Rapid Restitution of the Lung Functions Following Short-Term Breath-Holding During Moderate Intensity Cycling Exercise

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Abstract: Short periods of breath-holding (BH) occur frequently during various kinds of sports activities. Increased minute oxygen uptake (VO_2), increased minute carbon dioxide output (VCO_2) and increased minute ventilation (VE) are some of the typical responses just after breaking the BH and resuming breathing and these parameters return to their steady levels over time. This study examined the properties of the restitution processes of lung functions after BH. Exercise tests were carried out with a bicycle ergometer for seven healthy male athletes and the ventilation activities and expired gases were measured with a breath-by-breath based aeromonitor. The respiratory responses to 25 s BH were tested during continuous cycling at three exercise loads (2 W, 22 W and 102 W). A single exponential decay function with two parameters: initial amplitude (A) and time constant (τ), was fitted to the data of VO_2, VCO_2, VE, expiratory tidal volume (TVE) and respiratory rate (RR) after cessation of the BH. It was found that, except for RR, A was increased and τ was decreased with increasing the exercise intensity. The A of VCO_2 was smaller and τ was longer than those of VO_2 at all load intensities. The difference in the time courses of VO_2 and VCO_2 may be explained by the different buffer mechanisms of oxygen and carbon dioxide within the body.

INTRODUCTION

Breath-holding (BH) occurs during various physical activities. BH is an important element, not only in water sport activities, but also in dry-land sport activities, such as athletics (sprint running, jumping, throwing), contact sports (judo, kendo) and weight lifting, and many studies have been conducted for examining various aspects of BH [1-12]. A distinctive phenomenon during BH, which can be typically observed during face immersion with BH, is the dive reflex, characterized by peripheral vasoconstriction (i.e. decreased blood flow to the working muscles and other organs) with concomitant bradycardia. [for review, see 13]. These responses may result in a decrease in the oxygen uptake by the organs. Thus, the aim of the dive reflex may be oxygen conservation during BH [14, 15]. Once the BH is over, the body may try to restore the usual balance of gases within the body. In the lungs the carbon dioxide accumulated during BH in the body may be removed by consecutive expirations and the consumed oxygen may be replenished by consecutive inspirations. In order to get rid of the oxygen deficit and carbon dioxide overload, various respiratory and circulatory functions may be transiently accelerated. Compared with the processes that occur during BH, relatively little is known about the processes that occur during this restitution phase after BH. It would be interesting to compare the time courses of carbon dioxide output and oxygen uptake after BH under various degrees of exercise load and to investigate how their changes can be explained by characteristics of these gases and regulatory mechanisms of the body.

In this study we conducted an experiment using a cycle-ergometer with exercise loads of three levels (low, moderate and high), while monitoring various lung functions. We followed the time courses of minute oxygen uptake, minute carbon dioxide output and other lung functions after cessation of short-term BH and examined how they depended on the intensity of exercise load. In order to characterize these time courses, we fitted a single exponential decay curves to the lung functions and obtained the response amplitudes and the relaxation time constants.

MATERIALS AND METHODOLOGY

1. Subjects

Seven healthy male athletes took part in the study (two kendo athletes, two short distance swimmers, one soccer player, one sprinter, one volleyball player). None of them were members of national teams. All of them were non-smokers. Their average age and SD was 24.0 ± 2.9 years, height 174.0 ± 5.1 cm, weight 65.5 ± 10.6 kg, and vital capacity 4.27 ± 0.16 l. None of the subjects had experience of underwater free diving. They gave their written consent to participate in the study. All the procedures were approved by

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Kokushikan University Ethics Committee for Research on Human Subjects.

2. Study Protocol

Preparation for the exercise test: The subjects were instructed to come to the laboratory at least 2 hr after light meals, and without taking any caffeine-containing beverages. The room temperature was 21°C. The subjects were wearing a T-shirt and shorts. On the first day, the vital capacity was measured using a spirometer (Autospiro AS-300, Minato Medical Science, Japan) while the subjects were in a sitting position and wearing a nose clip. Five min stretching was done before starting the exercise test.

The exercise test: Each subject performed one exercise test per day. The subjects mounted on a cycle-ergometer (Rehor 500P, Groningen, The Netherlands) and breathed into a coupler while wearing the nose clip. Chest electrocardiograms were continuously monitored with an electrocardiograph (BSM-7201, Nihon Kohden, Japan). Exercise began as soon as a stable resting heart rate (HR) was obtained. Arterial hemoglobin oxygen saturation (SaO_2) was measured on the middle finger of the right hand using a finger pulse oximeter. The ventilation activities and expired gases were analyzed with a breath-by-breath based aeromonitor (AE 300S, Minato Medical Science, Japan). The subjects were requested to maintain a pedal cadence of 60 - 70 revolutions per min during the exercise test. Respiratory responses to BH of each subject were recorded during continuous cycling at three exercise loads (2 W, 22 W and 102 W) with one load on one day. The subjects were free to decide which intensity to select on which day. Warming-up of 2 W load was done in the first 7 min. In the next 3 min the load was increased linearly to one of the exercise loads, which was then maintained for 35 min. The subjects performed 4 sessions of 25 s BH, at the 20, 25, 30 and 35 min from the start of the test (Fig. **1**).

The start and the end of the BH were issued by the experiment leader. The subjects were instructed not to hyperventilate before BH. A BH started with a deep inspiration to a comfort level. Although we chose 25 s for the duration of BH as a tolerable period during exercise, the actual periods of BH were slightly shorter in average and differed at each loading: 24.4 ± 2.2 s (mean ± SD) at 2 W, 24.7 ± 1.9 s at 22 W, and 21.6 ± 0.9 s at 102 W. Responses to BHs shorter than 18 s or longer than 28 s were excluded from the study. (Two responses at 2 and 22 W were longer than 28 s, respectively; four responses at 102 W were shorter than 18 s).

3. Data Analysis

The analysis was divided into 2 steps: determination of the individual steady-state (SS) levels and determination of parameters of the restitution of lung functions to BH as relative values. In the latter, the initial amplitudes (A) of the restitution of the lung functions were first calculated, then single exponential decay curves were fitted on these responses and time constants (τ) of the restitution process were calculated.
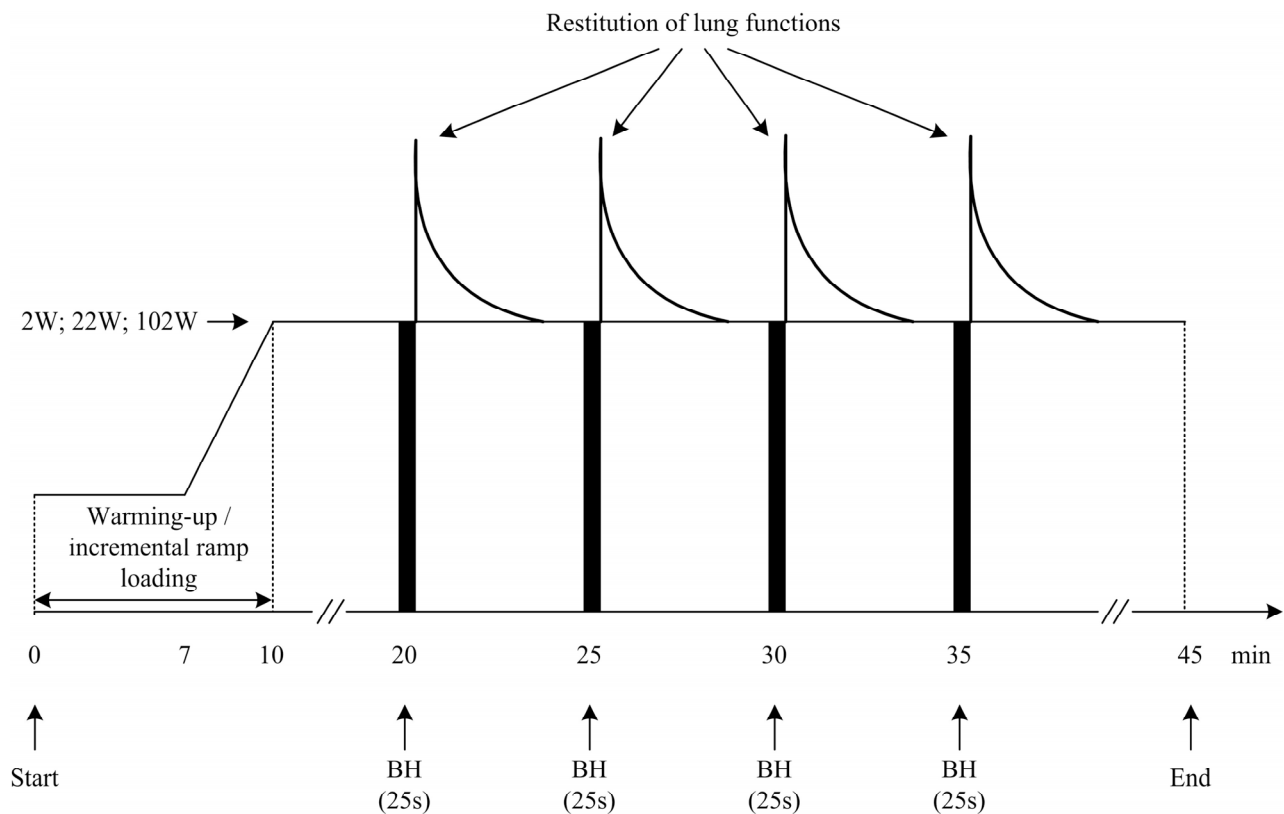
Fig. (1). Schematic illustration of the experimental protocol. Warming-up of 2 W load was done in the first 7 min. In the next 3 min the load was increased linearly to one of the exercise loads, which was then maintained for 35 min. The subjects performed 4 sessions of 25 s BH, at the 20, 25, 30 and 35 min from the start of the test.

Determination of the Steady-State Levels

The period just before the first BH was found to be appropriate for determining the steady state levels, because during the period the parameters examined were stable and variations were small. Thus, for each parameter examined, the average of the values of 30 consecutive expirations beginning at 15 min from the start of the test was calculated and used as the SS level. For each exercise load, the averages of the SS levels of various parameters for 7 subjects were calculated and are given in Table **1** (See Table **1** under Results).

Determination of the Restitution Process of the Lung Functions

Of the lung functions examined, the following five functions were selected for analyzing the restitution processes from BH: the minute oxygen uptake (VO_2), minute carbon dioxide output (VCO_2), expiratory tidal volume (TVE), respiratory rate (RR), minute ventilation (VE).

In each subject and each exercise load, the values of each lung function were first normalized by dividing them by the corresponding SS level. Then the normalized responses of 4 sessions to BHs were pooled by setting the time points of first breaths after the ends of BH as time zero and finally the responses of 7 subjects were pooled.

To obtain smoothed, averaged times courses of the data sets, a local quadratic polynomial regression model with Gaussian kernel was applied. This is a weighted average method with superior properties to simple moving average [16]. The value of the bandwidth was determined by AICc [17]. A single exponential model given below was also fitted to the data with a non-linear least squares method.

$$y(t) = A \times e^{-\frac{t}{\tau}} + 1 + error$$

where y(t) is the value of a normalized response function at time t; A is the initial amplitude and τ is the time constant of the exponential decay function. The curve fittings were performed using KyPlot 5.0 (KyensLab, Japan).

RESULTS

Table **1** gives the SS levels of 10 respiratory and cardiac function parameters at 2 W, 22 W and 102 W exercise loads. Means and SDs for 7 subjects are given.

VO_2, VCO_2 and VE changed markedly with the exercise load: they increased almost linearly with increasing the load

and the values at 102 W were 3 to 4 fold larger than those at 2 W. The values of TVE, RR and HR at 102 W were increased by about two-fold compared with those at 2 W. By contrast, no appreciable changes were found in ETO_2, $ETCO_2$, R, and SaO_2 with changes in the exercise load.

Of the respiratory function parameters examined, those showed large changes with the exercise load, i.e., VO_2, VCO_2, VE, TVE and RR, were selected and the changes after BH were examined. In Fig. (**2**), the time course of VO_2 (normalized by SS levels) in the restitution period after BH for each exercise load is shown. The data of all subjects were pooled and plotted.
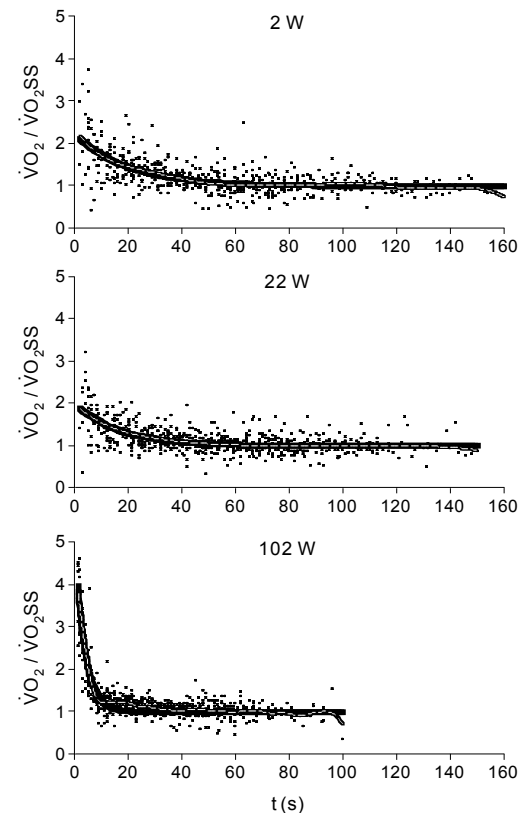
Fig. (2). Time courses of minute oxygen uptake relative to the steady-state level (VO_2/VO_2SS) in the restitution period after BH. Each point represents the average of 4 sessions of an exercise trial of a subject and the data of all subjects examined are pooled and plotted. Open dashed line: smoothing curve and solid line: exponential fit.

Table 1. **SS Levels of Various Parameters at 2 W, 22 W and 102 W Exercise Loads. Means ± SDs for 7 subjects. The Parentheses for the Data at 22 W and 102 W are the Ratios of the Means Compared with the Corresponding Values at 2 W**

	VO_2 (ml/min)	VCO_2 (ml/min)	VE (l/min)	TVE (ml)	RR (1/min)	ETO_2 (%)	$ETCO_2$ (%)	R	SaO_2 (%)	HR (1/min)
2 W	384.2 ±57.8	312.0 ±43.2	11.6 ±1.3	746.3 ±99.7	17.4 ±3.2	14.5 ±0.3	5.5 ±0.3	0.80 ±0.03	97.6 ±0.4	71.8 ±11.1
22 W	562.4 ±58.9 (1.46)	456.9 ±46.8 (1.46)	15.4 ±2.1 (1.33)	863.5 ±148.9 (1.16)	19.0 ±4.8 (1.09)	14.2 ±0.3 (0.98)	5.7 ±0.3 (1.04)	0.81 ±0.03 (1.01)	97.4 ±0.4 (1.00)	77.5 ±9.7 (1.08)
102 W	1541.2 ±66.8 (4.01)	1333.8 ±88.8 (4.28)	38.1 ±3.6 (3.28)	1570.8 ±233.7 (2.10)	25.4 ±5.1 (1.46)	14.1 ±0.3 (0.97)	6.0 ±0.2 (1.09)	0.87 ±0.03 (1.09)	96.7 ±0.5 (0.99)	133.7 ±2.5 (1.86)

Although the data were highly variable, at 2W exercise load, $\dot{V}O_2$ increased to about 2-fold of the SS level on average immediately after BH and returned to the SS level in about 100 s. The time course of $\dot{V}O_2$ at 22 W was similar to that at 2W, whereas at 102 W, the initial rise was much larger and the decay to the SS level was more rapid.

In order to estimate the average time courses, the data were numerically smoothed and were plotted as dashed curves in Fig. (**2**). At 2 W and 22 W, the curves were very smoothly and monotonically decreasing. At 102 W, after the initial steep decline the smoothed curved showed a small "hump" in the period about 10 to 30 s after cessation of BH.

A single exponential model was fitted to the data of $\dot{V}O_2$ and is shown as solid curves in Fig. (**2**). Except for the period of the hump at 102 W, the exponential fits were very close to the smoothed curves. The amplitudes (A) of the exponential model at each exercise load are given in Table **2** and the time constants (τ) in Table **3**.

Table 2. **Amplitudes (A) of Restitution Processes of 5 Lung Functions at 2 W, 22 W and 102 W Exercise Loads. A Single Exponential Decay Curve was Fitted to Pooled 20 Restitution Responses at 2 W; 23 Responses at 22 W; and 22 Responses at 102 W**

	2 W	22 W	102 W
$\dot{V}O_2$	1.22	0.99	3.91
$\dot{V}CO_2$	0.77	0.70	1.69
TVE	0.59	0.71	0.56
RR	-0.20	-0.17	0.49
VE	0.39	0.37	1.25

Table 3. **Time Constant (τ) of Restitution Processes of 5 Lung Functions at 2 W, 22 W and 102 W Exercise Loads. A Single Exponential Decay Curve was Fitted to Pooled 20 Restitution Responses at 2 W; 23 Responses at 22 W; and 22 Responses at 102 W**

	2 W	22 W	102 W
$\dot{V}O_2$	19.7	16.6	3.7
$\dot{V}CO_2$	35.2	27.6	7.2
TVE	23.0	21.5	8.4
RR	10.0	15.4	31.6
VE	47.9	36.6	13.9

The changes in $\dot{V}CO_2$ in the restitution period are shown in Fig. (**3**).

The time courses at 2 W and 22 W were similar to those of $\dot{V}O_2$ and the exponential fits were very close to the smoothed curves. At 102 W, the initial rise at the start of re-breathing was less pronounced than $\dot{V}O_2$ but the difference between the smoothed curve and exponential fit was large within the 10 s of the initial period. As in $\dot{V}O_2$, presence of a small hump was suggested by the smoothed curve in the period of about 10 to 30 s after cessation of BH.
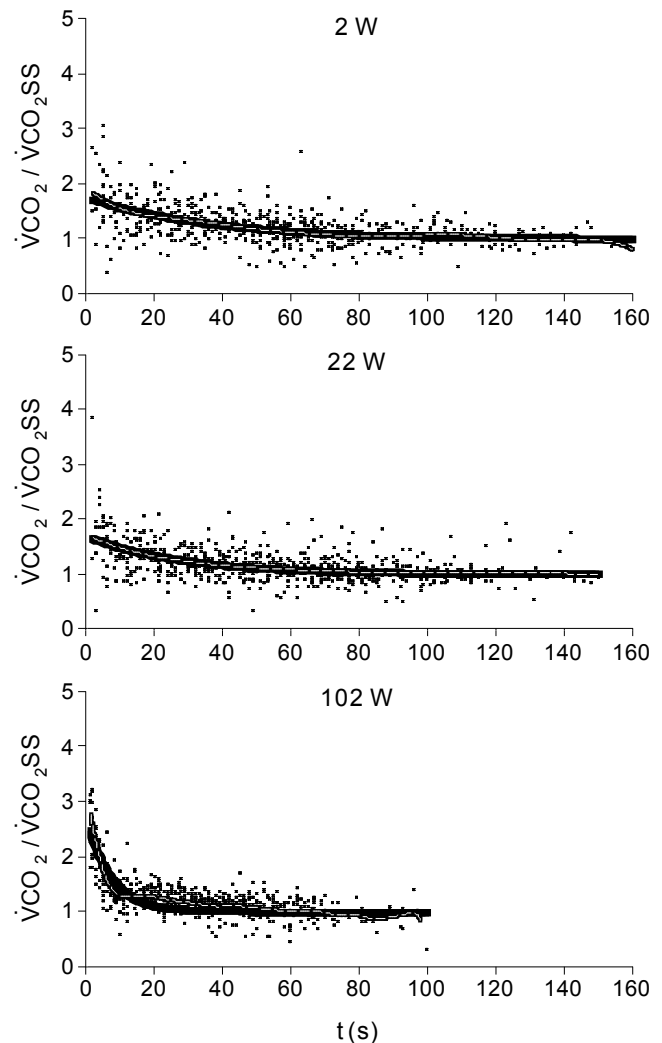
Fig. (3). Time courses of minute carbon dioxide output relative to the steady-state level ($\dot{V}CO_2/\dot{V}CO_2SS$) in the restitution period after BH. Other details are the same as in Fig. (**2**).

Fig. (**4**) gives the changes in TVE in the restitution period.

The time course at each exercise load was similar to $\dot{V}O_2$ and $\dot{V}CO_2$ and the smoothed curves and exponential fits were very close at all exercise loads.

In Fig. (**5**), the changes in RR are shown.

At 2 W and 22 W, no initial rise was observed and the values in the initial 50 s were slightly lower than the SS levels. This tendency was apparent in the smoothed curves and exponential fits and reflected in the negative values of the amplitudes (A) in Table **2**.

As shown in Fig. (**6**), the time courses of VE reflected those of TVE and RR, since it is given as the product of them.

At 102 W, a slight deviation from the exponential model was apparent in the initial period as in RR.

DISCUSSION

In this study, we investigated the restitution processes of various lung functions to short-term breath-holding during exercise. To our knowledge, this may be the first study to

examine the exercise intensity dependency of the rapidity in restitutions of lung functions to BH during dynamic leg exercise.

As given in Table **1**, increasing the exercise load resulted in marked increases of $\dot{V}O_2$, $\dot{V}CO_2$ and concomitant increases in respiratory and cardiac functions (VE, TVE and RR and HR). The fact that the even highest load (102 W) did not cause any appreciable changes in ETO_2, $ETCO_2$, R, and SaO_2 suggests that increased oxygen consumption and carbon oxide production due to increased exercise load up to this level were well compensated by regulatory mechanisms of the body.
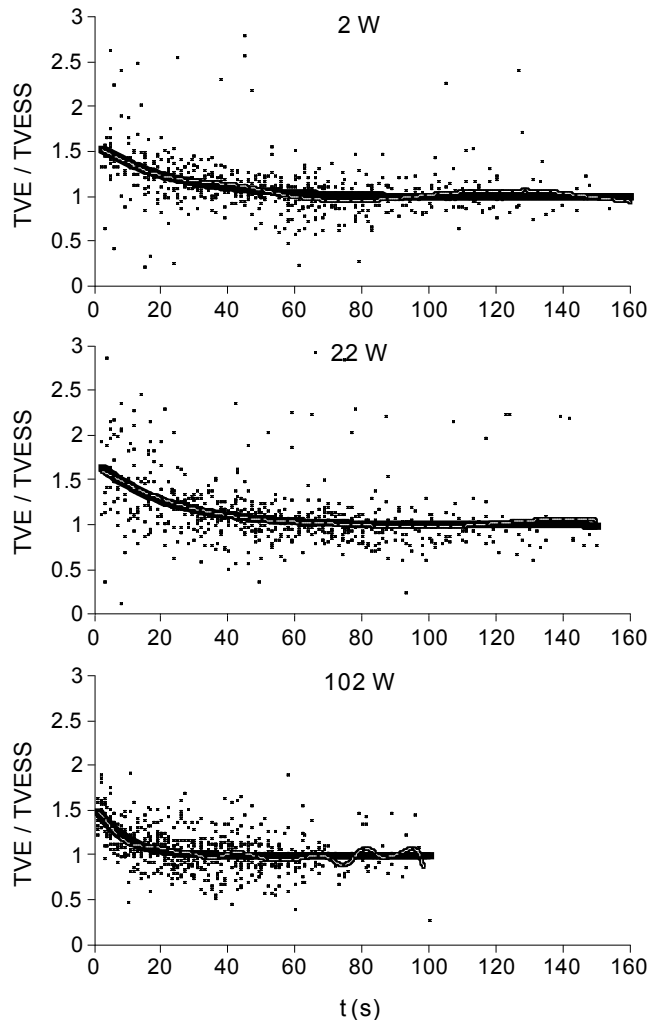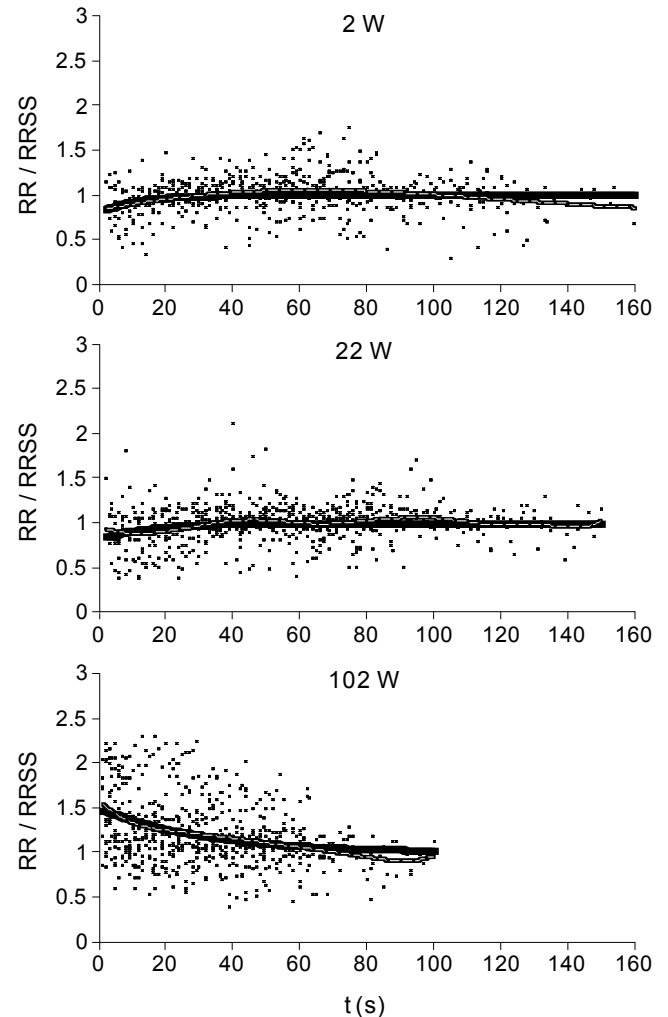

Fig. (4). Time courses of expiratory tidal volume relative to the steady-state level (TVE/TVESS) in the restitution period after BH. Other details are the same as in Fig. (**2**).

After cessation of 25 s BH, the values of $\dot{V}O_2$ and $\dot{V}CO_2$ rose promptly and then declined to the steady-state levels (Figs. **2**, **3**). Because they were considerably variable, a smoothing method was applied to the data. For both $\dot{V}O_2$ and $\dot{V}CO_2$, it gave very smooth and monotonically decreasing curves at low and moderate exercise loads, whereas at 102 W, small humps following steep initial rises were apparent.

In order to obtain numerical characteristics of the restitution processes of lung functions, a single exponential model was fitted to the data and the initial amplitudes and time constants were estimated. For $\dot{V}O_2$, except for the period of the hump at 102 W, the exponential fits were very close to the smoothed curves, suggesting that the single exponential model can sufficiently explain the restitution process of $\dot{V}O_2$. Similar results were obtained for $\dot{V}CO_2$, except that at 102 W, the discrepancy between the smoothed curve and exponential fit was large in the initial period. Thus, except for this initial phase of $\dot{V}CO_2$ and the periods of the humps for $\dot{V}O_2$ and $\dot{V}CO_2$ at 102 W, reasonable fitness of this model was obtained to the restitution data.

Fig. (5). Time courses of respiratory rate relative to the steady-state level (RR/RRSS) in the restitution period after BH. Other details are the same as in Fig. (**2**).

Increasing the exercise load may cause more intense oxygen deficit and carbon dioxide overload during BH and this may be reflected in the larger initial amplitudes of $\dot{V}O_2$ and $\dot{V}CO_2$ by increasing the load (Table **2**). Interestingly, the time constants became smaller with the increase in the load (Table **3**), suggesting that the regulatory mechanisms to restore usual balance of gases within the body were accelerated with the increase in the load. Accordingly, transient increases in respiratory functions, i.e., TVE, RR and VE after BH were observed (Figs. **4-6**), but there were differences in the load intensity dependency among the three functions. Thus, in TVE, the initial increases were observed but not changed by increasing the load, with an increase in

the decay at 102 W; in RR the initial increase was observed only at 102 W. Since VE is given as the product of TVE and RR, it can be seen that the initial increases at 2 W and 22 W were due to those in TVE and those of both TVE and RR contributed to the increase at 102 W. Another factor that may also contribute to the accelerated recovery of O_2 and CO_2 levels with the load intensity after BH, is circulatory regulatory mechanisms. The relative contributions of respiratory and circulatory mechanisms remain to be clarified.
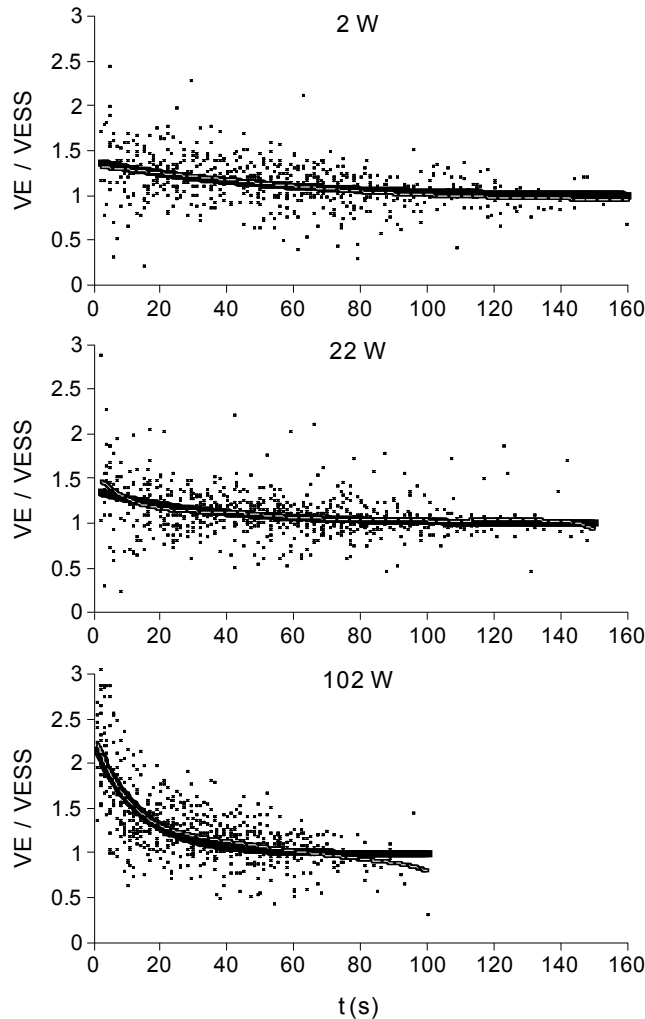
Fig. (6). Time courses of minute ventilation relative to the steady-state level (VE/VESS) in the restitution period after BH. Other details are the same as in Fig. (**2**).

Although the overall time courses of $\dot{V}O_2$ and $\dot{V}CO_2$ after BH were similar, they were not identical: the initial amplitudes of $\dot{V}CO_2$ were smaller and the time constants were longer than those of $\dot{V}O_2$ (Tables **2** and **3**) at all load intensities. The difference in the time courses of $\dot{V}O_2$ and $\dot{V}CO_2$ may be explained by the different buffer mechanisms of oxygen and carbon dioxide within the body. The chemical reaction kinetics for CO_2 is complex. CO_2 is produced in the peripheral tissues and is buffered in the interstitial space and in the blood. During BH, CO_2 may accumulate in these compartments and take time for the blood to transport it to the lungs [10, 18, 19]. This may introduce a secondary, slow component to the $\dot{V}CO_2$ recovery, thus leading to a longer

recovery time. The amount of oxygen deficit, on the other hand, may be much less than the amount of carbon dioxide overload. In order to restore the steady-state level of $\dot{V}O_2$, it may be sufficient to restore the usual balance inside the lungs. The changes of arterial hemoglobin oxygen saturation (SaO_2) during the BH could slow down the restitution of $\dot{V}O_2$, introducing a slow component, but in this study the duration of BH was rather short and the steady level of SaO_2 did not change with the load intensity. Therefore, the amount of SaO_2 decrease presumably did not reach the level where it could have affected the O_2 restitution.

CONCLUSION

This study showed that increasing the exercise intensity led to faster restitution of $\dot{V}O_2$ and $\dot{V}CO_2$ in response to short-term BH during a constant load cycling exercise. Further studies are requited to elucidate the respiratory and circulatory mechanisms that contributed to the accelerated recovery of respiratory gases. It would also be worthwhile to investigate the characteristics of the restitution of the lung functions to BH in athletes at various fitness levels and in various sports.

AUTHORS` CONTRIBUTION

NH: study design, data collection, data analysis, data interpretation, literature search.

SH: study design, data collection, data analysis, data interpretation.

KM: study design, data collection, data analysis.

KI: data interpretation.

IS: study design, data interpretation.

YK: data analysis, data interpretation.

PG: data interpretation.

ABBREVIATIONS

BH	=	Breath-holding
SS	=	Steady-state
τ	=	Time constant.
A	=	Amplitude
$\dot{V}O_2$	=	Minute oxygen uptake
$\dot{V}CO_2$	=	Minute carbon dioxide output
TVE	=	Expiratory tidal volume
RR	=	Respiratory rate
VE	=	Minute ventilation
$\dot{V}O_2/\dot{V}O_2SS$	=	Minute oxygen uptake relative to the steady-state level
$\dot{V}CO_2/\dot{V}CO_2SS$	=	Minute carbon dioxide output relative to the steady-state level

TVE/TVESS	=	Expiratory tidal volume relative to the steady-state level
RR/RRSS	=	Respiratory rate relative to the steady-state level
VE/VESS	=	Minute ventilation relative to the steady-state level
R	=	Respiratory quotient
ETO_2	=	End-tidal oxygen concentration
$ETCO_2$	=	End-tidal carbon dioxide concentration
HR	=	Heart rate
SaO_2	=	Arterial hemoglobin oxygen saturation

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