Respiratory Responses to Passive and Active Recovery from Exercise

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Abstract: To investigate the effect of the neural components associated with leg movements on the control of ventilation during recovery from exercise, we recorded the minute ventilation ($\dot{V}_E$), oxygen uptake ($\dot{V}_{O_2}$), and carbon dioxide output ($\dot{V}_{CO_2}$) of eight normal volunteers during recovery from moderate, steady-state cycle exercise (170 W). The recovery phases were undergone separately under two different conditions: 5 min of rest (passive recovery) on a bicycle ergometer and 3 min of pedaling at a work rate of 0 W (active recovery) followed by 2 min of rest. The phase-1 responses were observed in all the variables studied at the transition of passive recovery but not in the active recovery phase. The kinetics of $\dot{V}_{CO_2}$ during the off-

transition were significantly faster than those of $\dot{V}_E$ in both recoveries, indicating that the decreases in $\dot{V}_{CO_2}$ could precede the decreases in $\dot{V}_E$. Although the levels of $\dot{V}_E$ and $\dot{V}_{CO_2}$ during active recovery were significantly higher than those during passive recovery, the decline in $\dot{V}_E$ was closely proportional to that of $\dot{V}_{CO_2}$ under both recovery conditions, with resultant indications of similar $\dot{V}_E$ - $\dot{V}_{CO_2}$ regression lines. These findings suggest that the flux of CO_2 to the lungs is an important determinant of ventilatory drive during recovery, and that neither central command nor neural afferents from contracting muscles are requisite for the control of ventilation during recovery from exercise. [Japanese Journal of Physiology, 47, 59–65, 1997]

Key words: exercise recovery, neural components, $\dot{V}_E$ - $\dot{V}_{CO_2}$ coupling, control of ventilation, phase-1 response.

The electrical stimulation of efferents to the muscles produces increases in cardiovascular and respiratory responses with muscular contractions [1–4]. However, it is difficult to eliminate non-invasively the effect of neural afferents originating in contracting muscles on the cardiorespiratory response during exercise in healthy human subjects [4]. An immediate decrease in minute ventilation ($\dot{V}_E$) during the circulatory occlusion of the legs during the postexercise resting stage suggests that few or no muscle chemoreceptors activating neural afferent input to the respiratory center are involved, while the receptors have a major effect on mean arterial blood pressure [5, 6]. Thus, the effect of muscular chemoreflexes on the control of the respiratory system may be negligible, at least during the recovery of exercise hyperpnea. It is generally believed

that the neural drives of both central command and muscular mechanoreflexes are eliminated and/or reduced during stationary rest [5–7]. In this study, the recovery phase of exercise was examined under two conditions, with and without leg movement. Our hypothesis was that, if the neural components of both central command and muscular mechanoreflexes play a dominant role in controlling breathing, recovery with leg movement (active recovery) would exert a more facilitatory effect on the respiratory response than recovery without leg movement (passive recovery). We tested this hypothesis in this study.

METHODS

Subjects. Eight healthy young male subjects,

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aged 23.1 ± 2.4 year (mean $\pm$ SD), with height of 171.6 ± 4.7 cm, and weight of 63.0 ± 6.9 kg, volunteered for this study after their informed consent was obtained. No trained athletes were included in the subject group. Each subject was familiarized with the experimental procedure before data were collected.

Measurements of ventilatory and respiratory variables. The measurements of minute expiratory ventilation ($\dot{V}_E$), tidal volume (V_T), respiratory frequency (f), end-tidal pressures of O_2 and CO_2 ($P_{ET_{O_2}}$ and $P_{ET_{CO_2}}$), O_2 uptake ($\dot{V}_{O_2}$), CO_2 output ($\dot{V}_{CO_2}$), and the gas-exchange ratio ($R = \dot{V}_{CO_2} / \dot{V}_{O_2}$) were obtained on a breath-by-breath basis using an on-line automated system [8, 9]. Ventilatory airflow was monitored with a hot-wire-type pneumotachograph (RF-2, Minato). The composition of expired gas was continuously analyzed with a medical mass spectrometer (WSMR-1400, Westron). The mass spectrometer was calibrated with a standard reference gas mixture before each study. All data for respiratory variables were stored on diskettes for subsequent analysis by a personal computer (PC-98, NEC).

Experimental protocol. Each subject was required to exercise in the upright position on an electromagnetically-braked cycle ergometer (Corival 300, Lode). The braking system of the ergometer was externally controlled to ensure the desired work output [10]. During exercise and subsequent pedaling recovery periods, the subjects pedaled at a constant rate of 60 rpm paced by a metronome. The pedaling rate was monitored using an inductive revolution meter fitted to the ergometer.

Each subject completed two repetitions of the test under each of the two experimental conditions. Figure 1 outlines the experimental protocols. The subjects rested for 2 min in an upright, seated position on the cycle ergometer, followed by 5 min of exercise at a work rate of 170 W. The recovery from exercise was

performed separately under two different conditions: 1) rest for 5 min (passive recovery) on the ergometer and 2) pedaling at a work rate of 0 W for 3 min (active recovery) followed by complete rest for 2 min on the ergometer. Each subject placed his feet on the footrest near the flywheel during the rest period. The 170 W work rate was chosen because it could be easily continued by all of the subjects, and because we believed that it was intense enough to activate the known neural and humoral components related to exercise hyperpnea. Although the anaerobic threshold (AT) levels of the subjects had not been determined, the absolute work intensity of 170 W was considered to be the maximum aerobic exercise level or to slightly exceed the AT level for most subjects because the mean R during exercise was apparently around unity. All experiments were conducted at a room temperature of between 21 and 23°C.

Data analysis. Breath-by-breath data from two repetitions under each test condition for each subject were separately rearranged with a 5-s interval time base using the Lagrange interpolation, and these data were then averaged to yield a single dataset per subject. Group mean values were obtained from the eight individual single-averaged datasets. Steady-state values for rest and exercise were determined from the last minute of averaged data. Mean values for recovery were also determined at 10, 20, and 300 s. All values are expressed as mean $\pm$ SD.

To characterize the kinetic behaviors of $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, and $\dot{V}_E$ for the off-transients during passive recovery, the averaged response data of each subject were fitted by a first-order exponential function with a time constant and no pure time delay. Curve fittings were performed using a least-squares method. The Wilcoxon signed-ranks test was used to detect statistically significant differences among the time constants.

A two-way repeated measurement analysis of variance was used to compare the pairs of responses during passive recovery and during active recovery for the first 3 min. Differences in $\dot{V}_E$, V_T , f , $P_{ET_{O_2}}$, $P_{ET_{CO_2}}$, $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, and R between the passive and active recovery experiments were also examined by analysis of variance. The mean values for all test variables at 10 and 20 s under each of the two recovery conditions were compared to the corresponding steady-state values during exercise. Differences were considered significant for all statistical analyses at $p < 0.05$.

RESULTS

Figure 2 presents the temporal patterns of $\dot{V}_E$, $\dot{V}_{O_2}$, and $\dot{V}_{CO_2}$ responses during passive recovery and active re-

Fig. 1. Schematic representation of exercise protocols. Exercise in each protocol was performed for 5 min at a work rate of 170 W after 2 min of rest. The subsequent recovery phases consisted of 1) rest on the ergometer for 300 s (passive recovery) or 2) unloaded pedaling for 180 s (active recovery) followed by rest for 120 s. These were undergone separately.

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Fig. 2. Group mean values for expiratory minute ventilation ($\dot{V}_E$), O_2 uptake ($\dot{V}_{O_2}$), and CO_2 output ($\dot{V}_{CO_2}$) in passive recovery (rest, ○), active recovery (unloaded pedaling, ●), and subsequent rest (■). The vertical dashed line (at $t=0$) indicates the time of termination of exercise at the work rate of 170 W, and the vertical dotted line the termination of unloaded pedaling during active recovery.

covery followed by rest. The $\dot{V}_E$ and gas-exchange variables decreased exponentially during both recovery conditions. However, the values were significantly lower for passive recovery as compared to active recovery ($p < 0.001$). A typical phase-1 response consisting of an initial rapid fall in the first 10 s followed by a transient rise in the next 20 s could be identified in the time course of $\dot{V}_E$, $\dot{V}_{O_2}$, and $\dot{V}_{CO_2}$ during passive recovery in all of the subjects. In contrast, there was

no initial fluctuation in the respiratory variables during the transition from exercise to light exercise during active recovery, or from light exercise to rest after active recovery. Despite the difference between the recovery conditions in the early response during the first 20 s, the rates of decline during the subsequent recovery periods of 40–180 s were similar, as shown by the parallel falling curves. On average, the phase-1 responses observed during passive recovery accounted

Table 1. Respiratory variables before, during, and after exercise.

	Rest	Exercise	Passive recovery			Rest	Exercise	Active recovery		
			10 s	20 s	300 s			10 s	20 s	300 s
$\dot{V}_E$ (l/min)	8.61 ± 1.77	42.13 ± 6.81	32.78* ± 8.27	34.04* ± 7.05	12.28 ± 1.96	9.10 ± 1.41	43.94 ± 5.93	41.95† ± 7.77	38.35 ± 7.2	12.38 ± 1.85
V_T (l)	0.72 ± 0.23	2.08 ± 0.62	1.90 ± 0.52	1.82 ± 0.51	0.84 ± 0.23	0.73 ± 0.31	2.06 ± 0.54	2.04 ± 0.54	2.09 ± 0.72	0.86 ± 0.43
f (breaths/min)	12.99 ± 3.98	22.07 ± 7.43	18.53 ± 6.02	19.59 ± 5.09	15.33 ± 4.00	13.90 ± 3.96	22.96 ± 6.73	21.87 ± 5.42	20.23 ± 6.92	16.06 ± 3.92
$P_{ET_{O_2}}$ (mmHg)	104.2 ± 3.4	95.4 ± 6.9	98.6 ± 5.8	98.7 ± 4.2	110.3 ± 3.6	104.4 ± 3.4	97.0 ± 4.8	99.1 ± 4.6	99.5 ± 4.2	108.8 ± 5.9
$P_{ET_{CO_2}}$ (mmHg)	35.7 ± 1.9	48.1 ± 3.6	46.2 ± 3.3	47.4 ± 3.7	35.0 ± 1.9	35.9 ± 2.9	47.4 ± 2.9	46.2 ± 2.7	46.7 ± 2.2	35.0 ± 3.2
$\dot{V}_{O_2}$ (l/min)	0.25 ± 0.05	1.69 ± 0.26	1.24* ± 0.38	1.28** ± 0.27	0.30 ± 0.07	0.26 ± 0.06	1.71 ± 0.18	1.56 ± 0.28	1.42* ± 0.26	0.30 ± 0.09
$\dot{V}_{CO_2}$ (l/min)	0.19 ± 0.04	1.57 ± 0.21	1.17* ± 0.35	1.26* ± 0.28	0.28 ± 0.07	0.21 ± 0.04	1.62 ± 0.18	1.51† ± 0.28	1.40 ± 0.26	0.27 ± 0.07
R	0.79 ± 0.04	0.93 ± 0.08	0.95 ± 0.09	0.98 ± 0.07	0.93 ± 0.13	0.80 ± 0.06	0.95 ± 0.11	0.98 ± 0.10	0.99 ± 0.12	0.90 ± 0.12

Values are mean $\pm$ SD for 16 trials of eight subjects. $\dot{V}_E$, minute expiratory ventilation; V_T , tidal volume; f , respiratory frequency; $P_{ET_{O_2}}$, end-tidal P_{O_2} ; $P_{ET_{CO_2}}$, end-tidal P_{CO_2} ; $\dot{V}_{O_2}$, O_2 uptake; $\dot{V}_{CO_2}$, CO_2 output; R , gas exchange ratio. * and ** represent significant differences at $p < 0.05$ and 0.005, respectively, from the corresponding exercise values. † $p < 0.05$, significantly different from passive recovery value.

for $42.5 \pm 17.4\%$ of the total $\dot{V}_E$ decrement from the exercising to resting steady-state level, $41.6 \pm 9.3\%$ of the $\dot{V}_{O_2}$ decrement, and $39.7 \pm 11.9\%$ of the $\dot{V}_{CO_2}$ decrement. $P_{ET_{O_2}}$, $P_{ET_{CO_2}}$, and R were relatively stable during the first 20 s of both passive and active recovery (Table 1).

The off-transient kinetics of $\dot{V}_E$, $\dot{V}_{O_2}$, and $\dot{V}_{CO_2}$ during passive recovery were analyzed in each subject by fitting actual responses to a first-order exponential function with a time constant and no pure time delay. The time constants for $\dot{V}_E$, $\dot{V}_{O_2}$, and $\dot{V}_{CO_2}$ were 67 ± 17.3 , 44.2 ± 4.8 , and 59.3 ± 10.4 s, respectively. The difference between $\dot{V}_E$ and $\dot{V}_{CO_2}$ was statistically significant ($p < 0.05$, Wilcoxon signed-ranks test). The time constants for these variables during active recovery could not be determined by the curve-fitting technique because these variables did not attain steady states within the first 3 min of the recovery period. To compare the kinetics of $\dot{V}_{CO_2}$ and $\dot{V}_E$ during the off-transients of passive and active recoveries, time courses of consecutive 10-s averages of these variables were normalized by the differences between the two averaged values during the last 30 s before and during stimulus

Fig. 3. Mean $\pm$ SD values for normalized minute ventilation ($\dot{V}_E$) and CO_2 output ($\dot{V}_{CO_2}$) for passive recovery (upper panel) and active recovery (lower panel). Each point represents an average for data acquired for 10 s. All data are expressed as the % difference from that (100%) at time 0.

exercise (Fig. 3). The time courses of the normalized $\dot{V}_{\text{CO}_2}$ and $\dot{V}_E$ decreased exponentially under both recovery conditions. The $\dot{V}_{\text{CO}_2}$ was apparently lower than the corresponding $\dot{V}_E$, indicating that the off-transient kinetics of $\dot{V}_{\text{CO}_2}$ were also significantly faster than those of $\dot{V}_E$ (passive recovery, $p < 0.05$; active recovery, $p < 0.001$).

Figure 4 presents the relationship between $\dot{V}_E$ and $\dot{V}_{\text{CO}_2}$ obtained during the entire time courses of passive and active recovery followed by rest. The data is based on the group mean values. The degree of fit of these data to each of the regression lines indicates the consistency of the $\dot{V}_E$ - $\dot{V}_{\text{CO}_2}$ relationship under both recovery conditions. A summary of the regression analyses for each subject is presented in Table 2. A significant linear relationship between $\dot{V}_E$ and $\dot{V}_{\text{CO}_2}$ was found for each subject during both recovery conditions ($p < 0.001$). The mean slope of 22.96 during passive

recovery was not significantly different from the mean slope of 23.34 during active recovery and subsequent rest. Similarly, there was no difference in the intercepts between the two recovery conditions. Thus, the relationship between $\dot{V}_E$ and $\dot{V}_{\text{CO}_2}$ was unaffected by leg movement during recovery. The linear relationship between $\dot{V}_E$ and $\dot{V}_{\text{CO}_2}$ was almost unaffected by the phase-1 response during passive recovery.

DISCUSSION

Previous studies have demonstrated that the initial ventilatory response (phase-1) during the first 10–20 s after the onset of exercise is more rapid than the subsequent slower change [9, 11–14]. Similarly, our study demonstrates that the phase-1 responses of $\dot{V}_E$, $\dot{V}_{\text{O}_2}$, and $\dot{V}_{\text{CO}_2}$ following the cessation of constant-load work are readily distinguishable from the subsequent expo-

Fig. 4. Group mean values for $\dot{V}_E$ as a function of $\dot{V}_{\text{CO}_2}$ for passive recovery (rest, ○), active recovery (unloaded pedaling, ●), and subsequent rest (■). Each data point for $\dot{V}_E$ and $\dot{V}_{\text{CO}_2}$ is obtained from Fig. 2. The data points included in the phase-1 response during passive recovery are indicated by arrows with their appearance time. The solid line and dashed line represent the respective regression lines of the experimental data for passive recovery and active recovery with subsequent rest.

Table 2. Summary of regression analyses for $\dot{V}_E$ - $\dot{V}_{\text{CO}_2}$ relationships for each subject during passive and active recovery.

Subj	Passive recovery						Active recovery					
	Slope	Intercept	<i>r</i>	<i>n</i>	$\Delta\dot{V}_E$	$\Delta\dot{V}_{\text{CO}_2}$	Slope	Intercept	<i>r</i>	<i>n</i>	$\Delta\dot{V}_E$	$\Delta\dot{V}_{\text{CO}_2}$
1	21.65	9.3	0.990	67	33.2	1.38	23.84	8.1	0.991	67	36.6	1.53
2	19.30	4.6	0.990	67	25.7	1.26	19.99	7.8	0.985	67	23.9	1.19
3	22.02	6.7	0.981	67	49.1	2.21	22.61	6.3	0.989	67	47.3	1.98
4	25.55	6.1	0.994	67	35.4	1.33	24.41	6.6	0.990	67	36.8	1.43
5	23.16	7.4	0.995	67	37.9	1.54	24.63	6.1	0.990	67	41.1	1.73
6	27.48	4.6	0.995	67	59.2	2.05	25.50	4.7	0.993	67	46.6	1.99
7	26.23	7.3	0.996	67	35.7	1.33	26.82	7.8	0.990	67	39.9	1.46
8	18.29	7.7	0.989	67	25.3	1.38	18.92	8.4	0.985	67	29.8	1.44
Mean	22.96	6.7			37.69	1.56	23.34	7.0			37.75	1.59
±SD	±3.29	±1.6			±11.46	±0.36	±2.70	±1.3			±7.95	±0.28

Regression coefficients were calculated from a simple linear regression equation ($Y = AX + B$); where Y is ventilation ($\dot{V}_E$, l/min), A is the slope of the regression line, X is CO_2 output ($\dot{V}_{\text{CO}_2}$, l/min), and B is the y -intercept of the regression line. r , correlation coefficient; n , No. of $\dot{V}_E$ - $\dot{V}_{\text{CO}_2}$ determinations. $\Delta\dot{V}_E$ and $\Delta\dot{V}_{\text{CO}_2}$ represent the range of $\dot{V}_E$ and $\dot{V}_{\text{CO}_2}$ values over which n determinations were made.

ventilatory decrease to steady-state rest. Since $\dot{V}_T$ decreased slowly during recovery, the rapid drop in $\dot{V}_E$ is likely due to an equally rapid drop in f . The characteristics of the $\dot{V}_E$ phase-1 response are in agreement with those reported by Linnarsson [11]. On the contrary, we detected no phase-1 response during active recovery with continuous alternating limb movements after exercise. It has been generally recognized that phase-1 response is mainly mediated by some neural components which are activated during the transition from rest to exercise [15]. The phase-1 response at the start of exercise is only typical of rest-to-work transitions; however, there is only a small or no phase-1 component when exercise is introduced from or followed by a base-line exercise of mild intensity, such as unloaded cycling [12]. The lack of phase-1 response during the transition from exercise to no-resistance pedaling in active recovery would support the view prevailing in literature [15].

The mechanism underlying postexercise hyperpnea, as seen in this study, has not been fully clarified. Recently, an increase in arterial blood potassium concentration ($[K^+]_a$) has been implicated for the increase in $\dot{V}_E$ during exercise and after exercise, in which increased $[K^+]_a$ has been evidenced to stimulate the peripheral chemoreceptors [16]. This mechanism may be involved in the persistence of increased $\dot{V}_E$ after exercise in our subjects. However, some previous studies have shown a divergence in the falling patterns of $\dot{V}_E$ and $[K^+]_a$ after exercise [16, 17]. In addition, a discrepancy between the rates of lactate removal and $\dot{V}_E$ reduction has also been reported after the end of heavy exercise (i.e., the highest lactate concentration in arterial blood occurred 1–2 min after heavy exercise had ceased) [18, 19]. Therefore, it seems unlikely that changes in $[K^+]_a$ and lactate-related blood $[H^+]$ would be significant determinants of ventilatory drive, at least during recovery from exercise in humans [6, 20].

Several studies have demonstrated that there is a close relationship between the dynamic responses of $\dot{V}_E$ and $\dot{V}_{CO_2}$ to various patterns of work load changes [21, 22]. In this study, tight coupling between $\dot{V}_E$ and $\dot{V}_{CO_2}$ was observed during the recovery phase, this being in agreement with a previous study [20]. The $\dot{V}_E$ - $\dot{V}_{CO_2}$ relationship was also virtually identical in the passive and active recovery conditions although the time courses of the two variables were different. Brice *et al.* [3] compared the $\dot{V}_E$ - $\dot{V}_{CO_2}$ relationship during voluntary leg exercise with that during electrically-induced exercise and found similar values of the slope in both exercise conditions (31.7 vs. 32.9). Their slope values were somewhat higher than those in our study. Brice *et al.* [4] also studied the $\dot{V}_E$ - $\dot{V}_{CO_2}$ relationship

during electrically-induced exercise in paraplegic subjects and observed a similar relationship to that in fully ambulatory human subjects, suggesting no role of either the peripheral neural afferents from the contracting muscles or the central command in ventilatory response during exercise. Haouzi *et al.* [6] demonstrated that postexercise hyperpnea after the release of cuffs occluding limb circulation for 2 min following exercise is augmented by increased muscle blood flow and a resultant increase in CO_2 flow to the lungs. Their results suggest the exclusion of a possible role of intramuscular chemoreceptors in the control of ventilation during recovery because the rate of decline of $\dot{V}_E$ was enhanced in spite of the high concentration of metabolic products trapped within the muscle during circulatory occlusion. Any change in alveolar P_{CO_2} with changes in ventilation must result in rapid dissociation of CO_2 from pulmonary capillary blood to alveolar gas due to enzyme carbonic anhydrase, even in a small gradient of P_{CO_2} between the blood and the alveoli. This situation would inevitably cause tight coupling between $\dot{V}_E$ and $\dot{V}_{CO_2}$. In this case, changes in $\dot{V}_E$ would precede those of $\dot{V}_{CO_2}$. If this is the case during recovery, the time constant of a decrease in $\dot{V}_E$ would be shorter than that in $\dot{V}_{CO_2}$. However, our experiment demonstrated that the recovery response of $\dot{V}_E$ was slower than that of $\dot{V}_{CO_2}$ during both passive and active recovery. This suggests that ventilatory response follows the change in $\dot{V}_{CO_2}$. In this study, both central command and muscular neural mechanoreflexes were more likely to be absent during passive recovery at stationary rest [5–7]. Therefore, the present findings suggest that the flux of CO_2 to the lungs is an important determinant of ventilatory drive during recovery. Consequently, our results provide evidence to support the assertion that neither central command nor muscular afferent neural mechanisms are requisite for $\dot{V}_E$ response during recovery from exercise.

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