Blood Lactate during Recovery from Intense Exercise: Impact of Inspiratory Loading

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BASIC SCIENCES

ABSTRACT

CHIAPPA, G. R., B. T. ROSEGUINI, C. N. ALVES, E. L. FERLIN, J. A. NEDER, and J. P. RIBEIRO. Blood Lactate during Recovery from Intense Exercise: Impact of Inspiratory Loading. *Med. Sci. Sports Exerc.*, Vol. 40, No. 1, pp. 111–116, 2008. **Purpose:** It has long been suggested that inspiratory muscle activity may impact blood lactate levels ($[Lac^-]_B$) during the recovery from dynamic exercise. In this study, we tested the hypothesis that inspiratory muscle activation during recovery from intense exercise would contribute to La^- clearance, thus leading to reduced $[Lac^-]_B$. **Methods:** Twelve healthy men underwent two maximal, incremental exercise tests on different days. During a 20-min inactive recovery period, they breathed freely or against a fixed inspiratory resistance of 15 cm H_2O. During recovery, pulmonary gas exchange was continuously monitored, and serial samples of arterialized venous blood were obtained for $[Lac^-]_B$, pH, PCO_2, and HCO_3^-. **Results:** Subjects presented similar ventilatory and gas-exchange responses at peak exercise during both experimental conditions. $[Lac^-]_B$ during recovery was reduced with inspiratory resistance (7.7 ± 1 vs 10.4 ± 1, 7.8 ± 2 vs 10.3 ± 2, and 7.3 ± 1 vs 9.7 ± 2 mM at 5, 7, and 9 min of recovery, respectively; $P < 0.05$), but no differences were found for blood acid–base status. Inspiratory resistance was associated with increased metabolic demand ($\dot{V}O_2$ and $\dot{V}CO_2$) but improved ventilatory efficiency, with lower $\dot{V}_E/[\dot{V}CO_2]$ and increased alveolar ventilation. **Conclusion:** These data are consistent with the notion that inspiratory muscles may be net consumers of lactate during recovery from intense exercise. **Key Words:** LACTATE METABOLISM, EXCESS POSTEXERCISE OXYGEN CONSUMPTION, INSPIRATORY MUSCLES, WORK OF BREATHING

During recovery from intense exercise, blood lactate concentration ($[Lac^-]_B$) decreases more rapidly when light to moderate exercise is performed (2,3,9,30). In fact, during active recovery at 30–70% of peak oxygen uptake ($\dot{V}O_2$peak), La^- is used as a substrate for oxidative metabolism, thereby increasing the rate of La^- removal from the circulatory system (3). Moreover, the rate of decline in $[Lac^-]_B$ is also influenced by the training state, as demonstrated by the higher rates of La^- removal in endurance-trained subjects (4,25).

It has also been suggested that the inspiratory muscles may affect $[Lac^-]_B$ levels during exercise (11,20,28). A number of studies have demonstrated that, after specific inspiratory muscle training, $[Lac^-]_B$ is lowered at a given intensity of exercise, which has been partially attributed to an improvement in the ability of inspiratory muscles to metabolize La^- (21,28). Moreover, earlier animal studies have suggested that inspiratory muscles might be net consumers of La^- during exercise (11,20). However there is no evidence that the activation of inspiratory muscles during recovery of exercise may alter La^- removal. Therefore, the purpose of the present study was to test the hypothesis that activation of inspiratory muscles during recovery of intense exercise would contribute to La^- clearance, thus leading to reduced $[Lac^-]_B$ when compared with passive recovery.

METHODS

Subjects. Twelve healthy men (mean (± SD) age, 28 ± 2 yr; body weight, 78 ± 5 kg; height, 179 ± 3 cm) participated in the study. All subjects had normal medical history, physical examination, and resting and exercise electrocardiograms. None of the subjects were taking any medications or smoked. They were informed of the discomforts and risks involved, and their written informed consent was obtained before the study. The experimental protocol was approved by the committee for ethics in research of the Hospital de Clinicas de Porto Alegre.

Protocol. Subjects came to the laboratory on two nonconsecutive days to perform maximal incremental

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TABLE 1. Results obtained at peak exercise with and without inspiratory resistance in the recovery period.

	With Resistance	Without Resistance	*P* Value
Peak heart rate (bpm)	189 ± 11	188 ± 9	0.90
Peak SBP (mm Hg)	172 ± 10	174 ± 11	0.60
Peak $\dot{V}O_2$ (mL·min^{-1}·kg^{-1})	45 ± 5	46 ± 5	0.90
Peak $\dot{V}_E$ (L·min^{-1})	150 ± 21	147 ± 25	0.89
Peak $\dot{V}CO_2$ (L·min^{-1})	4.5 ± 0.6	4.4 ± 0.6	0.80
Peak RER	1.3 ± 0.1	1.3 ± 0.2	0.60
Peak V_T (L)	2.8 ± 0.7	2.8 ± 0.8	0.44
Peak T_I	0.58 ± 0.12	0.6 ± 0.1	0.12
Peak T_E	0.56 ± 0.13	0.6 ± 0.2	0.25
Peak *f* (breaths per minute)	55 ± 18	54 ± 13	0.40
Peak power (W)	301 ± 16	298 ± 14	0.65

Data are presented as means ± SD. SBP, systolic blood pressure; $\dot{V}O_2$, oxygen uptake; $\dot{V}_E$, minute ventilation, $\dot{V}CO_2$, carbon dioxide output; RER, respiratory exchange ratio; V_T, tidal volume; T_I, inspiratory time; T_E, expiratory time; *f*, breathing frequency.

exercise, followed by 20 min of recovery. Before both experiments, a canulla was inserted into one of the superficial dorsal hand veins (near the dorsal vein arch), and a heating pad was used to obtain arterialized venous blood samples of blood gases and $[Lac^-]_B$. In one experimental condition, an inspiratory resistance fixed at 15 cm H_2O was connected to the breathing apparatus immediately after the end of the test. This inspiratory resistance has been previously shown to affect the oxygen-uptake kinetics during exercise (8). In the other experimental condition, subjects performed the same test, followed by a 20 min recovery period, without an inspiratory load. The order of the tests, with and without inspiratory resistance, was randomized.

Maximal incremental exercise tests. After a period of adaptation to the procedures, subjects set quietly for 15 min to obtain baseline values for resting oxygen uptake ($\dot{V}O_2$). Experiments were performed on an electrically braked cycle ergometer (ER-900, Ergoline, Jaeger, Würzburg, Germany). The incremental exercise test started with a period of 3 min without resistance, followed by increments of 20 W every min until the subjects reached volitional fatigue. Pedaling rate was maintained at 60–70 rpm. During the procedure, gas exchange was measured breath-by-breath by a previously validated system (Metalyzer 3B, CPX System, Cortex, Leipzig, Germany) (23), which includes rapid O_2 and CO_2 analyzers and a turbine. The system was connected to a two-way Lloyd valve (Warren E. Collins, Inc., Braintree, MA) with low resistance (< 1.5 cm H_2O at 3 L·s^{-1}). For the recovery period with added inspiratory resistance, a threshold system device (Threshold, IMT, Healthscan Products Inc., Cedar Grove, NJ) was inserted on the inspiratory part of the valve. The same system was also used for the control situation, but without inspiratory resistance. This type of inspiratory load maintains a constant resistance, whatever the ventilation level (10). Heart rate was measured from the R–R interval of an electrocardiogram

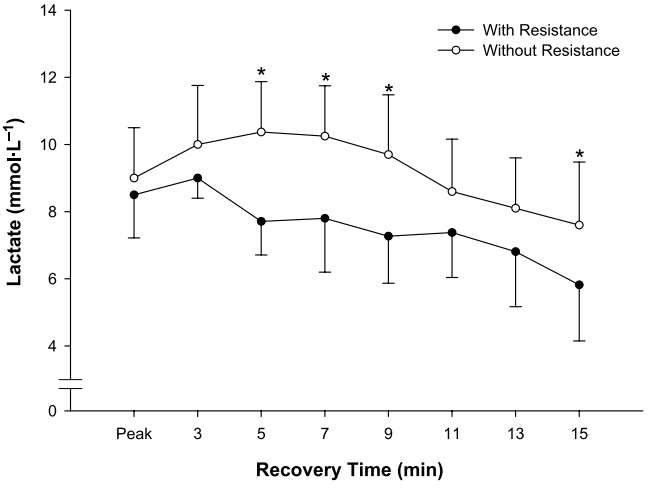

FIGURE 1—Mean (± SD) arterialized blood lactate concentration during recovery with (*filled circles*) or without (*open circles*) inspiratory resistance. ANOVA for repeated measures: group effect $P > 0.05$; time effect $P < 0.05$; interaction $P < 0.05$. * Significantly different by Tukey–Kramer's *post hoc* procedure.

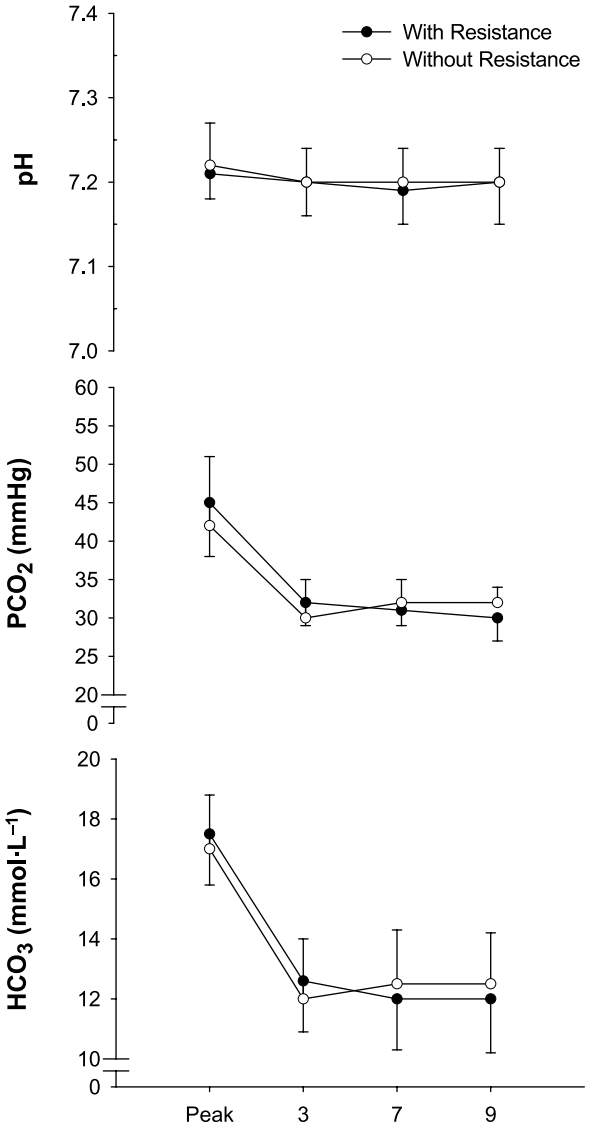

FIGURE 2—pH, PCO_2, and HCO_3^- mean values (± SD) with (*filled circles*) or without (*open circles*) inspiratory resistance in the recovery period. ANOVA for repeated measures: $P > 0.05$.

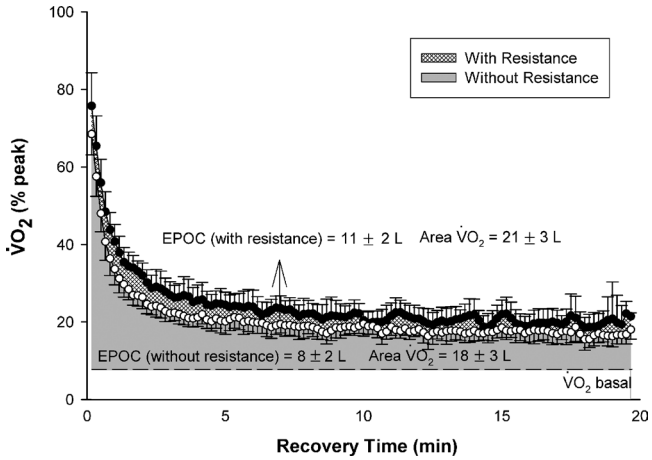

FIGURE 3—Mean (± SD) $\dot{V}O_{2peak}$, expressed as a percentage of $\dot{V}O_{2peak}$, during recovery with (*filled circles*) or without (*open circles*) inspiratory resistance. $P < 0.05$ for the mean area under the curve in the two conditions. EPOC, excess postexercise oxygen consumption.

(Nihon Khoden Corp., Tokyo, Japan), using a 12-lead arrangement. Blood pressure was measured every 2 min with a standard cuff sphygmomanometer.

Values for $\dot{V}O_2$, carbon dioxide output ($\dot{V}CO_2$), minute ventilation ($\dot{V}_E$), end-tidal partial pressure of carbon dioxide ($P_{ET}CO_2$), ventilatory equivalent for carbon dioxide ($\dot{V}_E/[\dot{V}CO_2]$), ventilatory equivalent for oxygen ($\dot{V}_E/[\dot{V}O_2]$), tidal volume (V_T), alveolar ventilation ($V_A = \dot{V}_E\ (1 - V_D/[V_T])$, and inspiratory/expiratory time ratio (T_I/T_{TOT}) were calculated from the measured variables and smoothed by using a 10-s moving average to reduce noise from the respiratory cycle, breath-to-breath V_T variation. Excess postexercise oxygen consumption (EPOC) (amount of oxygen consumed in excess of resting after exercise, as measured in liters) was calculated as the integrated area for the recovery $\dot{V}O_2$ curve minus the mean preexercise value (19).

Blood sampling. Arterialized venous blood samples were drawn before exercise, immediately after exercise, and at 3, 5, 7, 9, 11, 13, and 15 min during recovery. These samples were deproteinized on perchloric acid, centrifuged, and later analyzed in duplicate for $[Lac^-]_B$ using a standard enzymatic method (16,26). Arterialized venous blood was also taken immediately after exercise and at 3, 7, and 9 min in the recovery period for pH, PCO_2, and HCO_3^- calculation (RapidLab 865, Bayer, East Walpole, MA).

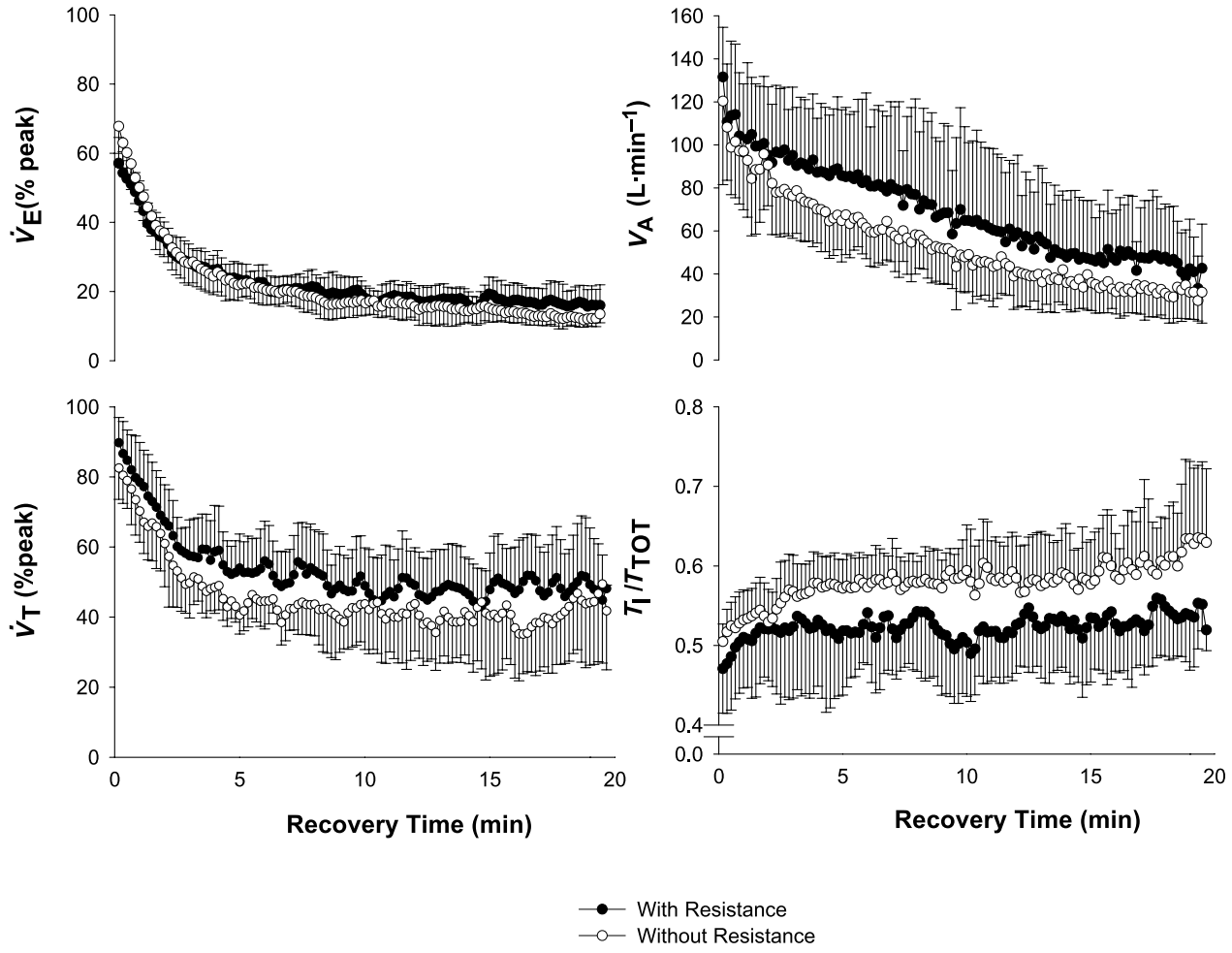

FIGURE 4—Mean (± SD) $\dot{V}_E$ and V_T, expressed as percentages of peak values (*left panels*), as well as V_A and T_I/T_{TOT} (*right panels*) during recovery with (*filled circles*) or without (*open circles*) inspiratory resistance. $P > 0.05$ for the mean area under the curve in the two conditions for $\dot{V}_E$. $P < 0.05$ for the mean area under the curve in the two conditions for V_T, V_A, and T_I/T_{TOT}.

Statistical analysis. Statistical analysis was performed using a commercial statistical software package (SigmaStat 3.1, Richmond, CA). Descriptive data are reported as means ± SD. Paired Student's *t*-tests were used to compare the results of maximal exercise tests and areas under the curves (EPOC, $\dot{V}CO_2$, $\dot{V}_E$, V_A, V_T, $\dot{V}_E/[\dot{V}CO_2]$, $\dot{V}_E/[\dot{V}O_2]$, T_I/T_{TOT}, and $[Lac^-]_B$). During recovery from exercise, variables were compared by one-way analysis of variance for repeated measures. Where appropriate, multiple comparisons were performed with the Tukey–Kramer's *post hoc* procedure. Pearson correlation coefficient was used to evaluate associations between changes in variables. Differences were considered significant for $P < 0.05$.

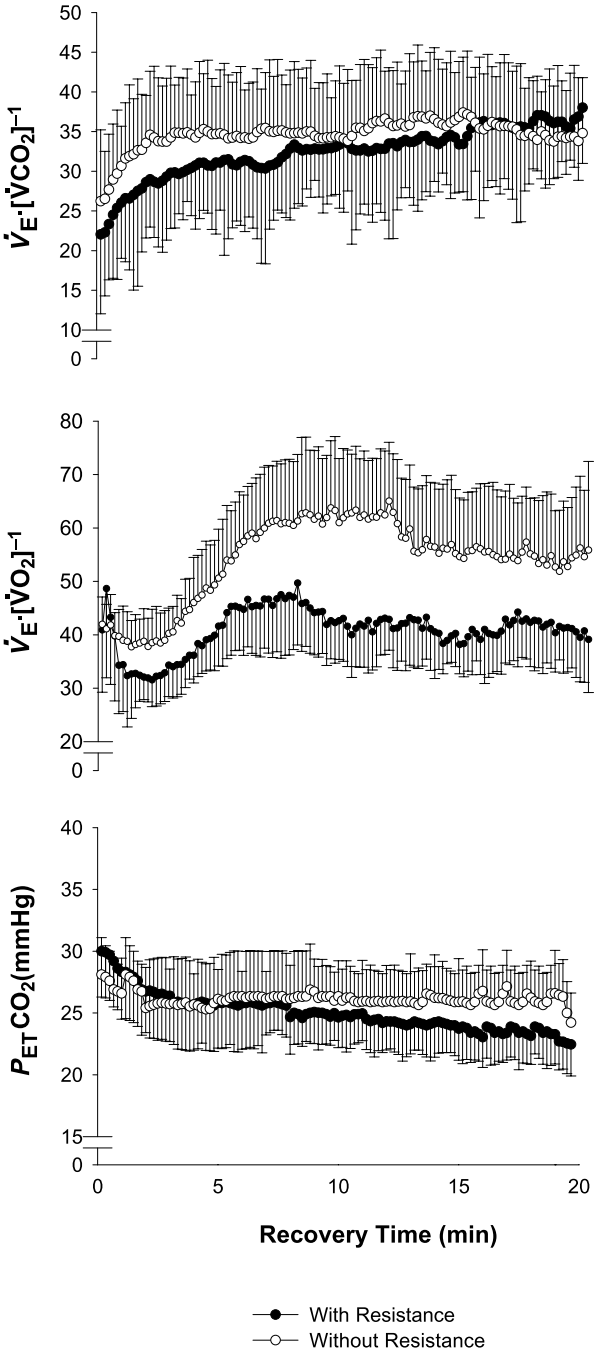

FIGURE 5—Mean (± SD) $\dot{V}_E/[\dot{V}CO_2]$, $\dot{V}_E/[\dot{V}O_2]$, and $P_{ET}CO_2$ during recovery with (*filled circles*) or without (*open circles*) inspiratory resistance. $P < 0.05$ for the mean area under the curve in the two conditions for $\dot{V}_E/[\dot{V}CO_2]$ and $P_{ET}CO_2$.

RESULTS

As shown in Table 1, subjects presented similar gas-exchange variables at peak exercise during both experimental conditions. $[Lac^-]_B$ at 5, 7, 9, and 15 min of recovery was significantly reduced with inspiratory resistance (Fig. 1). Likewise, the mean area under the curve for $[Lac^-]_B$ was significantly smaller with inspiratory resistance (111 ± 18 vs 132 ± 26 [mM]·min); $P < 0.05$). In contrast, arterialized venous blood pH, PCO_2, and HCO_3^- were not altered by the application of inspiratory resistance during the recovery phase (Fig. 2).

Total $\dot{V}O_2$ during recovery (with resistance, 21 ± 3 L; without resistance, 18 ± 3 L; $P < 0.01$) as well as EPOC (with resistance, 11 ± 2 L; without resistance, 8 ± 2 L; $P < 0.01$) increased significantly with inspiratory resistance (Fig. 3). Therefore, the difference of the area under the curve for $\dot{V}O_2$ in the recovery with and without inspiratory resistance (2.8 ± 2 L) indicates the magnitude by which the inspiratory muscles were further activated during the loaded condition. There was no significant correlation between the change in $\dot{V}O_2$ and the change in La^- with inspiratory loading ($r = -0.34$, $P = 0.272$). Likewise, total $\dot{V}CO_2$ during recovery was significantly increased with inspiratory resistance (with resistance, 4 ± 0.4 L; without resistance, 3.5 ± 0.3 L; $P < 0.01$). Despite the aforementioned increase in recovery $\dot{V}O_2$ and $\dot{V}CO_2$, $\dot{V}_E$ responses were similar in the two conditions (Fig. 4). Consequently, inspiratory loading resulted in significant improvements in ventilatory efficiency, as indicated by lower $\dot{V}_E/[\dot{V}CO_2]$ and $\dot{V}_E/[\dot{V}O_2]$ (Fig. 5). Consistent with these findings, V_A was significantly enhanced with inspiratory resistance, with a concomitant decrease in $P_{ET}CO_2$ (Fig. 5). Breathing pattern was significantly changed with the addition of inspiratory resistance, with increased V_T and reductions in duty cycle (T_I/T_{TOT}) responses (Fig. 4).

DISCUSSION

The major finding of this study was that the addition of inspiratory resistance during recovery from intense exercise decreased $[Lac^-]_B$ levels in a group of healthy young males. These data are in agreement with the concept that the activation of inspiratory muscles may increase the clearance of Lac^- after exercise.

Blood La^- removal after intense exercise is the product of a complex interplay of a variety of factors, which include fractional uptake by the liver (24), the heart (29), the brain (17), and the skeletal muscles (6,7). In particular, it is known that oxidative skeletal muscles that contract during the submaximal steady-state condition are ideally suited for La^- consumption (12). In this setting, several studies have

clearly demonstrated that light to moderate cycle exercise after exhaustive efforts resulted in approximately 5–26% lower $[Lac^-]_B$ when compared with passive recovery (4,30). Thus, our finding of approximately 16% lower $[Lac^-]_B$ during active recovery restricted to the inspiratory muscles compared with the control passive situation extend those from prior reports and suggest that respiratory muscles may also influence La^- removal during recovery from exhaustive exercise. However, the pattern of faster blood La^- decline in our experiments seems to differ from some studies in which mild skeletal muscle exercise was performed (2,4,9,30). In our study, differences are apparent within the first 5 min of recovery, whereas it takes longer in the mild exercise studies. It is possible that the different metabolic capacities of inspiratory muscles when compared with skeletal muscle may account, at least in part, for these findings (19).

Indirect evidence suggests that inspiratory loading may increase respiratory muscle blood flow at the expense of leg blood flow (13,14). In fact, increased respiratory muscle work has been shown to promote reflex sympathoexcitation and vasoconstriction in systemic vascular beds, probably redistributing blood flow from the locomotor limb toward respiratory muscles (15). This augmented perfusion, superimposed with the high capillary density and oxidative capacity of the diaphragm and accessory respiratory muscles, would create a favorable condition for La^- consumption by these muscles. Thus, in our study, it seems reasonable to suggest that at least part of the lower $[Lac^-]_B$ during the loaded condition could be caused by an elevated uptake of La^- by the inspiratory muscles or the heart (29).

The concept that respiratory muscles may use La^- as an energy source is not necessarily new. Fregosi et al. (11) first suggested that inspiratory muscles, like other muscles with a high oxidative capacity, may be net consumers of La^- during exercise. These authors found an augmented La^- concentration in diaphragms of rats in the absence of glycogen use during moderate to severe exercise, which they partially attributed to La^- uptake by the diaphragm from the perfusate arterial blood (12). In humans, a number of studies have reported lower $[Lac^-]_B$ at equivalent intensities of exercise after specific respiratory muscle training (22,27,28), which was explained by a potential training-induced improvement in the ability of inspiratory muscles to metabolize La^- (28). However, to our knowledge, this is the first study to demonstrate that La^- removal can be enhanced by increasing inspiratory work in humans.

In agreement with previous findings, we failed to find an association between postexercise oxygen consumption (EPOC) and $[Lac^-]_B$ (1,3). As previously suggested (5), in our study, the augmented EPOC and $\dot{V}CO_2$ during the loaded condition seem to reflect the additional work of breathing induced by inspiratory resistance. Interestingly, however, the ventilatory response ($\dot{V}_E$) was lower than expected for the increased metabolic demand (Fig. 4), whereas V_A was higher during recovery. Therefore, because Pa_{CO2} was unaltered (Fig. 2), it seems that the increase in V_A closely matched the increased $\dot{V}CO_2$. In this context, it is instructive to consider the metabolic determinants of the $\dot{V}_E$ response: $\dot{V}_E/[\dot{V}CO_2] = 1/Pa_{CO2}\ (1 - V_D/[V_T])$, where $V_D/[V_T]$ is the fractional dead-space ventilation. Therefore, because Pa_{CO2} did not increase with inspiratory resistance (Fig. 2), $\dot{V}_E/[\dot{V}CO_2]$ could have only decreased if V_D/V_T was lower. In fact, loaded breathing was associated with higher V_T (Fig. 4) and, thus, lower $V_D/[V_T]$ and elevated V_A.

An intriguing finding of the present study was the apparently paradoxical lower $[Lac^-]_B$ with similar $[HCO3^-]$ and pH during loaded compared with unloaded breathing (Figs. 1 and 2). To interpret these data, the Stewart's physicochemical approach was used (18). According to this approach, the "dependent" variables ($[HCO3^-]$, pH, and $[H^+]$) can only change if the "independent" variables (PCO_2, the total amount of all weak acids, and the strong ion difference) allow this change (18). In the present study, PCO_2 and, presumably, the strong ion difference (to which La^- is contributory), remained constant. Consequently, we can speculate that $[HCO3^-]$ was not higher with loaded breathing, because the strong ion difference (strong cations minus Cl^- plus La^-) did not increase substantially, despite lower $[Lac^-]_B$ levels. Unfortunately, however, we did not measure blood electrolytes, and we could not confirm this hypothesis.

This study has several limitations. Although we have clearly shown that the addition of inspiratory resistance results in lower $[Lac^-]_B$ during recovery (Fig. 1), we were unable to elucidate the precise mechanisms underlying this finding. Therefore, future studies should address whether, indeed, the activation of inspiratory muscles during recovery results in oxidation of La^- by these muscles, or whether other related mechanisms, such as La^- oxidation by the heart or redistribution of blood flow, could be involved. Likewise, future studies should also measure the strong ion difference to evaluate the mechanisms responsible for the lack of changes in blood acid–base status, despite reductions in $[Lac^-]_B$ with inspiratory loading.

In conclusion, the addition of inspiratory resistance during recovery from intense exercise results in increased oxygen uptake, reduction $[Lac^-]_B$, and change in breathing pattern, without changes in arterialized blood gases or ventilation. These findings are in agreement with the concept that inspiratory muscles are net consumers of La^- during recovery from exercise.

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