recent study showed that ventilatory response during recovery from intense exercise is not associated with pH and $PaCO_2$ but is associated with a central neural mechanism (Yamanaka et al. 2011). Involvement of neural factors in $\dot{V}E$ control during recovery from intense exercise has also been suggested by Clement et al. (Clement et al. 1996).

In order to extract only the effect of $PaCO_2$ on $\dot{V}E$, we decided to study $\dot{V}E$ control during recovery from work of a very light intensity that induces no metabolic acidosis. Furthermore, it has not been determined whether central and/or peripheral neural factors affect $\dot{V}E$ during recovery from exercise of very light intensity. Therefore, the purpose of the present study was to determine the relationship between $PaCO_2$ and $\dot{V}E$ and to determine whether neural factors are involved in $\dot{V}E$ control during recovery from an impulse-like exercise that induces no metabolic acidosis.

Methods

Subjects

Six healthy males participated in this study. The subjects' mean age, height and body weight were 21.6 ± 2.1 (SD) yr, 173.6 ± 8.2 cm and 66.7 ± 8.7 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

Each subject attended our laboratory to perform one test. 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.

Experimental protocol

Each subject performed one test consisting of one impulse-like exercise for 20 sec. The test was performed with resistive load of 100 watts at 80 rpm by a bicycle ergometer (Ergometer 232 CXL, Combi, Tokyo, Japan). Each subject came to the laboratory 1 hour before the start of the test. An experimental instrument was attached to each subject before the experiment. Subjects performed 100 watts impulse-like test after resting for 3 min on a bicycle seat.

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 (Zavorsky et al. 2007). 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 the test. Blood was sampled at rest and after 1 min, 5 min, and 10 min during the recovery period.

Data on respiration gas exchange were obtained using a respiratory gas analyzer (AE-280S, Minato Medical Science, Osaka, Japan). $\dot{V}E$ 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%). Respiration gas exchange was measured continuously during rest, exercise, and recovery periods.

To obtain continuous data of $PaCO_2$, it was estimated from end tidal CO_2 pressure ($PETCO_2$) and tidal volume (V_T) using the formula from Jones, Robertson & Kane (1979):

Predicted $PaCO_2$ ($PaCO_{2\,pre}$) = 5.5 + 0.90 $PETCO_2$ – 0.0021V_T.

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 Dunnet post-hoc test was used for comparison. A value of $p < 0.05$ was regarded as statistically significant.

Results

No significant change was observed in arterialized pH, La^-, K^+, HCO_3^-, PaO_2 and $PaCO_2$ levels during recovery at any time point versus rest time ($p>0.05$). Mean values of these humoral factors are presented in Table 1.

$PaCO_2$ was predicted from $PETCO_2$ and tidal volume (V_T) . Both $PaCO_{2\,pre}$ and $PETCO_2$ were increased during exercise and peaked at 20 sec during recovery (41.05 ± 1.82 mmHg and 42.08 ± 2.14 mmHg, respectively) and then decreased to the resting values (34.69 ± 1.97 mmHg

and 34.33 ± 2.5 mmHg, respectively). They were significantly higher at 15 sec and 20 sec during exercise and until 70 sec during recovery compared with the rest values ($p<0.05$). The kinetics of $PETCO_2$ and $PaCO_{2\ pre}$ was the same during impulse-like exercise and during recovery from impulse-like exercise. These changes are shown in Figure 1.

As can be seen in Figure 2, respiratory exchange ratio (RER) decreased during exercise and the first 20 sec during recovery. It was significantly lower at 20 sec during exercise and until 50 sec during recovery versus the rest value ($p<0.05$). $\dot{V}E$ increased during exercise, reached the highest level at 20 sec of exercise (27.45 ± 4.02 $l.min^{-1}$), and then decreased during recovery from impulse-like exercise. However, the decline in $\dot{V}E$ at 5 sec of recovery (starting point of recovery) was not significant compared to $\dot{V}E$ at the end of exercise and it was elevated and significantly higher than the rest value (12.09 ± 1.69 $l.min^{-1}$) until 155 sec during recovery from impulse-like exercise ($p<0.05$).

There was a significant relationship between $\dot{V}E$ and $PaCO_{2\ pre}$ during the first 70 sec of recovery in each subject (Figure 3). The correlation coefficients obtained for all subjects ranged from $r = 0.572$ to $r = 0.805$ ($p<0.05$).

Discussion

The subjects in the present study performed an impulse-like exercise with work intensity of 100 watts and duration of 20 sec. $PETCO_2$ and $PaCO_{2\ pre}$ were significantly higher than the rest values during recovery from 100 watts impulse-like exercise until 70 sec. $\dot{V}E$ was significantly higher than the rest value during recovery until 155 sec. There was a significant relationship between $PaCO_{2\ pre}$ and $\dot{V}E$ in each subject using the first 70 sec of recovery.

It has been reported that lactate is produced during a very short exercise with high intensity (Gaitanos et al. 1993) and that blood lactate level increases during recovery from this type of exercise and may stimulate peripheral chemoreceptors during this period (Clement et al. 1992, Clement et al. 1996). Another factor that is known to increase during exercise and recovery and to stimulate peripheral chemoreceptors and subsequent increase in $\dot{V}E$ response is K^+ (Paterson et al. 1989, Yoshida et al. 1990). The intensity of work used in the present study was too light and induced no metabolic acidosis and no change in arterialized blood La^-, pH, or K^+ during recovery. Thus, the elevated $\dot{V}E$ during recovery cannot be attributed to these factors. However, a significant change in $PaCO_{2\ pre}$ level was observed during exercise and the first 70 sec of recovery. The mechanism by which $PaCO_2$ ($PaCO_{2\ pre}$) increased during exercise and the first few seconds of recovery in this study is not clear. However, it has been reported that the slightly slower time course of $\dot{V}E$ than that of $\dot{V}CO_2$ may elicit a small transient rise in $PaCO_2$ (Haouzi et al. 2002, Whipp 1983). Another possible mechanism is that $\dot{V}E$ would have been insufficient for expiring CO_2 from the lungs that results in a reduction in RER. This would lead to an increase in body CO_2 stores and consequently increase in $PaCO_2$ level. $PaCO_2$ is known to be another important factor for stimulation of peripheral and central chemoreceptors, and these chemoreceptors are capable of modulating changes in $\dot{V}E$. Carotid bodies respond rapidly to hypercapnia (Eyzaquirre and Zapata, 1984), and central chemoreceptors would be stimulated more by hypercapnia than by acute metabolic acidosis of arterial blood because the blood-brain barrier is relatively impermeable to H^+ but is permeable to CO_2 (Clement et al. 1992). In the present study a significant, though not high, correlation coefficient was obtained in the relationship between $\dot{V}E$ and $PaCO_{2\ pre}$ during the first 70 sec of recovery in each subject. Furthermore, $\dot{V}E$ response at the starting point of recovery was not significantly different from $\dot{V}E$ response at the end point of exercise, while an abrupt decline in $\dot{V}E$ response is expected at

the end of exercise due to the disappearance of neural signals from mechanical receptors in working muscle (Turner 1991). These findings demonstrate that $\dot{V}E$ is mediated partly by $PaCO_2$ and that the stimulatory effect of $PaCO_2$ on $\dot{V}E$ in the first 70 sec of recovery may prevent the expected decline of $\dot{V}E$ at the start of recovery.

The other possible factor 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 (Eldridge et al. 1985). The time constant for after-discharge has been reported to range from 51 to 57 sec in anesthetized cats (Eldridge and Gill-kumar 1978, Eldridge and Gill-kumar 1980). Thus, it can be assumed that after-discharge is involved in $\dot{V}E$ control in the first 70 sec of recovery.

The results of this study showed that $\dot{V}E$ was still significantly higher than the rest value until 155 sec of recovery, of which time $PaCO_{2\,pre}$ had already recovered to the rest value. This result suggests that factors other than humoral factors mediate $\dot{V}E$ during this period. Our result is consistent with the results of a study performed by Clement et al. in 1996 in which they concluded that $\dot{V}E$ remains stimulated at 30 min after the end of exercise by processes other than post-exercise metabolic acidosis and likely by the central influence (Clement et al. 1996). Although we did not measure the levels of any neural factors in this study, it is possible that some of the neural factors that have been proposed in previous reports are involved in this elevated response of $\dot{V}E$. For example, Yamanaka et al. in 2011 suggested that ventilatory response during recovery after intense exercise is associated with effort sense indirectly elicited by central motor command (Yamanaka et al. 2011). Thin fiber afferents (i.e., groups III and IV) in working muscles that are thought to respond to mechanical and metabolic stimuli (Kaufman et al. 1983, McCloskey and Mitchell 1972) and also to respond to mechanical distension of the peripheral vascular network and change in the volume of blood in the venular system (Haouzi et al. 2001) have also been reported to be involved in $\dot{V}E$ response during recovery from exercise (Fukuba et

al. 2007, Haouzi et al. 1993). The results of the study performed by Fukuba et al in 2007 showed that femoral vascular occlusion significantly reduced $\dot{V}E$ response during recovery from either supra-AT or sub-AT exercise; however, the deficit was much larger during supra-AT than during sub-AT exercise. Based on those results, they concluded that metabolites do not play an important role in post-exercise $\dot{V}E$ through the intramuscular chemoreflex and rather mechanisms related to the hemodynamic effects of suddenly altered muscle perfusion seem more consistent with this phenomenon (Fukuba et al. 2007). Similarly, obstruction of blood flow to lower limbs reduced the normal $\dot{V}E$ response to impulse-like exercise, and the involvement of groups III and IV afferent fibers in $\dot{V}E$ control was proposed (Haouzi et al. 2001). Haouzi et al. in 2002, who investigated the effect of body position on $\dot{V}E$ response following an impulse exercise, speculated that the higher $\dot{V}E$ response in the upright (U) position than in the supine (S) position could be partly related to higher stimulation of thin muscle afferent fibers in the U position than in the S position, since the load imposed on venous return was much higher in the U position than in the S position (Haouzi et al. 2002).Therefore, the possibility that thin fiber afferents are involved in $\dot{V}E$ response during recovery from impulse-like exercise without metabolic acidosis exists and needs to be proved experimentally in future studies.

In conclusion, the results of the present study demonstrate that $\dot{V}E$ response during the first 70 sec of recovery after impulse-like exercise of 100 watts intensity is attributed partly to $PaCO_2$, and $\dot{V}E$ recovers to the rest value later than $PaCO_2$ at 155 sec of recovery time, with neural factors presumably driving $\dot{V}E$ in this period.

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Table 1. Mean values ± SD of blood ions and gases at rest and during recovery from 100 watts impulse-like exercise.

		Recovery		
	Rest	1 min	5 min	10 min
pH	7.41±0.02	7.40±0.02	7.41±0.02	7.39±0.01
PaO_2(mmHg)	86.1±5.9	87.3±6.8	89.9±4.2	86.3±4.1
$PaCO_2$(mmHg)	38.4±2.8	39.6±2.5	39.7±2.2	40.4±2.2
K^+ (mmol.l^{-1})	3.90±0.24	3.94±0.19	3.97±0.25	3.81±0.20
La^- (mmol.l^{-1})	1.00±0.21	1.12±0.23	1.00±0.18	0.99±0.18
HCO_3^-(mmol.l^{-1})	24.4±1.6	24.7±1.1	25.2±1.2	24.9±1.3

Values represent means ± SD (N= 6) for each time point. Arterialized blood pH, lactate (La^-), potassium (K^+), partial pressure of oxygen (PaO_2) and partial pressure of carbon dioxide ($PaCO_2$) were not significantly different from rest values during recovery ($p>0.05$)

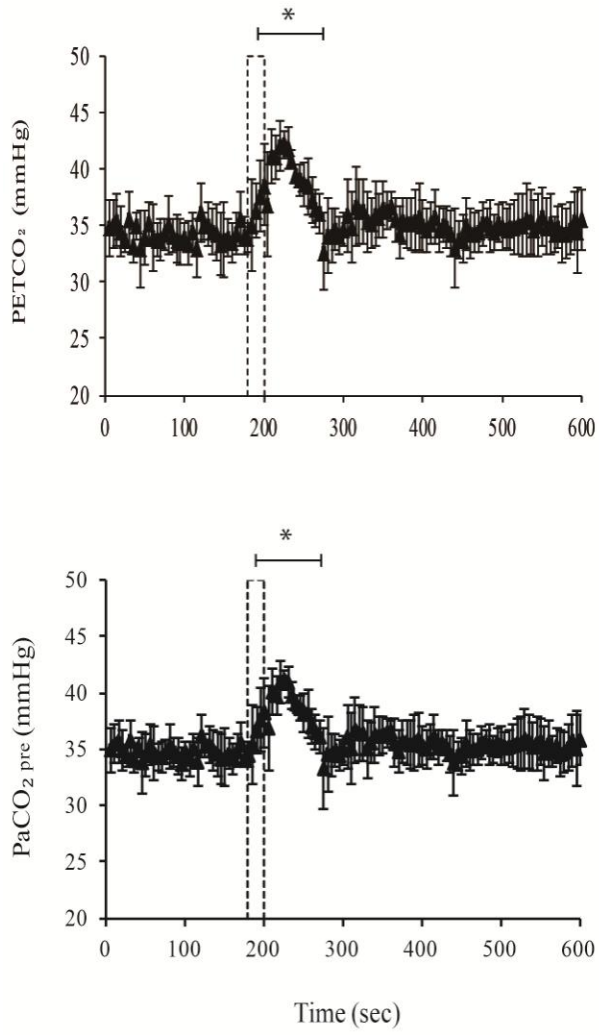

Fig. 1 Changes in end tidal CO_2 pressure ($PETCO_2$) (upper panel) and predicted arterial carbon dioxide ($PaCO_{2\ pre}$) (lower panel) during 100 watts impulse-like exercise and recovery from 100 watts impulse-like exercise. Vertical dashed line bar indicates exercise time. Data presented are means ± SD. *significantly different from rest value ($p<0.05$).

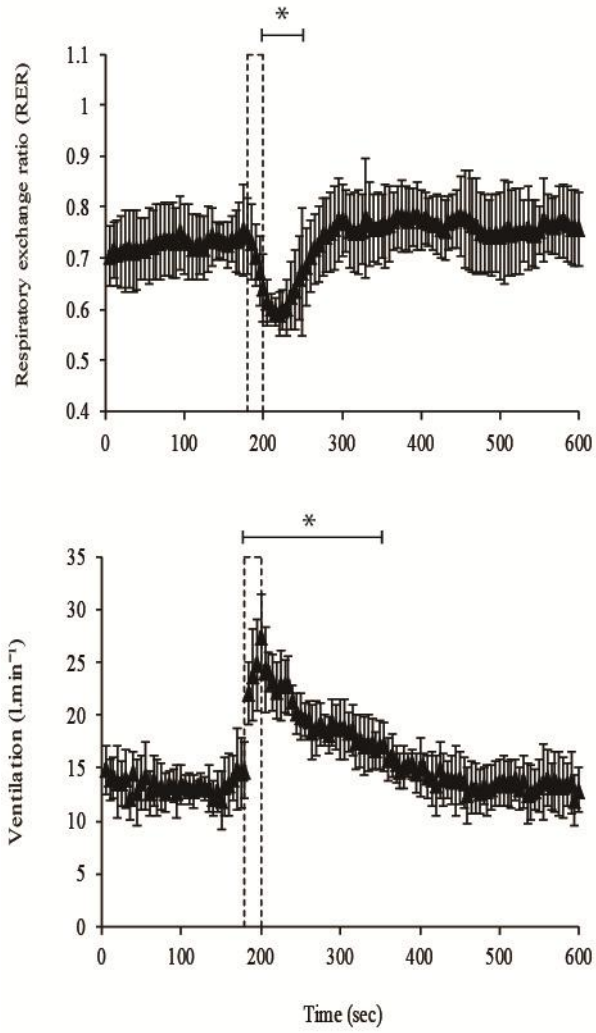

Fig. 2 Changes in respiratory exchange ratio (RER) (upper panel) and ventilation ($\dot{V}E$) (lower panel) during 100 watts impulse-like exercise and recovery from 100 watts impulse-like exercise. Vertical dashed line bar indicates exercise time. Data presented are means ± SD. *significantly different from rest value ($p<0.05$).

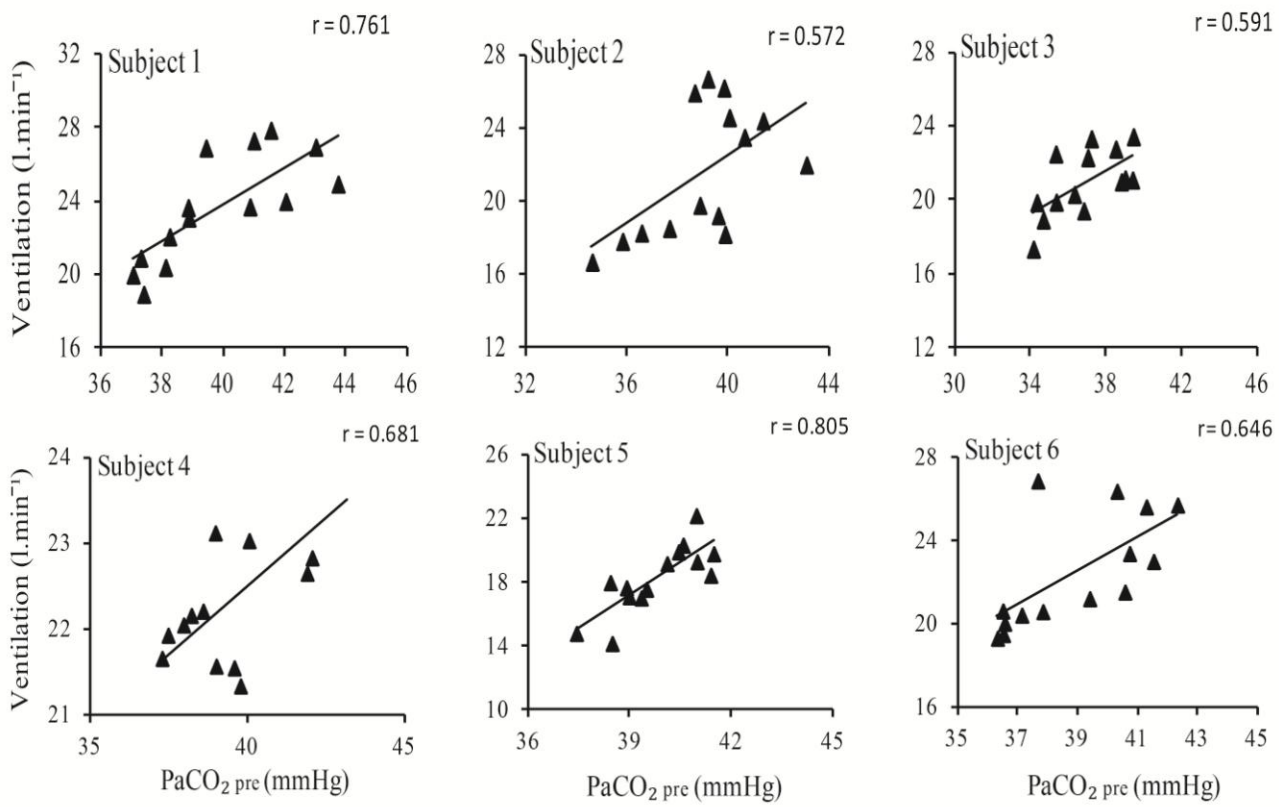

Fig. 3 Relationships between predicted arterial carbon dioxide ($PaCO_{2\ pre}$) and ventilation ($\dot{V}E$) during recovery from 100 watts impulse-like exercise in each subject ($r = 0.579 \sim r = 0.846$; $p<0.05$). Data presented are data for the first 70 sec of recovery in each subject.