Active and Passive Recovery and Acid-Base Kinetics Following Multiple Bouts of Intense Exercise to Exhaustion

J.C. Siegler, J. Bell-Wilson, C. Mermier, E. Faria, and R.A. Robergs

The purpose of this study was to profile the effect of active versus passive recovery on acid-base kinetics during multiple bouts of intense exercise. Ten males completed two exercise trials. The trials consisted of three exercise bouts to exhaustion with either a 12 min active (20% workload max) or passive recovery between bouts. Blood pH was lower in the passive (p) recovery compared to active (a) throughout the second and third recovery periods [second recovery: 7.18 ± 0.08 to 7.24 ± 0.09 (p), 7.23 ± 0.07 to 7.32 ± 0.07 (a), P < 0.05; third recovery: 7.17 ± 0.08 to 7.22 ± 0.09 (p), 7.23 ± 0.08 to 7.32 ± 0.08 (a), P < 0.05]. Exercise performance times did not differ between recovery conditions (P = 0.28). No difference was found between conditions for recovery kinetics (slope and half-time to recovery). Subsequent performance during multiple bouts of intense exercise to exhaustion may not be influenced by blood acidosis or mode of recovery.

Key Words: blood acidosis, pH, lactate, training specificity

The ability to recover quickly from multiple short duration, high-intensity exercise bouts is essential in sports incorporating such an activity pattern (e.g., soccer, rugby, hockey, and basketball). The most effective method to obtain full recovery (as assessed by various sport related performance indices) in relatively short time frames has yet to be established. In related work, numerous studies have indicated that low-intensity exercise during recovery enhances metabolic waste removal from muscle (predominately lactate and H+) into the blood more rapidly than passive recovery (1, 2, 6, 18, 19, 27). In sport application, the importance of active recovery, however, may be negligible if performance during subsequent high-intensity exercise bouts is not enhanced.

Although commonly assumed by sport participants that active recovery presents a competitive advantage, research evidence remains equivocal. A comparative review of the literature reveals that of the studies demonstrating a beneficial
performance effect during subsequent exercise bouts while implementing active recovery modes (1, 4, 28), an almost equal number have found little or no improvement (18, 30). Recovery profiles are often difficult to establish, as most studies implement short duration (1 to 5 min), high-intensity sprints integrated with short recovery time frames (< 30 s). Variation in exercise application (intensity, mode, subject populations, etc.) and measurement techniques have also made between study comparisons problematic.

Establishing metabolic (blood acid-base) recovery profiles during multiple bouts of short duration, high-intensity exercise may provide insight toward establishing an explanation for performance discrepancies between studies. To our knowledge, such comparative (active versus passive) models of blood acid-base recovery profiles have not been presented. Therefore, we studied the effects of an active or passive recovery on the acid-base response to and recovery from multiple bouts of high-intensity exercise to exhaustion. We hypothesized that the recovery kinetics of both blood pH and lactate, as seen through various recovery time points and recovery slopes, would differ between the active and passive recovery conditions. In addition, due to differences in the metabolic byproduct production rate of lactate and protons, we hypothesized that blood recovery kinetics would also differ between lactate and protons.

**Methods**

**General Procedures**

Ten male subjects (age, 30.0 ± 6.8 y, height, 180.3 ± 7.5 cm; weight, 76.3 ± 9.7 kg; VO\textsubscript{2max}, 55.7 ± 6.5 mL · kg\textsuperscript{-1} · min\textsuperscript{-1}; body fat, 11.6 ± 4.3%) completed two trials in random order [Trial I: passive recovery (PRP) and Trial II: active recovery (ARP)] separated by 1 wk. Prior to the two trials, subjects underwent familiarization procedures and a baseline VO\textsubscript{2max} test. The study protocol was approved by the university institutional review board. All testing was conducted at an altitude of 1570 m, and temperature, percent relative humidity (RH), and barometric pressure recorded for each trial.

**Exercise Protocol**

**Orientation and Exercise Familiarization Trials.** Subject orientation began with an initial consultation of the study expectations and a review/signing of the institutionally approved informed consent and health questionnaire. All subjects were informed of the procedures and possible risks and side effects, and excluded on the basis of self-reported pre-existing medical conditions contraindicative to the study’s testing regimen. Subjects were instructed as to the importance of nutritional intake and told to control, record, and duplicate intake for all trials. All participants kept written logs of food intake the evening prior to the first trial, and were told to avoid spicy foods, include adequate carbohydrates (as per RDA recommendations), and avoid excessive intake of meat protein. Subjects were also required to refrain from exercise 24 h prior to each trial. On the morning of all exercise procedures, the subjects ingested only two cans (325 mL, 220 kcal each) of liquid meal replacement
(Slim-Fast, Slim-Fast Foods Co., West Palm Beach, FL) and 500 mL of water 3 h before exercise to standardize caloric, acid, and base dietary intake.

The exercise familiarization trials were designed to orient the subjects with the effort requirement for cycling until volitional fatigue (\(VO_{2\max}\) trial), as well as exercising under successive supramaximal conditions (110% workload maximum) (9). Subjects that asked to use clip-less pedals were required to use them throughout the study, otherwise subjects used standard Lode cycle ergometer toe clips. During this visit, subjects were also familiarized with breathing through a two-way non-rebreathing valve used during all exercise bouts (\(VO_2\) data collection).

\(VO_{2\max}\). The second visit to the laboratory required the subjects to perform a \(VO_{2\max}\) test on a Lode cycle ergometer (Lode BV, Groningen, The Netherlands). All subjects participated in a standardized warm-up prior to the test (2 min at 80 RPM and 50 W). Either a 25 or 30 W/min (subject size dependent) ramped incremental protocol was used to attain volitional fatigue (~ 10 to 12 min). Expired gas fractions were measured by metabolic analyzers (AEI Technologies) and data for \(VO_2\) and \(VCO_2\) were computed for each breath throughout exercise. The analyzers and expired air flow were calibrated prior to each exercise test. For all subjects, \(VO_{2\max}\) was defined as the attainment of a plateau in \(VO_2\) where an increase in \(VO_2\) of \(\leq 50\) mL/min occurred with increasing workload. \(VO_{2\max}\) was identified using a \(\pm 15\) s average around the highest \(VO_2\). Maximal power output (MWO) was recorded from the corresponding time of \(VO_{2\max}\) attainment. Heart rate was monitored continuously and recorded every 15 s throughout the exercise trial using an electrocardiography system (Quinton, Q4000, Bothell, WA).

**Blood Sampling and Analysis (PRP and ARP)**

Prior to PRP and ARP, a catheter was inserted into a heated dorsal hand vein (10 min, 35°C hot water bath). Resting baseline blood samples (2 mL discard followed by a 1 mL sample for blood pH, bicarbonate (\(HCO_3^-\)), and base excess (BE) and another 1 mL sample for lactate) were obtained before all trials. The hand remained heated throughout the test using a fan-forced heater. The catheter was connected to a three-way stopcock and the stopcock and catheter were flushed with 2 mL of sterile saline between blood draws. Blood samples (2 mL flush and two 1 mL samples) were drawn in separate syringes. The 1 mL samples (acid-base) were drawn into a heparin coated 1 mL syringe, immediately capped, and placed on ice until subsequent (performed immediately following collection) blood gas and acid-base analyses using a clinical blood gas analyzer (Bayer Rapidlab 865, Pittsburgh, PA). The other 1 mL sample was capped, centrifuged at 3000 RPM (Marathon 21K/BR, Fisher Scientific, Pittsburgh, PA), and the plasma removed and refrigerated for later analysis of lactate using enzymatic spectrophotometry (10vis, Thermo Spectronic, Madison, WI).

**PRP – 110% (3 Bouts) and Passive Recovery Periods.** Once catheterized, subjects completed a standardized 5 min warm-up of cycle ergometry exercise based upon their MWO from the \(VO_{2\max}\) test (Table 1) (3). Upon completing the warm-up, subjects were required to sit quietly on the cycle for 5 min (pilot work determined the need for 5 min rest to allow \(VO_2\) to return to resting levels after warm-up as we collected breath-by-breath \(VO_2\) data during all exercise bouts).
Subjects then performed a bout of intense cycle ergometer exercise with an external load and cadence of 90 to 100 RPM (equivalent to ~110% of their MWO). The duration of the test was determined by using a handheld stopwatch, started on the initial downward pedal stroke and ending the first drop in power output below 60 RPM. The same research assistant performed the timing for all tests to reduce tester variability. The exercise bout was repeated two more times (three bouts total).

The PRP began immediately after each exercise bout and lasted for 12 min. Recovery blood samples were drawn (2 mL discard, 1 mL acid-base, and 1 mL lactate) immediately post-exercise (time 0) for each of the three bouts and at 2, 4, 6, 8, 10, and 12 min. All recovery samples were drawn with the subject seated on the cycle ergometer. After 12 min of passive rest, the subject again exercised under the same conditions as stated previously. Upon volitional fatigue, recovery samples were again drawn [21 total for three post exercise bouts, approximately 88 mL per trial (including baseline)]. This pattern continued until all three exercise bouts were completed.

**ARP – 110% (3 Bouts) and Active Recovery Period.** The exercise protocol was replicated from the prior bout (as described above). Similarly, all blood samples were drawn as per the PRP. The only difference between the protocols was the recovery activity. Recovery samples were drawn with the subject cycling at 60 RPM and 20% of their MWO for 12 min (8, 25).

**Statistical Analyses**

Data are presented as mean ± standard deviation. Dependent variables considered were blood pH, lactate, HCO₃⁻ and BE. Prior to analysis, all data were assessed for normal distribution, homogeneity of variance, and independence of errors. An a priori power analysis for pH and lactate variables (standard deviation from previous data collected in our lab) also revealed a sample size of 10 was sufficient to achieve a power of 0.8. The blood data were analyzed using a two-way repeated measures ANOVA design, where one effect was the condition (passive recovery, active recovery) and the second effect was time. One-way repeated measures ANOVA

**Table 1 Standardized Warm-up Protocol for All Supramaximal Trials (110%)**

<table>
<thead>
<tr>
<th>Time</th>
<th>%MWO</th>
<th>RPM</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 min</td>
<td>25</td>
<td>70-80</td>
</tr>
<tr>
<td>30s</td>
<td>50</td>
<td>90-100</td>
</tr>
<tr>
<td>30s</td>
<td>25</td>
<td>70-80</td>
</tr>
<tr>
<td>30s</td>
<td>65</td>
<td>90-100</td>
</tr>
<tr>
<td>30s</td>
<td>25</td>
<td>70-80</td>
</tr>
<tr>
<td>30s</td>
<td>80</td>
<td>90-100</td>
</tr>
<tr>
<td>30s</td>
<td>25</td>
<td>70-80</td>
</tr>
</tbody>
</table>

*Note.* All subjects warmed up at a percentage of their maximal Watt output (MWO) and for the same time intervals [minutes and seconds (s)].
design was used to assess recovery kinetics, average performance VO₂, and performance times (time to fatigue) between trials. A two-phase exponential slope function \( Y = \text{Span}_1 \times (-K_1 \times X) + \text{Span}_2 \times (-K_2 \times X) + \text{plateau} \) was used to assess recovery slope for all variables between conditions. The slope of the second function (second phase) was presented due to the recovery nature of all variables. Second function slope data was analyzed starting from the low recovery value and ending at the end recovery value. Statistical analysis was done using Statistica software (StatSoft, Inc., Tulsa, OK) and GraphPad Prism 3.0 (GraphPad Software, Inc., San Diego, CA). Post-hoc analysis was conducted for all significant interactions using Tukey’s HSD. Statistical significance was set at \( P < 0.05 \).

Results

Data Presentation

The group mean ± standard deviation data are presented numerically and in table form as phases of blood acid-base changes for pre-exercise [PE], low-recovery values [LRV] (lowest recorded value during the recovery period), end-recovery values [ERV] (last recorded value during the recovery period), slope of second function and performance results (time to fatigue). The recovery of blood pH, lactate, \( \text{HCO}_3^- \) and BE were best fit with non-linear, two-phase exponential functions (Table 2). Graphic representations are presented for recovery curves and values of pH and lactate for illustration purposes and to aid in reader clarity.

Pre-Exercise

There was no difference prior to exercise between the PRP and ARP trials for the conditions of blood pH (passive: 7.39 ± 0.03; active: 7.40 ± 0.02), lactate (passive: 1.6 ± 0.5 mmol/L; active: 1.9 ± 0.8 mmol/L), \( \text{HCO}_3^- \) (passive: 22.7 ± 3.1 mmol/L; active 23.3 ± 3.0 mmol/L) and BE (passive: –2.1 ± 2.1; active: –1.1 ± 2.8).

Low and End-Recovery Values

pH. The interaction between time and trial for LRV was significant \( [F(2,18) = 5.96; P = 0.010] \). Post hoc analysis revealed differences between passive and active LRV during the second and third recovery periods (Recovery 2, \( P < 0.001 \); Recovery 3, \( P < 0.001 \)) (Figures 1b and c). ERV were also different between time and trial \([F(2,18) = 8.95; P < 0.001]\). Post hoc analysis illustrated differences between passive and active ERV during all three recovery periods (Recovery 1, \( P < 0.001 \); Recovery 2, \( P < 0.001 \); Recovery 3, \( P < 0.001 \)) (Figures 1a, b and c).

Lactate. Interaction between time and trial was not significant for either value of high lactate (HLV) \([F(2,18) = 1.44; P = 0.264]\) or ERV \([F(2,18) = 3.26; P = 0.062]\) (Figures 2a, b, and c). Graphic representations of the recovery curves are presented to illustrate trends and for comparisons between lactate and pH recovery kinetics.

\( \text{HCO}_3^- \) and BE. LRV and ERV of \( \text{HCO}_3^- \) were not significantly different \([LRV: F(2,10) = 0.08; P = 0.926; ERV: F(2,10) = 1.27; P = 0.323]\) (Table 3). LRV was
Table 2  Group Mean Data ($r^2 \pm$ Standard Deviation) Represents a Comparison Between Curvilinear (2 Phase Exponential Slopes) vs. Linear Regression Lines of Best Fit for All Variables (Passive and Active Recoveries)

<table>
<thead>
<tr>
<th></th>
<th>Passive Recovery</th>
<th></th>
<th></th>
<th>Active Recovery</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Recovery 1</td>
<td>Recovery 2</td>
<td>Recovery 3</td>
<td>Recovery 1</td>
<td>Recovery 2</td>
<td>Recovery 3</td>
</tr>
<tr>
<td></td>
<td>2 phase Linear</td>
<td>2 phase Linear</td>
<td>2 phase Linear</td>
<td>2 phase Linear</td>
<td>2 phase Linear</td>
<td>2 phase Linear</td>
</tr>
<tr>
<td>pH</td>
<td>0.85 ± 0.2</td>
<td>0.40 ± 0.3</td>
<td>0.91 ± 0.1</td>
<td>0.95 ± 0.1</td>
<td>0.46 ± 0.3</td>
<td>0.96 ± 0.0</td>
</tr>
<tr>
<td></td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
</tr>
<tr>
<td>HCO$_3^-$</td>
<td>0.92 ± 0.1</td>
<td>0.35 ± 0.3</td>
<td>0.95 ± 0.1</td>
<td>0.85 ± 0.2</td>
<td>0.32 ± 0.3</td>
<td>0.79 ± 0.3</td>
</tr>
<tr>
<td></td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
</tr>
<tr>
<td>BE</td>
<td>0.94 ± 0.1</td>
<td>0.29 ± 0.4</td>
<td>0.95 ± 0.0</td>
<td>0.27 ± 0.2</td>
<td>0.21 ± 0.2</td>
<td>0.28 ± 0.3</td>
</tr>
<tr>
<td></td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
</tr>
<tr>
<td>La$^-$</td>
<td>0.81 ± 0.1</td>
<td>0.43 ± 0.4</td>
<td>0.76 ± 0.2</td>
<td>0.39 ± 0.3</td>
<td>0.84 ± 0.2</td>
<td>0.72 ± 0.1</td>
</tr>
<tr>
<td></td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
</tr>
</tbody>
</table>

Note: 2 phase, 2 phase exponential function; Linear, Linear regression; sig, significance value.
Figure 1a-c—Presented are group data (means ± standard deviation) for blood pH for all recovery periods (active and passive conditions). The lines represent the average 2-phase recovery slopes for mean data. Included in the graphs are numerical presentations of low recovery values (LRV) and end recovery values (ERV).

a) Recovery Period 1

- **Passive**
- **Active**

*7.32±0.06 (ERV)*

7.23±0.05 (LRV)

7.21±0.05 (LRV)

7.35

7.30

7.25

7.20

7.15

0 2 4 6 8 10 12

Time (min)

* = statistically different from passive (p < 0.001)

LRV = low recovery value

ERV = end recovery value

b) Recovery Period 2

- **Passive**
- **Active**

*7.32±0.07 (ERV)*

7.23±0.07 (LRV)

7.18±0.07 (LRV)

7.24±0.09 (ERV)

7.35

7.30

7.25

7.20

7.15

0 2 4 6 8 10 12

Time (min)

* = statistically different from passive (p < 0.001)

LRV = low recovery value

ERV = end recovery value

c) Recovery Period 3

- **Passive**
- **Active**

6.5±0.7 (HLV)

6.1±1.2 (HLV)

5.8±0.9 (ERV)

4.0

4.5

5.0

5.5

6.0

6.5

7.0

0 2 4 6 8 10 12

Time (min)

HLV = high lactate value

ERV = end recovery value
Figure 2a-c—Presented are group data (means ± standard deviation) for blood lactate for all recovery periods (active and passive conditions). The lines represent the average 2-phase recovery slopes for mean data. Included in the graphs are numerical presentations of high lactate values (HLV) and end recovery values (ERV).
Table 3  Group HCO$_3^-$ and BE Data (Mean ± Standard Deviation) is Presented for Low Recovery Values (LRV) [High Recovery Values (HRV) in the Case of BE] and End Recovery Values (ERV)

<table>
<thead>
<tr>
<th>Trial</th>
<th>HCO$_3^-$ (mmol/L)</th>
<th>BE (meq/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Recovery 1</td>
<td>Recovery 2</td>
</tr>
<tr>
<td>PRP</td>
<td>LRV</td>
<td>ERV</td>
</tr>
<tr>
<td></td>
<td>11.7 ± 2.4</td>
<td>12.8 ± 2.9</td>
</tr>
<tr>
<td>ARP</td>
<td>14.0 ± 6.7</td>
<td>16.5 ± 7.1</td>
</tr>
<tr>
<td>PRP</td>
<td>HRV</td>
<td>ERV</td>
</tr>
<tr>
<td></td>
<td>–14.1 ± 2.8</td>
<td>–12.7 ± 3.3</td>
</tr>
<tr>
<td>ARP</td>
<td>–14.0 ± 2.2</td>
<td>–10.0 ± 2.5$^a$</td>
</tr>
</tbody>
</table>

Note. $^a$significantly different from passive ($P < 0.05$); $^b$significantly different from previous recovery (within trial) ($P < 0.05$); PRP, passive recovery period; ARP, active recovery period; LRV, low recovery value; HRV, high recovery value; ERV, end recovery value.
not different for BE values either \([F(2,10) = 1.04; P = 0.389]\), however there was a significant interaction between time and trial for ERV for BE \([F(2,10) = 4.25; P = 0.046]\) (Table 3).

**Slope of Second Function**

Group mean recovery slopes are presented for both passive and active recovery conditions in Table 4. Limited data points resulted in the inability to elicit differences between slopes for any of the measured variables.

**Average Peak Performance VO\(_2\)**

*(Final 30 s of Exercise)*

Average peak VO\(_2\) did not vary between conditions for any of the exercise trials (Table 5).

**Performance**

Time to fatigue for all trials is presented in Table 6. No differences in fatigue times were observed between conditions \([F(2,18) = 1.35; P = 0.284]\).

**Discussion**

The unique findings of this study were that 1) although blood acid-base recovery was affected by recovery mode (passive versus active), subsequent performance times remained similar, 2) recovery slopes (specific to their allotted recovery time frames) for most variables were similar and independent of recovery mode, and 3) blood acid-base recovery from such bouts may be influenced by training specificity.

**Blood Acid-Base Recovery and Subsequent Performance**

The relative acidotic state that subjects remained in during the second and third passive recovery trials (as evidenced in Figure’s 1 and 2 ERV) illustrates the enhanced metabolic waste removal associated with active recovery. This result, although rarely documented for multiple bouts, has been shown in numerous other studies implementing similar exhaustive single bout exercise protocols (1, 2, 6, 18, 19, 27). Due to the exponential rise in the concentration of blood H\(^+\) and lactate caused by the high non-mitochondrial ATP turnover during the exercise bouts, it was expected that an eventual limiting factor to performance would be the overall depletion of muscle and blood buffering capacities (as confirmed by others; 14, 17, 29). However, our subjects displayed a wide range of blood buffering depletion, resulting in high standard deviations for all HCO\(_3^-\) mean values (Table 3) (This discrepancy will be addressed further in the discussion under Training Specificity). Further, the variations in ERV levels of HCO\(_3^-\) did not appear to affect the subjects as evidenced in their performance times (Table 6). Ventilatory compensation (or buffering) may also be excluded based on the unaltered state of oxygen consumption during either condition (Table 5). These results suggest blood buffering capacity...
Table 4  Group Data for the Second Exponential Slope of pH, Lactate, \( HCO_3^- \) and BE

<table>
<thead>
<tr>
<th>Trial</th>
<th>Recovery 1</th>
<th>Recovery 2</th>
<th>Recovery 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>PRP</td>
<td>0.17 ± 0.15</td>
<td>0.15 ± 0.10</td>
<td>0.15 ± 0.10</td>
</tr>
<tr>
<td>ARP</td>
<td>0.39 ± 0.21</td>
<td>0.24 ± 0.20</td>
<td>0.34 ± 0.21</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Trial</th>
<th>Recovery 1</th>
<th>Recovery 2</th>
<th>Recovery 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>PRP</td>
<td>0.13 ± 0.20</td>
<td>0.41 ± 0.40</td>
<td>0.19 ± 0.26</td>
</tr>
<tr>
<td>ARP</td>
<td>0.20 ± 0.15</td>
<td>-0.10 ± 0.89</td>
<td>1.86 ± 3.93</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Trial</th>
<th>Recovery 1</th>
<th>Recovery 2</th>
<th>Recovery 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>PRP</td>
<td>1.96 ± 2.83</td>
<td>0.73 ± 0.28</td>
<td>10.36 ± 12.35</td>
</tr>
<tr>
<td>ARP</td>
<td>0.83 ± 0.49</td>
<td>1.91 ± 3.28</td>
<td>1.77 ± 2.29</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Trial</th>
<th>Recovery 1</th>
<th>Recovery 2</th>
<th>Recovery 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>PRP</td>
<td>0.31 ± 0.23</td>
<td>0.56 ± 0.54</td>
<td>0.91 ± 0.36</td>
</tr>
<tr>
<td>ARP</td>
<td>0.48 ± 0.54</td>
<td>0.31 ± 0.17</td>
<td>0.43 ± 0.34</td>
</tr>
</tbody>
</table>

Note. Values are means ± standard deviation. PRP, = passive recovery period; ARP, = active recovery period.

Table 5  Group Data for Peak VO\(_2\) (Last 30s Averaged Breath-by-Breath) for All Exercise Bouts

<table>
<thead>
<tr>
<th></th>
<th>Peak VO(_2) (L/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Passive</td>
<td>4.46 ± 0.77</td>
</tr>
<tr>
<td>Active</td>
<td>4.09 ± 0.40</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Peak VO(_2) (L/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Passive</td>
<td>4.47 ± 0.89</td>
</tr>
<tr>
<td>Active</td>
<td>4.02 ± 0.44</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Peak VO(_2) (L/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Passive</td>
<td>4.07 ± 0.55</td>
</tr>
<tr>
<td>Active</td>
<td>4.15 ± 0.37</td>
</tr>
</tbody>
</table>

Note. Values are means ± standard deviation.

Table 6  Group Data for Time to Fatigue in Seconds for All Exercise Bouts

<table>
<thead>
<tr>
<th>Trial</th>
<th>Time to fatigue (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bout 1</td>
</tr>
<tr>
<td>PRP</td>
<td>126.4 ± 24.1</td>
</tr>
<tr>
<td>ARP</td>
<td>121.5 ± 24.2</td>
</tr>
</tbody>
</table>

Note. PRP, passive recovery period; ARP, active recovery period.
may be secondary to localized, intramuscular activity (i.e., muscle buffering, fiber-type recruitment, or Ca²⁺ regulation).

At the time of the present study, the authors were able to locate only a few studies investigating active versus passive intramuscular acid-base recovery (24). Using phosphorus-31 magnetic resonance spectroscopy (³¹P-MRS), Sairyo et al. reported similar intramuscular pH recovery curves to the blood data presented in this article (24). However, the authors did not quantify performance between recovery bouts nor were there multiple exercise sessions, leaving associations between blood and muscle recovery profiles as they relate to performance open to interpretation. A similar intramuscular comparison was conducted recently by McAinch et al. (18). Unlike the Sairyo study, this project focused on performance indices (quantified work) between active and passive recovery conditions. Intramuscular recovery profiles were similar to the ones presented by Sairyo et al., however, recovery mode did not influence total work between bouts [similar to the performance results presented in the current study (Table 6)] (18).

Variation between exercise protocols (exercise duration, intensity, subject’s trained status, etc.) may also influence acid-base (muscle or blood) perturbation and should be addressed when comparing raw data (i.e., peak lactate values) or performance times. The designated time frame and intensity of the current protocol was implemented to similarly exhaust the relative glycolytic capacity of our subjects [2 to 3 min of high-intensity exercise (20)] during all exercise bouts. Other studies employing similar intensities but longer exercise durations have reported higher absolute lactate values during either single or multiple bout recoveries (21, 27). However, intra-study comparison is difficult due to the varying demands on the body’s energy systems (glycolytic capacity versus aerobic power). Conversely, studies presenting with multiple short duration (10 to 30 s), high-intensity exercise protocols measure various byproducts and/or subsequent replenishment of the phosphagen systems and are generally exhausted prior to glycolytic activity (1, 4, 7).

In addition to the varying energy demands between studies, the length of recovery time prescribed between exercise bouts must also be considered. Due to the relative trained state of the subjects participating in our study, the length of recovery time may have influenced the subsequent exercise bouts, regardless of the mode of recovery (Table 6 and addressed further in Training Specificity and Recovery, below). However, the original objective of this project was profiling acid-base recovery kinetics within the confines of exhaustive exercise (110% of workload maximum). Through previous work conducted in our lab, we determined that the minimal duration necessary for profiling and comparing H⁺ and lactate recovery (T₀.₅) from such exercise was 12 min (22). A shorter recovery (< 12 min) would not provide adequate time for the assessment of slope and recovery (T₀.₅) data (H⁺, lactate, HCO₃⁻, and BE), and therefore would not aid in understanding the acid-base recovery kinetics after multiple bouts of exhaustive glycolytic activity (22).

**Recovery Kinetics (Second Function Slope)**

The premise for measuring the slope of the metabolite concentrations during recovery (blood pH, lactate, HCO₃⁻ and BE) was based upon previous work conducted in our lab that illustrated differences between recovery kinetics for single bout exhaustive exercise (22). Under prolonged recovery conditions (60 min), blood
lactate recovery was slow compared to blood pH, with almost triple the half time to recovery ($t_{0.5}$) (~ 30 min vs. 12 min) (22). In the present study, however, the unexpected two-phase recovery for all variables (Figures 1 and 2) limited the number of data points for the second recovery phase and, in turn, widely influenced the second slope. With relatively few data points, we were unable to show significant variation between slopes for pH and lactate (as the previous study indicated). Trends, however, suggest differences between recovery modes, as the active recovery slopes were generally steeper during all three recovery stages for blood pH (Table 4, Figure 1). The slopes for the other three variables, lactate, HCO$_3$– and BE, varied greatly and no trends were easily discernable.

**Training Specificity and Recovery**

Performance times and recovery slopes between trials may have been influenced by the length of time prescribed for recovery (12 min). However, a more likely scenario may be the trained status of our subjects. Subjects were all considered highly trained (55.7 ± 6.5 mL · kg$^{-1}$ · min$^{-1}$), but due to subject recruitment policies, training variation was not controlled. Of the 10 subjects, three were conditioned road cyclists, four were competitive mountain bikers, and three were actively competing in cycle cross. The metabolic demands of these sports differ, and the preceding training may have altered the method of buffering [combination of cellular and pulmonary-related alterations in proton buffering potential—as further illustrated in the variations of HCO$_3$– and BE (Table 3)].

As intramuscular acidosis and fiber-type profiling was not the aim of this study, we are only able to speculate as to the cause of the variability. However, of the 10 subjects, the mountain and cycle cross trained individuals generally projected the least amount of recovery variability (single subject comparison presented in Figure 3). Presumably, this would be due to the training regimens of these subjects, as both mountain and cycle cross sports require multiple high-intensity sprint bouts coincided with rapid recoveries. Other studies have reported variation in buffering capacities and subsequent removal of blood lactate between endurance and sprint trained subjects (10, 11, 12, 26), but to the authors’ knowledge, recovery profiles illustrating differences between various cycling events have not been published. Further research is necessary to determine training influence on muscle and blood buffering capacity during varying recovery modes.

**Establishing Appropriate Training Measurement Techniques**

Historically, researchers have viewed the accumulation of blood lactate as a limiting factor of intense exercise performance and recovery due to the inhibition of glycolytic enzymes and/or intermediates (5, 13, 15, 23). A more critical view of the glycolytic pathway however, reveals that the production of lactate through the lactate dehydrogenase (LDH) reaction actually consumes a proton [H$^+$], therefore working to alleviate acidosis during intense exercise (23). The question arises then as to the relevance of measuring and using blood lactate during multiple bouts of intense exercise as a predictor of performance. Excessive lactate spillover in the
blood from initial exercise bouts may make basing training protocols on lactate levels difficult under such conditions. In applying this question, Lau et al. examined the effect of active versus passive recovery and subsequent lactate removal on skating performance (16). The investigation concluded that there was no difference in performance (seven-shift skating test) between recovery conditions, regardless of lactate removal (16). A similar profile was evident in the current study (Figure 2), and with limited changes in performance during multiple bouts (regardless of lactate levels), basing training protocols on lactate levels under such conditions may be irrelevant.
Conclusions

Although blood acid-base recovery was enhanced by an active recovery mode, performance times during the second and third exercise bouts remained similar. The results of the current study question the relevance and applicability of measuring blood acid-base perturbation during exhaustive glycolytic exercise, and suggest that perhaps intramuscular activity during such exercise may be more reflective of buffering potential. The results also suggest that training specificity may influence time to recovery during multiple bout exhaustive exercise independent of recovery mode (active or passive).

References