

# Inspiratory Loading Intensity Does Not Influence Lactate Clearance during Recovery

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<sup>1</sup>Sport, Health and Performance Enhancement Research Group, School of Science and Technology, Nottingham Trent University, Clifton, Nottingham, UNITED KINGDOM; <sup>2</sup>Division of Nutritional Sciences, School of Biosciences, Sutton Bonington Campus, The University of Nottingham, Leicestershire, UNITED KINGDOM; <sup>3</sup>Department of Gene Therapy, National Heart and Lung Institute, Imperial College London, London, UNITED KINGDOM; and <sup>4</sup>School of Life Sciences, Northumbria University, Newcastle upon Tyne, UNITED KINGDOM

## ABSTRACT

JOHNSON, M. A., D. E. MILLS, D. BROWN, K. BAYFIELD, J. GONZALEZ, and G. R. SHARPE. Inspiratory Loading Intensity Does Not Influence Lactate Clearance during Recovery. *Med. Sci. Sports Exerc.*, Vol. 44, No. 5, pp. 863–871, 2012. **Purpose:** This study examined the effects of different pressure threshold inspiratory loads on lactate clearance and plasma acid–base balance during recovery from maximal exercise. **Methods:** Eight moderately trained males ( $\dot{V}O_{2\text{peak}} = 4.29 \pm 0.46 \text{ L}\cdot\text{min}^{-1}$ ) performed, on different days, four maximal incremental cycling tests (power started at 0 W and increased by  $20 \text{ W}\cdot\text{min}^{-1}$ ) of identical duration (exercise time during the first trial was  $16.32 \pm 1.12 \text{ min}$ ). During 20-min recovery, subjects either rested passively or breathed through a constant pressure threshold inspiratory load of 10 (ITL10), 15 (ITL15), or 20 (ITL20) cm H<sub>2</sub>O. Plasma lactate concentration ( $[\text{La}^-]$ ) was measured, and acid–base balance was quantified using the physicochemical approach, which describes the dependency of  $[\text{H}^+]$  on the three independent variables: strong ion difference ( $[\text{Na}^+] + [\text{K}^+] - [\text{Cl}^-] + [\text{La}^-]$ ), the total concentration of weak acids, and the partial pressure of carbon dioxide. **Results:** Peak exercise responses were not significantly different between trials. During recovery, the area under the plasma  $[\text{La}^-]$  curve was not different between trials (pooled mean =  $261 \pm 60 \text{ mEq}$ ) and the  $[\text{La}^-]$  measured at the end of the 20-min recovery was also similar (passive recovery =  $9.2 \pm 3.1 \text{ mEq}\cdot\text{L}^{-1}$ , ITL10 =  $9.3 \pm 3.1 \text{ mEq}\cdot\text{L}^{-1}$ , ITL15 =  $8.7 \pm 2.8 \text{ mEq}\cdot\text{L}^{-1}$ , ITL20 =  $8.7 \pm 3.2 \text{ mEq}\cdot\text{L}^{-1}$ ). Similarly, changes in other strong ions contributing to strong ion difference and total concentration of weak acids, partial pressure of carbon dioxide, and, therefore,  $[\text{H}^+]$  were not different between trials. **Conclusions:** These data suggest that, in individuals of moderate endurance training status, inspiratory loading at the intensities used in the present study does not accelerate lactate clearance or modify plasma acid–base balance during recovery from maximal exercise. **Key Words:** INSPIRATORY LOADING, RECOVERY, LACTATE CLEARANCE, ACID–BASE BALANCE, CYCLING

Elevated muscle and blood lactate concentrations ( $[\text{La}^-]$ ) and the accompanying  $\text{H}^+$  accumulation have long been associated with impaired muscle function and exercise tolerance (15). After high-intensity exercise, an active recovery using the previously exercised muscles quickens the decline in blood  $[\text{La}^-]$  (12,29,32,41) and, compared with passive recovery, may result in better maintenance of performance in subsequent exercise bouts (3,31). Conversely, active recovery using the previously exercised muscles may impair subsequent performance because of slower rates of muscle reoxygenation and phosphocreatine

resynthesis (6,39). Because the primary fate of lactate during exercise and recovery is oxidative metabolism by skeletal muscle (2,17,18), the high capillary density and oxidative capacity of the inspiratory muscles (33) make them ideally suited to lactate metabolism. The use of inspiratory loading during recovery may thus provide an alternative strategy by which lactate clearance may be enhanced without compromising the repletion of intramuscular energy stores within the previously exercised locomotor muscles.

Two recent issues of *Medicine & Science in Sports & Exercise*® (5,9) present conflicting evidence regarding whether inspiratory muscle loading can quicken blood lactate clearance after high-intensity exercise. Chiappa et al. (9) showed that a pressure threshold inspiratory load (15 cm H<sub>2</sub>O) during recovery from high-intensity exercise greatly accelerated lactate clearance from the blood. Specifically, when compared with passive recovery, the absolute blood  $[\text{La}^-]$  and area under the curve (AUC) for blood  $[\text{La}^-]$  were lower with inspiratory loading by around  $2.5 \text{ mEq}\cdot\text{L}^{-1}$  (–24%) and 21 mEq (–16%), respectively. Furthermore, a follow-up study showed that peak cycling power in the second of two successive Wingate tests increased by 25% after this recovery

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Submitted for publication July 2011.

Accepted for publication November 2011.

0195-9131/12/4405-0863/0

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DOI: 10.1249/MSS.0b013e31824079d0

intervention (8). However, despite using a similar methodology, Brown et al. (5) failed to reproduce the observations of Chiappa et al. (8,9). Specifically, lactate clearance during recovery from high-intensity exercise was accelerated by a 15 cm H<sub>2</sub>O pressure threshold inspiratory load only after inspiratory muscle training and then only by a much smaller degree to that reported by Chiappa et al. (8,9).

Interestingly, Chiappa et al. (9) reported large reductions in blood [La<sup>-</sup>] with inspiratory loading without the concomitant changes in plasma [H<sup>+</sup>] and [HCO<sub>3</sub><sup>-</sup>] that would be expected to occur. According to the physicochemical approach to acid–base balance (40), [H<sup>+</sup>] in intracellular and extracellular fluid is, together with HCO<sub>3</sub><sup>-</sup>, a dependent variable whose value is dependent on the equilibrium state reached by the independent variables: strong ion difference ([SID], fully dissociated cations minus anions, which, in plasma, is equal to [Na<sup>+</sup>] + [K<sup>+</sup>] – [Cl<sup>-</sup>] + [La<sup>-</sup>]), the total concentration of weak acids ([A<sub>tot</sub><sup>-</sup>]), and partial pressure of carbon dioxide (PCO<sub>2</sub>) (for reviews, see Johnson et al. [21], Kowalchuk and Scheuermann [23], and Lindinger et al. [25]). Therefore, for plasma [H<sup>+</sup>] to have remained unchanged with inspiratory loading, the large reduction in blood [La<sup>-</sup>] observed by Chiappa et al. (9) must have been balanced by corresponding changes in the concentration of other strong ions and/or [A<sub>tot</sub><sup>-</sup>] (40). This hypothesis was not supported by the work of Brown et al. (5), which showed that concentrations of other strong ions and [A<sub>tot</sub><sup>-</sup>] were unaffected by inspiratory loading both before and after inspiratory muscle training. Indeed, the lower blood [La<sup>-</sup>] with inspiratory loading after inspiratory muscle training was exclusively responsible for an increase in [SID] and, subsequently, a lower [H<sup>+</sup>] (5).

One potential explanation for the disagreement between the findings of Chiappa et al. (8,9) and Brown et al. (5) may be that the magnitude of the pressure threshold load is an important determinant of whether inspiratory loading accelerates lactate clearance during recovery. Specifically, after inspiratory muscle training, which increased participants' maximum inspiratory mouth pressure (MIP) by 34%, lactate clearance was accelerated only when inspiratory loading was performed at the same absolute (15 cm H<sub>2</sub>O or 10% MIP) but not relative (20 cm H<sub>2</sub>O or 13% MIP) intensity (5).

Therefore, the aim of the present study was to resolve some of the controversies surrounding the effects of inspiratory loading during recovery from high-intensity exercise. Consequently, we investigated the effects of different pressure threshold inspiratory loads (10, 15, and 20 cm H<sub>2</sub>O), applied during recovery from high-intensity exercise, on lactate clearance and plasma acid–base balance quantified using the physicochemical approach.

## METHODS

**Participants.** After approval from Nottingham Trent University's ethics committee, eight healthy nonsmoking males with normal lung function (Table 1) provided written

TABLE 1. Descriptive characteristics of the participants.

Variable	
Age (yr)	26 ± 6
Body mass (kg)	80 ± 6
Height (cm)	184 ± 4
FVC (L)	5.54 ± 0.51 (101 ± 9)
FEV <sub>1</sub> (L)	4.40 ± 0.52 (96 ± 10)
FEV <sub>1</sub> /FVC (%)	80 ± 4 (96 ± 5)
MVV <sub>10</sub> (L·min <sup>-1</sup> )	190 ± 36 (112 ± 22)
MIP (cm H <sub>2</sub> O)	145 ± 39 (126 ± 34)

Data are presented as mean ± SD. Values in parentheses represent the percentage of predicted values (26,45).

FVC, forced vital capacity; FEV<sub>1</sub>, forced expiratory volume in 1 s; MVV<sub>10</sub>, maximal voluntary ventilation in 10 s.

informed consent to participate in the study. Throughout the study, participants were instructed to adhere to their habitual training regimen and not to engage in any strenuous exercise the day preceding and the day of a trial. Participants arrived at the laboratory 2 h postprandially having abstained from alcohol and caffeine in the 24 h before testing.

**Experimental design.** Participants attended the laboratory on five separate occasions, at a similar time of day, separated by at least 48 h but no more than 1 wk. During the first laboratory visit, pulmonary function and MIP were measured. During subsequent visits, participants performed an incremental cycling exercise test (the first of which was continued to the limit of tolerance) followed by a 20-min recovery period comprising either passive recovery or breathing against a constant inspiratory pressure threshold load of 10 (ITL10), 15 (ITL15), or 20 cm H<sub>2</sub>O (ITL20). The order of the exercise trials was randomized. The exercise duration achieved during the incremental exercise test of the first trial was replicated in all subsequent trials.

**Pulmonary function and MIP.** Pulmonary function was assessed according to published guidelines (30) using a pneumotachograph (Pneumotrac; Vitalograph, Buckingham, United Kingdom) calibrated using a 3-L syringe. A handheld mouth pressure meter (MicroRPM; CareFusion, Hampshire, United Kingdom) measured MIP as an index of global inspiratory muscle strength. The mouthpiece assembly incorporated a 1-mm orifice to prevent glottic closure during inspiratory efforts. Maneuvers were performed in an upright standing posture, initiated from residual volume, and sustained for at least 1 s. Repeat efforts separated by 30 s were performed until three serial measures differed by no more than 10% or 10 cm H<sub>2</sub>O, whichever was smallest (5). The highest value recorded was used for subsequent analysis. For each participant, MIP was compared with predicted values using the equation of Wilson et al. (45), where  $MIP_{\text{predicted}} = 142 - (1.03 \times \text{age})$ .

**Maximal incremental cycling test.** Exercise was performed on an electromagnetically braked cycle ergometer (Excalibur Sport; Lode, Groningen, The Netherlands). During the first trial, a maximal incremental cycling test was performed, whereby power started at 0 W and increased by 20 W·min<sup>-1</sup> until participants could not maintain a cadence greater than 60 rpm despite verbal encouragement. In an

attempt to standardize the magnitude of elevation of plasma  $[La^-]$ , the exercise duration achieved during this first test was recorded for each participant, and during subsequent tests, each participant was required to exercise for an identical duration, at which point the test was terminated by the investigators. Cycling cadence was self-selected during the first test and repeated during subsequent tests. Participants wore a face mask (model 7940; Hans Rudolph, Kansas City, MO) connected to a pneumotachograph and a two-way non-rebreathing valve (model 2730; Hans Rudolph). Respiratory variables were measured breath by breath (ZAN 600USB; nSpire Health, Oberthulba, Germany). The final power attained and the highest oxygen uptake ( $\dot{V}O_2$ ) recorded during any 30-s period defined maximal power ( $\dot{W}_{max}$ ) and  $\dot{V}O_{2peak}$ , respectively. The prescribed recovery intervention (see below) was imposed immediately upon cessation of exercise.

**Passive recovery.** At the cessation of the incremental exercise test, a 0.5-m length of wide-bore (35-mm internal diameter) corrugated tubing was attached to the inspiratory port of the two-way valve, and participants remained seated at rest on the cycle ergometer for 20 min. The tubing attached to the inspiratory port was used in the inspiratory loading trials to connect the participant to the inspiratory loading device (see below). Blood samples were taken at rest, upon the cessation of exercise, and every 4 min during recovery. Upon the cessation of exercise and every 2 min during recovery, HR was recorded using short-range telemetry (Polar S610; Polar Electro, Kempele, Finland), and arterial oxygen saturation ( $SpO_2$ ) was estimated using a finger pulse oximeter (model 8500; Nonin Medical, Plymouth, MN).

**Inspiratory pressure threshold loading.** Inspiratory loading trials were identical with the passive recovery trial, except that immediately after exercise, the 0.5-m length of the wide-bore tube connected to the inspiratory port of the two-way valve was connected distally to a custom-built weighted plunger pressure threshold inspiratory loading device identical with that used previously (5,19,20) and that has been shown to be flow independent over the physiological range (19); for a full description of the device, see Johnson et al. (20). The threshold opening pressure during ITL10, ITL15, and ITL20 represented  $8\% \pm 3\%$ ,  $12\% \pm 4\%$ , and  $16\% \pm 5\%$  of MIP, respectively. Absolute pressure threshold loads were used to allow direct comparison with the work of Chiappa et al. (8,10) and Brown et al. (5). We have previously shown that the performance of our breath-by-breath gas analyzer is unaffected by the negative pressures generated during inspiratory loading (5).

**Blood sampling and analysis.** Arterialized venous blood (6 mL) was drawn from a dorsal hand vein via an indwelling 21-gauge cannula (28). Arterialization was achieved by immersing the hand in water at  $40^\circ C$  for 10 min before cannulation and by warming the hand during trials using an infrared lamp. Blood was analyzed immediately for  $PCO_2$  and pH (ABL520; Radiometer, Copenhagen, Denmark), which were subsequently used to calculate plasma bicarbonate

concentration ( $[HCO_3^-]$ ) using the Henderson–Hasselbalch equation:

$$pH = pK + \log \frac{[HCO_3^-]}{0.03PCO_2}$$

A portion (5 mL) of each blood sample was immediately centrifuged for 10 min at 3000g, and the plasma supernatant was removed. Plasma  $[La^-]$  was subsequently determined using an automated analyzer (Biosen C\_line Sport; EKF Diagnostics, Barleben, Germany). Plasma  $[Na^+]$ ,  $[K^+]$ , and  $[Cl^-]$  were determined using ion-selective electrodes, and total protein concentration ( $[PPr^-]$ ) was assayed by immunoturbidimetry (ABX Pentra 400; Horiba, Northampton, United Kingdom).  $[A_{tot}^-]$  was calculated as  $2.45 \times [PPr^-]$  (37). Plasma  $[SID]$  was calculated as the sum of the strong cations minus the sum of the strong anions (40):

$$[SID] = ([Na^+] + [K^+]) - ([Cl^-] + [La^-])$$

Plasma  $[H^+]$  was calculated using the following equation, which describes the dependency of  $[H^+]$  on the three independent physicochemical variables ( $[SID]$ ,  $PCO_2$ , and  $[A_{tot}^-]$ ), as well as on mass action equilibria, conservation of mass, and electroneutrality (40):

$$[H^+]^4 + (K_A + [SID])[H^+]^3 + \{K_A([SID] - [A_{tot}^-]) - (K_C PCO_2 + K'_W)\} \times [H^+]^2 - \{K_A(K_C PCO_2 + K'_W) + (K_3 K_C PCO_2)\}[H^+] - (K_A K_3 K_C PCO_2) = 0$$

Values used for the equilibrium constants were  $K_A = 3.0 \times 10^{-7} \text{ Eq}\cdot\text{L}^{-1}$ ,  $K_C = 2.45 \times 10^{-11} (\text{Eq}\cdot\text{L}^{-1})^2$ ,  $K_3 = 6.0 \times 10^{-11} \text{ Eq}\cdot\text{L}^{-1}$ , and  $K'_W = 4.4 \times 10^{-14} \text{ Eq}\cdot\text{L}^{-1}$ .

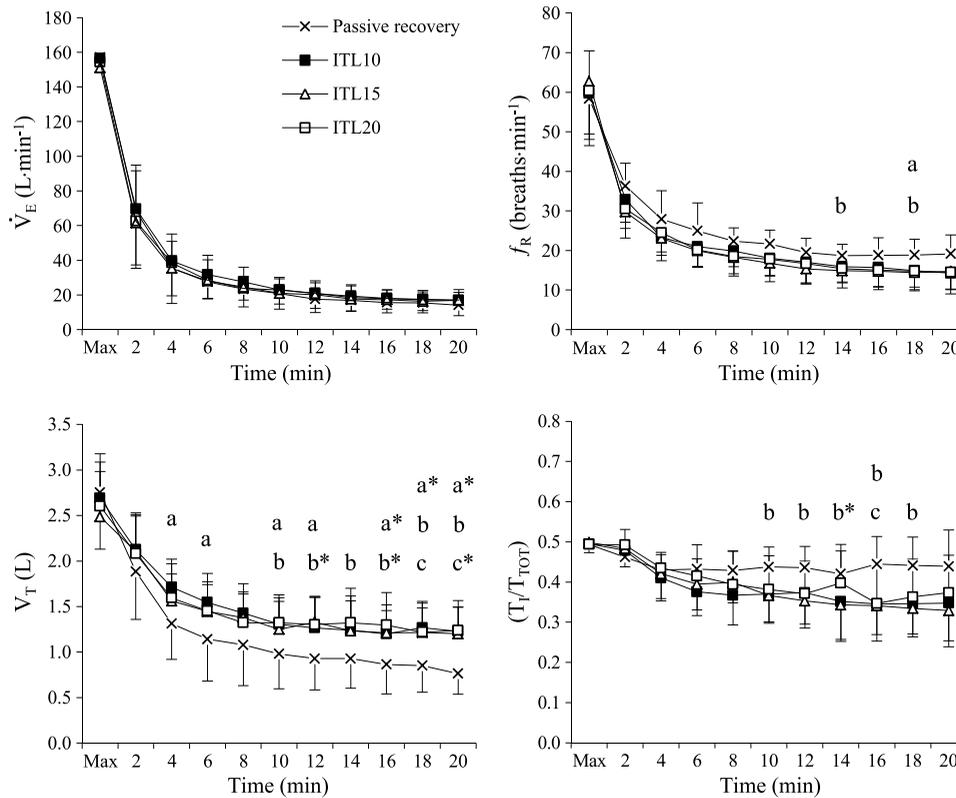
To assess the individual contributions of the three independent variables ( $[SID]$ ,  $PCO_2$ , and  $[A_{tot}^-]$ ) to changes in plasma  $[H^+]$ , the latter was calculated as one of the three variables was changed to the measured value, whereas the others were held constant at resting values (35,40). Hereafter, the measured and calculated  $[H^+]$  are called  $[H^+]_{measured}$  and  $[H^+]_{calculated}$ , respectively.

**Data and statistical analysis.** The AUC for  $\dot{V}O_2$  and plasma  $[La^-]$  during recovery was calculated using the trapezoidal rule. Data were analyzed using repeated-measures ANOVA, and pairwise comparisons were made according to Bonferroni-adjusted 95% confidence intervals. Statistical significance was set at  $P < 0.05$ . Results are presented as mean  $\pm$  SD.

## RESULTS

For each participant, the exercise duration and, thus,  $\dot{W}_{max}$  achieved during the first incremental exercise test (group mean:  $16.32 \pm 1.12$  min and  $318 \pm 20$  W) were replicated in all subsequent tests.

**Ventilatory responses, pulmonary gas exchange, HR, and arterial  $SpO_2$ .** Breathing pattern, minute ventilation ( $\dot{V}_E$ ) ( $155 \pm 27 \text{ L}\cdot\text{min}^{-1}$ ),  $\dot{V}O_{2peak}$  ( $4.29 \pm 0.46 \text{ L}\cdot\text{min}^{-1}$ ),  $\dot{V}CO_{2peak}$  ( $5.30 \pm 0.75 \text{ L}\cdot\text{min}^{-1}$ ), HR ( $178 \pm 9 \text{ beats}\cdot\text{min}^{-1}$ ), and  $SpO_2$  ( $96\% \pm 2\%$ ) at the end of maximal exercise were not different between trials (values in parentheses are pooled



**FIGURE 1**—Ventilatory responses at the end of maximal exercise and during 20-min recovery. Values are mean  $\pm$  SD. Difference between trials ( $P < 0.05$ ): <sup>a</sup>passive recovery versus ITL10; <sup>b</sup>passive recovery versus ITL15; <sup>c</sup>passive recovery versus ITL20. \* $P < 0.01$ .

data from all trials).  $\dot{V}_E$  throughout recovery was not different between trials (Fig. 1). Respiratory frequency ( $f_R$ ) during passive recovery ( $23 \pm 4$  breaths per minute) was greater than that during ITL10 ( $19 \pm 4$  breaths per minute) and ITL15 ( $18 \pm 4$  breaths per minute) (main effect for trial,  $P < 0.05$ ). There was also a trend ( $P = 0.09$ ) for  $f_R$  to be higher during passive recovery compared with ITL20 ( $19 \pm 4$  breaths per minute). Tidal volume during passive recovery ( $1.07 \pm 0.35$  L) was lower (main effect for trial) than that during ITL10 ( $1.43 \pm 0.30$  L) ( $P < 0.01$ ), ITL15 ( $1.39 \pm 0.27$  L) ( $P < 0.01$ ), and ITL20 ( $1.42 \pm 0.31$  L) ( $P < 0.05$ ). Duty cycle ( $T_V/T_{TOT}$ ) during passive recovery ( $0.44 \pm 0.04$ ) was greater than that during ITL15 ( $0.38 \pm 0.06$ ) (main effect for trial,  $P < 0.05$ ). There was a trial  $\times$  time interaction effect for  $T_V/T_{TOT}$  ( $P < 0.01$ ), and differences were observed for passive recovery versus ITL15 and ITL20 in the latter half of recovery (Fig. 1). There were no differences between trials for mean  $\dot{V}O_2$  during recovery (pooled mean =  $0.77 \pm 0.28$  L·min<sup>-1</sup>) or the AUC for  $\dot{V}O_2$  during recovery (pooled mean =  $19.4 \pm 2.9$  L). Mean  $\dot{V}CO_2$  during passive recovery ( $0.72 \pm 0.14$  L·min<sup>-1</sup>) was not different from ITL15 ( $0.83 \pm 0.19$  L·min<sup>-1</sup>) but was less than ITL10 ( $0.87 \pm 0.14$  L·min<sup>-1</sup>) and ITL20 ( $0.86 \pm 0.13$  L·min<sup>-1</sup>) (main effect for trial,  $P < 0.05$ ). HR during recovery was not different between trials (pooled mean =  $101 \pm 15$  beats·min<sup>-1</sup>). In all trials, SpO<sub>2</sub> was  $98\% \pm 1\%$  at rest and  $97\% \pm 1\%$  during recovery.

**Plasma ions contributing to [SID].** Changes in plasma ions contributing to [SID] are shown in Figure 2. Changes in [La<sup>-</sup>], [Na<sup>+</sup>], [K<sup>+</sup>], and [Cl<sup>-</sup>] after maximal exercise and during recovery were not different between trials; thus, the following data reflect the pooled mean  $\pm$  SD. Plasma [La<sup>-</sup>] increased to  $15.4 \pm 2.7$  mEq·L<sup>-1</sup> after maximal exercise and peaked at  $16.0 \pm 2.8$  mEq·L<sup>-1</sup> after 4 min of recovery, after which values progressively declined. The mean AUC for plasma [La<sup>-</sup>] during recovery was  $261 \pm 60$  mEq and was not different between trials. Plasma [Na<sup>+</sup>] increased  $6.2 \pm 2.7$  mEq·L<sup>-1</sup> from rest after maximal exercise (main effect for time,  $P < 0.01$ ) and remained elevated after 4 min of recovery ( $P < 0.05$ ). Thereafter, values were not different from rest. Plasma [K<sup>+</sup>] increased  $1.1 \pm 0.3$  mEq·L<sup>-1</sup> from rest after maximal exercise (main effect for time,  $P < 0.01$ ) and returned to rest after 4 min of recovery. Plasma [Cl<sup>-</sup>] was unchanged from rest after maximal exercise but fell by  $2.0 \pm 1.6$  mEq·L<sup>-1</sup> after 4 min of recovery (main effect for time,  $P < 0.05$ ). Plasma [Cl<sup>-</sup>] remained below rest at 8, 12 ( $P < 0.01$ ), and 16 min ( $P < 0.05$ ) of recovery and returned to rest after 20 min of recovery.

**Plasma protein and the independent variables [SID], [A<sub>tot</sub><sup>-</sup>], and PCO<sub>2</sub>.** Changes in [PPr<sup>-</sup>], [SID], [A<sub>tot</sub><sup>-</sup>], and PCO<sub>2</sub> after maximal exercise and during recovery were not different between trials (Fig. 3); thus, the following data reflect the pooled mean  $\pm$  SD. [PPr<sup>-</sup>] increased  $0.9 \pm 0.3$  g·dL<sup>-1</sup> from rest after maximal exercise

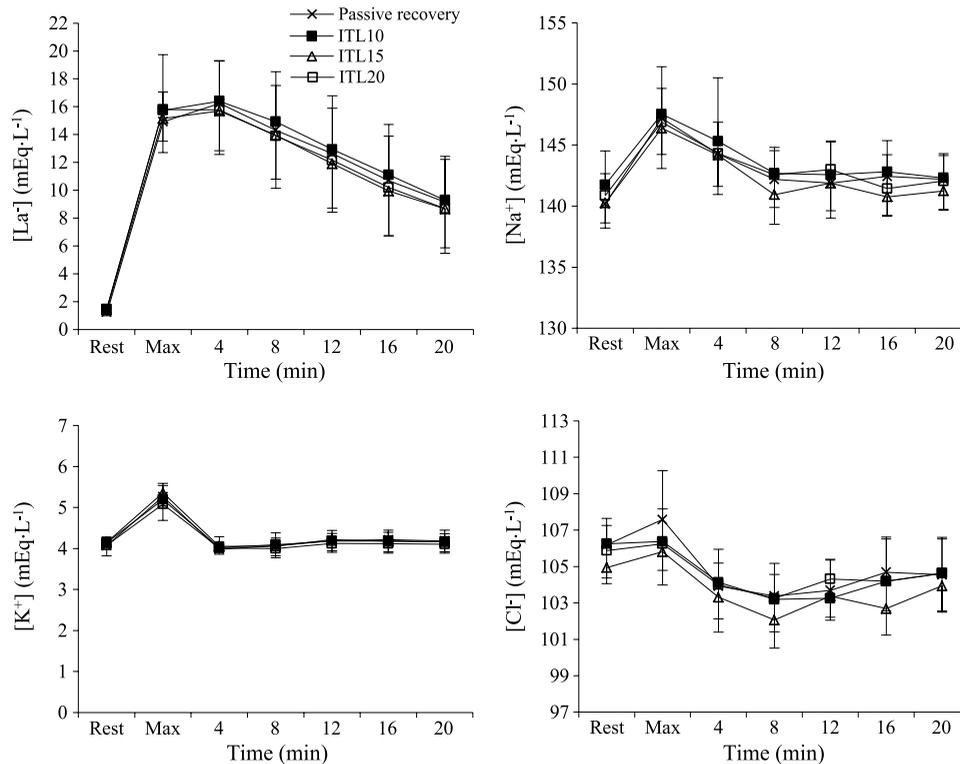


FIGURE 2—Plasma ion concentrations at rest, after maximal exercise, and during 20-min recovery. Values are mean  $\pm$  SD.

(main effect for time,  $P < 0.01$ ) and remained above rest throughout recovery (4–16 min inclusive,  $P < 0.01$ ; 20 min,  $P < 0.05$ ). [SID] fell  $7.3 \pm 1.8$  mEq·L<sup>-1</sup> below rest after maximal exercise (main effect for time,  $P < 0.01$ ) and remained below rest throughout recovery ( $P < 0.01$ ).  $[A_{\text{tot}}^-]$  increased  $2.2 \pm 0.7$  mEq·L<sup>-1</sup> from rest after maximal exercise (main effect for time,  $P < 0.01$ ) and remained above rest throughout recovery (4–16 min inclusive,  $P < 0.01$ ; 20 min,  $P < 0.05$ ). PCO<sub>2</sub> remained unchanged from rest after maximal exercise but then declined and remained  $7.3 \pm 4.0$  mm Hg below rest throughout recovery (main effect for time: 4–16 min inclusive,  $P < 0.01$ ; 20 min,  $P < 0.05$ ).

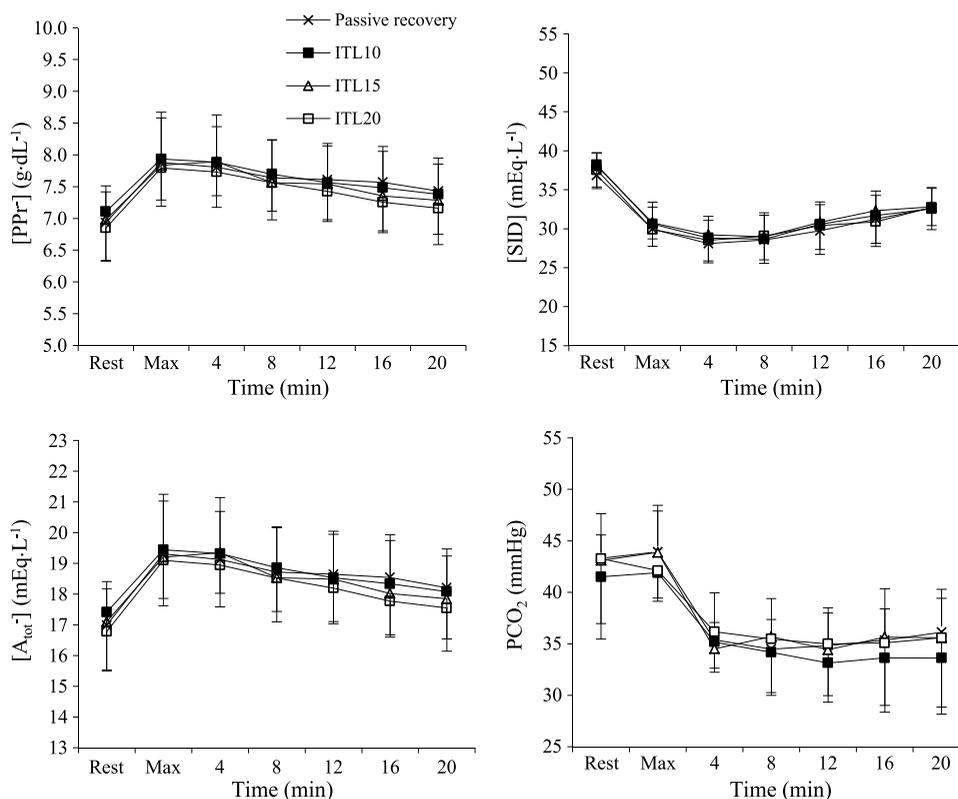
**Changes in the dependent variables [H<sup>+</sup>] and [HCO<sub>3</sub><sup>-</sup>].** Changes in  $[H^+]_{\text{measured}}$  and  $[HCO_3^-]$  after maximal exercise and during recovery were not different between trials (Fig. 4); thus, the following data reflect the pooled mean  $\pm$  SD.  $[H^+]_{\text{measured}}$  increased  $20.9 \pm 8.2$  nEq·L<sup>-1</sup> after maximal exercise (main effect for time,  $P < 0.01$ ) and remained above rest during 12 min of recovery (4 min,  $P < 0.01$ ; 8–12 min,  $P < 0.05$ ). Thereafter, values were not different from rest. The mean AUC for  $[H^+]_{\text{measured}}$  was  $1095 \pm 137$  nEq and was not different between trials.  $[HCO_3^-]$  fell  $8.2 \pm 2.0$  mEq·L<sup>-1</sup> below rest after maximal exercise and remained below rest throughout recovery (main effect for time,  $P < 0.01$ ).

A change in plasma  $[H^+]$  reflects the net change of the three independent variables ([SID], PCO<sub>2</sub>, and  $[A_{\text{tot}}^-]$ ) (40). The proportion of change in plasma  $[H^+]$  caused by a corresponding change in a discrete independent variable

was calculated while assuming no change from rest in the remaining two independent variables. Similar to previous reports (5,35), an excellent agreement was observed between  $[H^+]_{\text{measured}}$  and  $[H^+]_{\text{calculated}}$  ( $r = 0.848$ ,  $P < 0.0001$ ). Plasma  $[H^+]_{\text{calculated}}$  and the origins of change in  $[H^+]_{\text{calculated}}$  due to changes in the three independent variables are shown in Figure 5, with positive values representing an acidifying effect and negative values representing an alkalinizing effect.  $[H^+]_{\text{calculated}}$  increased by  $22.5$  nEq·L<sup>-1</sup> after maximal exercise during the passive recovery trial. Of this increase, 67% ( $15.1$  nEq·L<sup>-1</sup>) was attributable to a decrease in [SID]. The slight increase in PCO<sub>2</sub> after maximal exercise contributed a further  $1.6$  nEq·L<sup>-1</sup> (+7%), whereas the increase in  $[A_{\text{tot}}^-]$  contributed  $3.4$  nEq·L<sup>-1</sup> (+15%).  $[H^+]_{\text{calculated}}$  began to fall after 4 min of recovery. This was primarily due to a fall in PCO<sub>2</sub> that reduced  $[H^+]_{\text{calculated}}$  by  $7.2$  nEq·L<sup>-1</sup> and countered the further decrease in [SID], which alone would have increased  $[H^+]_{\text{calculated}}$  by  $20.9$  nEq·L<sup>-1</sup>. For the remainder of recovery, the decline in  $[H^+]_{\text{calculated}}$  toward resting values was primarily due to a gradual increase in [SID] and the maintenance of PCO<sub>2</sub> below rest. As evident in Figure 5, the origins of change in  $[H^+]_{\text{calculated}}$  during passive recovery were markedly similar to that observed during inspiratory loading trials.

## DISCUSSION

The main finding of the present study was that lactate clearance and changes in plasma acid–base balance during

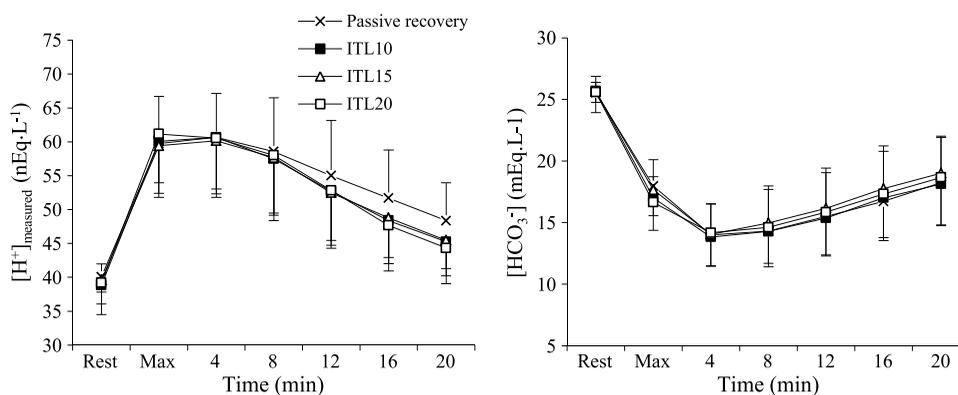


**FIGURE 3**—[PPr<sup>-</sup>] and the independent variables [SID], [A<sub>tot</sub><sup>-</sup>], and PCO<sub>2</sub> at rest, after maximal exercise, and during 20-min recovery. Values are mean ± SD.

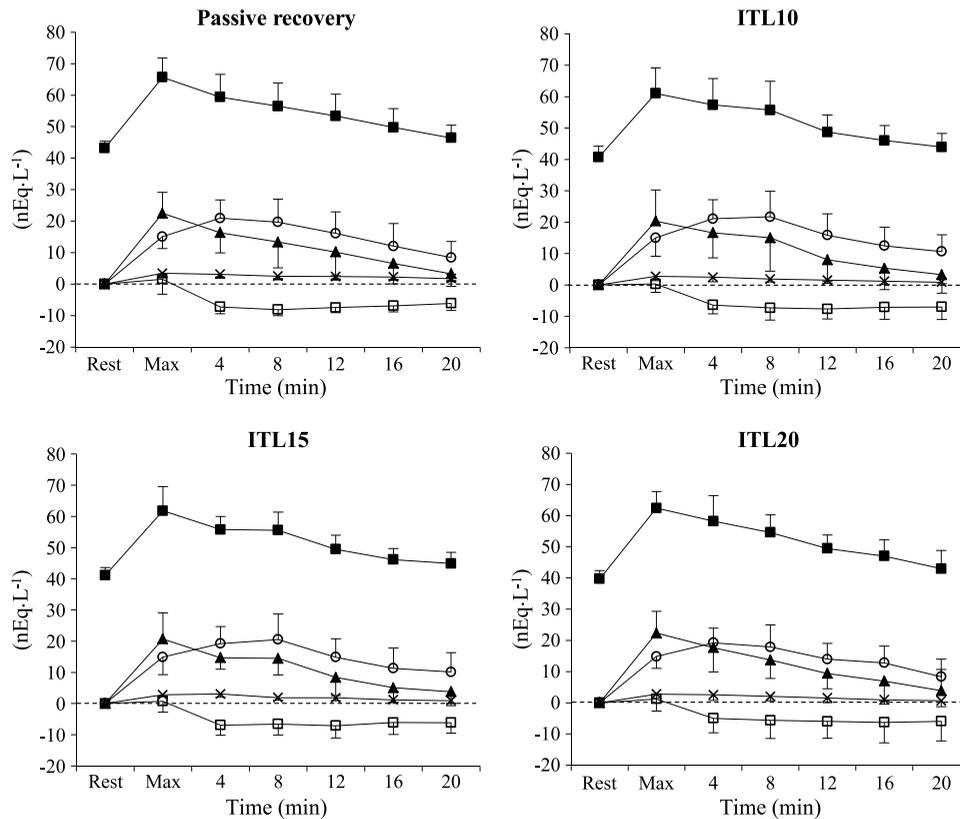
recovery from maximal exercise were not affected by pressure threshold inspiratory loading at 10, 15, or 20 cm H<sub>2</sub>O.

That ITL15 did not influence lactate clearance during recovery after maximal incremental exercise concurs with our previous observations (5). In contrast, using the same inspiratory load and a similar incremental exercise test, Chiappa et al. (9) reported an approximate 2.5-mEq·L<sup>-1</sup> (25%) reduction in blood [La<sup>-</sup>] during 15-min recovery. Given the positive relationship between active muscle mass during recovery and rates of lactate clearance (17,32), it is surprising that the relatively small inspiratory muscles can affect lactate clearance to a similar magnitude as the leg muscles (12,29,41). However, the inspiratory muscles do

possess a superior capillary density and oxidative capacity compared with limb muscle (33), which makes them ideally suited for lactate metabolism. Chiappa et al. (9) also report large reductions in blood [La<sup>-</sup>] with inspiratory loading without corresponding changes in [H<sup>+</sup>]. In our previous study (5), inspiratory loading lowered blood [La<sup>-</sup>] (after inspiratory muscle training), thereby increasing [SID], which lowered [H<sup>+</sup>]. In the absence of a change in blood PCO<sub>2</sub>, the unchanged [H<sup>+</sup>] observed by Chiappa et al. (9) was attributed to changes in other (nonmeasured) strong ions that prevented an increase in [SID]. For this to be the case, the net change in the other strong ions must have been positive and equimolar to the reduction in [La<sup>-</sup>] (i.e., the



**FIGURE 4**—Dependent variables [H<sup>+</sup>]<sub>measured</sub> and [HCO<sub>3</sub><sup>-</sup>] at rest, after maximal exercise, and during 20-min recovery. Values are mean ± SD.



**FIGURE 5**—Plasma  $[H^+]_{\text{calculated}}$  (■), change in  $[H^+]_{\text{calculated}}$  (▲), and origins of change in  $[H^+]_{\text{calculated}}$  from changes in the three independent variables:  $[SID]$  (○),  $PCO_2$  (□), and  $[A_{\text{tot}}]$  (×), after maximal exercise and during 20-min recovery. Values are mean  $\pm$  SD.

net change in cations and  $[Cl^-]$  would need to balance the  $2.5\text{-mEq}\cdot\text{L}^{-1}$  reduction in  $[La^-]$ . Inspiratory loading–induced reductions in  $[K^+]$  and/or  $[Na^+]$  and/or increases in  $[Cl^-]$  would allow this. After exercise, transient increases in  $[K^+]$  largely reflect significant efflux from muscle followed by rapid reuptake (22,24). Evidence to support the notion that inspiratory loading could significantly reduce  $K^+$  efflux from locomotor muscles or accelerate  $K^+$  reuptake is lacking. Furthermore, increases in plasma  $[Na^+]$  and  $[Cl^-]$  after exercise are largely attributed to reduced plasma volume (21,44). We can find no reason why inspiratory loading in recovery would either lessen the postexercise reduction in plasma volume (thereby causing a relative decrease in  $[Na^+]$ ) or accentuate the decrease in plasma volume (thereby increasing  $[Cl^-]$ ). Besides, any change in plasma volume results in simultaneous changes in  $[Na^+]$  and  $[Cl^-]$  causing little net change in  $[SID]$  and, therefore,  $[H^+]$ . Our data (Fig. 2) and those of others (22,24) also show that postexercise changes in  $[Na^+]$  and  $[K^+]$  are transient and therefore unlikely to balance changes in  $[La^-]$  during a recovery period of 15 min. Inspiratory loading during recovery is thus likely to influence  $[SID]$  only through changing  $[La^-]$  (5). Furthermore, inspiratory loading is unlikely to influence plasma  $[A_{\text{tot}}]$ , which also increases after maximal exercise secondary to exercise intensity–dependent reductions in plasma volume (21,35). Although Chiappa et al. (9) did not measure strong ion concentrations, our data do not support

their assertion that changes in these ions may explain their findings. Thus, given our current understanding of physicochemical principles (40), it is difficult to resolve the reason(s) why their results differ from ours. However, our results demonstrate no effect of inspiratory loading during recovery on lactate kinetics, and these data are also supported by our measurement of strong ions.

The differences between our findings and those of Chiappa et al. (8,9) are intriguing because experimental protocols are very similar. Compared with Chiappa et al. (8,9), we observed a higher  $\dot{V}O_{2\text{peak}}$  (present study =  $53.6\text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ , Brown et al. (5) =  $52.4\text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ , Chiappa et al. (8) =  $47.9\text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ , Chiappa et al. (9) =  $45.5\text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) and, during recovery, a shorter time to peak  $[La^-]$  and a faster decline in  $[La^-]$ , which implies a greater endurance training status in our participants (16,42,43). Because training tends to increase rates of blood and muscle lactate clearance (2,13,16), it seems counterintuitive that individuals with inferior endurance training status would demonstrate greater lactate clearance with inspiratory loading. However, coincident with a lower blood  $[La^-]$  with inspiratory loading during recovery, Chiappa et al. (8) also reported increased HR and mean arterial blood pressure and, as measured by near-infrared spectroscopy, reduced m. vastus lateralis reoxygenation (suggestive of reduced convective oxygen delivery). These responses were attributed to respiratory muscle metaboreflex

activation, whereby increased afferent discharge from inspiratory muscles and a concurrent increase in sympathetic vasoconstrictor activity result in a redistribution of blood flow from the periphery to the inspiratory muscles (11). A resultant increase in inspiratory muscle perfusion would probably increase lactate uptake by inspiratory muscles (18). Conversely, by virtue of endurance training-induced increases in inspiratory muscle mitochondrial enzyme activity (34), the “threshold” for respiratory muscle metaboreflex activation may have been higher in our participants (1,26,46), thus precluding changes in HR, cardiac output distribution, and, hence, systemic lactate distribution. Indeed, recent work by Callegaro et al. (7) demonstrates that, compared with sedentary individuals ( $\dot{V}O_{2\text{peak}} = 36.7 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ), those with a history of whole-body endurance training ( $\dot{V}O_{2\text{peak}} = 52.9 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) are more resistant to activation of the inspiratory muscle metaboreflex. The unchanged HR responses during inspiratory loading compared with passive recovery in the present and in our previous study (5) partly support this notion. Of course, from an ecological validity perspective, if the efficacy of loaded breathing in aiding recovery is restricted to untrained individuals, one may question its utility.

Previously, Brown et al. (5) showed that after inspiratory muscle training, which increased participants' MIP by 34%, lactate clearance was accelerated at the same absolute (15 cm H<sub>2</sub>O or 10% MIP) but not relative (20 cm H<sub>2</sub>O or 13% MIP) inspiratory load, thus suggesting that the magnitude of the pressure threshold load may be an important determinant of whether inspiratory loading accelerates lactate clearance. The findings of the present study do not support this hypothesis, although the possibility remains that inspiratory loads above 20 cm H<sub>2</sub>O may have increased lactate clearance due to activation of a respiratory muscle metaboreflex (see above). However, it is noteworthy that anecdotal reports from our participants suggest that 20 cm H<sub>2</sub>O was approaching the maximal tolerable load (most participants reported severe air hunger especially during the first 5 min of recovery). Notwithstanding this, it seems increasingly likely that a training-induced increase in the

oxidative capacity of the inspiratory muscles (4,27,36,46), rather than the intensity of the load relative to MIP, primarily explains our previous observation of increased lactate clearance during recovery with inspiratory loading after inspiratory muscle training (5).

Consistent with previous observations (5,9), inspiratory loading modified breathing pattern compared with passive recovery. Specifically, the same  $\dot{V}_E$  was achieved with a lower  $f_R$  and  $T_I/T_{\text{TOT}}$  but greater tidal volume. This breathing strategy was probably generated by the respiratory controller to minimize the energy cost of breathing by reducing the pressure-time integral of the inspiratory muscles (14,47). Furthermore, reducing  $T_I/T_{\text{TOT}}$  prolongs expiration, thus increasing inspiratory muscle relaxation time and reducing end-expiratory lung volume, which enhances inspiratory muscle force-generating capacity (14,38,47). This breathing pattern could also affect gas exchange by increasing the ratio of alveolar to dead space ventilation, which may explain why inspiratory loading in the present study increased  $\dot{V}CO_2$  and resulted in a trend toward lower  $[H^+]_{\text{measured}}$  (Fig. 4). These changes were, however, very small and probably of limited significance.

In conclusion, pressure threshold inspiratory loading at 10, 15, and 20 cm H<sub>2</sub>O had no effect on lactate clearance or plasma acid-base balance when applied during recovery from maximal incremental exercise. These findings support our recent observations (5) but contradict those of Chiappa et al. (8,9). The reason(s) for this disagreement remains unclear but may be related to interindividual differences in endurance training status. The findings of the current study and of Brown et al. (5) also suggest that training of the inspiratory muscles may be necessary for this muscle group to engage in measurable lactate uptake with inspiratory loading. The influence of endurance training status on the efficacy of inspiratory muscle loading during recovery thus provides an attractive avenue for future investigation.

No funding was received for this work.

The authors have no conflicts of interest to declare.

The results of the present study do not constitute endorsement by the American College of Sports Medicine.

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