Induced lactacidemia does not affect postexercise $O_2$ consumption

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ROTH, DAVID A., WILLIAM C. STANLEY, AND GEORGE A. BROOKS. Induced lactacidemia does not affect postexercise $O_2$ consumption. J. Appl. Physiol. 65(3): 1045-1049, 1988.—To study the effects of circulatory occlusion on the time course and magnitude of postexercise $O_2$ consumption ($VO_2$) and blood lactate responses, nine male subjects were studied twice for 50 min on a cycle ergometer. On one occasion, leg blood flow was occluded with surgical thigh cuffs placed below the buttocks and inflated to 200 mmHg. The protocol consisted of a 10-min rest, 12 min of exercise at 40% peak $O_2$ consumption ($VO_2$ peak), and a 28-min resting recovery while respiratory gas exchange was determined breath by breath. Occlusion (OCC) spanned min 6-8 during the 12-min work bout and elicited mean blood lactate of 5.2 ± 0.8 mM, which was 380% greater than control (CON). During 18 min of recovery, blood lactate after OCC remained significantly above CON values. $VO_2$ was significantly lower during exercise with OCC compared with CON but was significantly higher during the 4 min of exercise after cuff release. $VO_2$ was higher after OCC during the first 4 min of recovery but was not significantly different thereafter. Neither total recovery $VO_2$ (gross recovery + net recovery) nor excess postexercise $VO_2$ (net recovery $VO_2$ above an asymptotic base line) was significantly different for OCC and CON conditions (13.71 ± 0.45 vs. 13.44 ± 0.61 liters and 4.93 ± 0.26 vs. 4.17 ± 0.35 liters, respectively). Manipulation of exercise blood lactate levels had no significant effect on the slow ("lactacid") component of the recovery $VO_2$.

AT THE ONSET OF EXERCISE, the $O_2$ uptake ($VO_2$) by an isolated muscle or by the whole organism lags behind the mechanical power output and rate of energy transduction, thus giving rise to an "$O_2$ deficit." When the exercise is constant and submaximal, $VO_2$ increases with time until it reaches a steady level. When exercise is suddenly terminated, $VO_2$ decreases rapidly at first, giving rise to a fast, "alactacid" component, then, more slowly, approaching the preexercise level asymptotically (slow, "lactacid" component) (15). These two recovery volumes together have traditionally been called the "$O_2$ debt" but are more descriptively referred to as the "excess postexercise $VO_2$" (EPOC) (9) or "recovery $O_2$" (18). Regardless of terminology, the EPOC volume was originally hypothesized to represent the quantity of $O_2$ used by muscle and liver to oxidize 20% of the lactate formed during exercise, thus providing energy for reconversion of the other 80% of the lactate back to its precursor, glycogen.

If this were to hold true, any manipulation of blood lactate pool size should govern both temporal and quantitative declines in $VO_2$ during recovery. Segal and Brooks (16) demonstrated that glycogen-depleted subjects had significantly lowered blood lactate concentrations during and after exercise but had unaltered recovery $VO_2$ compared with glycogen-sufficient controls. Increased blood lactate concentrations may be induced by circulatory occlusion of exercising limbs (1). Thus the purpose of this study was to further investigate the relationship between blood lactate and the EPOC in humans by observing the effects of experimentally induced lactacidemia on the slow component of the recovery $VO_2$ curve.

METHODS

Nine healthy male subjects volunteered to exercise three times in the early morning (6:30-8:00 A.M.) in the postabsorptive state (>8-h fast). Subjects did not perform vigorous exercise during the 36-h period preceding the tests. The protocol was approved by the University of California, Berkeley, Committee for the Protection of Human Subjects (no. 83-10-48), and signed informed consent was obtained. A physical description of subjects is given in Table 1. Subjects first performed a progressive continuous test on a Quinton Uniwork 844 electrically braked cycle ergometer for determination of peak $VO_2$ ($VO_2$ peak) as previously described (19). In previous studies from our laboratory (19) utilizing circulatory occlusion during cycle ergometry, it was found that the work load that elicited $VO_2$ of 40% $VO_2$ peak was a manageable work load for subjects to complete the 2 min of exercise with circulatory occlusion.

Subjects completed two 50-min exercise tests within 10 days. The order of the performance of the two tests was alternated among subjects. Both control (CON) and occlusion (OCC) experiments consisted of a 10-min resting period, a 12 min exercise period, and a 28 min resting recovery, all while the subjects were seated on the ergometer. Before the initial rest period began, electrocardiogram leads were taped to the chest and monitored in the $V_6$ position; gas exchange headgear was adjusted and attached, and surgical thigh cuffs (20 cm wide) were placed high around the tops of both thighs, below the buttocks, and taped to the subjects' shorts to prevent slippage. The entire cuff was then wrapped with duct tape to ensure homogeneous inflation to 200 mmHg.
with in 6 s via a tank of compressed air. Exercise began at min 10 of all experiments at the prescribed work load and continued through min 22. In OCC tests, cuff inflation was initiated 6 s before min 16 (Figs. 1 and 2) and remained constant for exactly 2 min, when cuffs were deflated within 2 s. Subjects continued pedaling for the remaining 4 min of the work period (min 18-22), then stopped exercise to begin the 28-min resting recovery period seated on the cycle ergometer. With the exception of cuff inflation, the exercise protocol and blood withdrawal schedule were identical in CON and OCC experiments (Figs. 1 and 2).

Twenty 100-μl blood samples per test were taken by finger stick using sterilized microlancettes on cleansed prewarmed digits and were immediately deproteinized in 1 ml of cold 8% HClO₄. Blood weights and optical density measurements were made in subsequent enzymatic assays for blood lactate using the method of Hohorst (4).

Respiratory gas exchange was monitored breath by breath by a previously described on-line system (19). All input channels were sampled at 50 Hz. Calculations were made by a Digital MINC-II computer, and the values for each breath were stored on a floppy disk. Values for cardiorespiratory parameters were calculated using the technique of Beaver et al. (2). The system was calibrated with both room air and a known gas standard before every test and was validated against Tissot spirometer gas collection, as described by Stanley et al. (19).

The V̇O₂ data points for each subject and for the grouped mean were then plotted against time throughout CON and OCC experiments (Fig. 2). Recovery O₂ volumes were calculated in three different ways.

1) Total recovery O₂ consumption (TROC) is the total gross volume of O₂ consumed during the 28-min period of seated resting recovery. No base-line subtraction was made. 2) Excess postexercise V̇O₂ (EPOC₁) is the net volume of O₂ consumed after exercise during seated resting recovery above the resting, preexercise base line. The postexercise base-line V̇O₂ was calculated as 28 times the mean resting V̇O₂. 3) EPOC₂ is the net volume of O₂ consumed after exercise during seated resting recovery above the lowest value reached during recovery (the asymptotic recovery base line).

Two other O₂ volumes were calculated from the marked differences between the V̇O₂ during CON and OCC exercise conditions. During the 2-min OCC exercise period (mins 16-18) and the subsequent 4 min of exercise (mins 18-22) during the OCC test, we observed occlusion-induced “undershoots” and “overshoots” of V̇O₂.

Blood lactate concentration, rest, exercise and recovery V̇O₂ values (TROC, EPOC₁, EPOC₂), undershoot, and overshoot volumes were all tested for significant differences using two-tailed matched-pairs correlated t tests (P < 0.05) with 8 degrees of freedom. All results are reported as means ± SE.

RESULTS

Blood lactate concentration. At rest, mean blood lactate concentrations in CON and OCC conditions were not significantly different. At the onset of exercise, blood lactate levels increased in both conditions but were not significantly different up through min 17 (Fig. 1).

In CON experiments, exercise resulted in a significant 38% increase in blood lactate over resting to a peak of 1.13 ± 0.15 mM at min 20. After cessation of exercise at min 22, lactate concentration declined slowly, reaching preexercise values 5 min after exercise. In OCC conditions, cuff release at min 18 resulted in a 265% increase in blood lactate concentration over resting to 3.1 ± 0.38 mM. This was a 117% increase over preoccluded exercise. One minute after cuff release (min 19), the blood lactate increased to 508% over resting and 262% over preocluded exercise levels (5.17 ± 0.81 mM). Lactate concentration fell steadily from its peak at min 19 to cessation of exercise at min 22 (4.56 ± 0.53 mM). Blood lactate continued to fall steadily in recovery to min 30, then more slowly to min 50. In the OCC condition, lactate concentration never returned to base-line values during the 28-min recovery period.

After cuff release there was a significant overshoot in

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**TABLE 1. Physical characteristics of subjects**

<table>
<thead>
<tr>
<th>Age, yr</th>
<th>Wt, kg</th>
<th>V̇O₂ peak, l/min</th>
<th>Power Output, kg·m⁻¹·min⁻¹</th>
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</thead>
<tbody>
<tr>
<td>Mean ± SE</td>
<td>30.0 ± 2.05</td>
<td>78.1 ± 3.6</td>
<td>4.77 ± 0.20</td>
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<tr>
<td>Range</td>
<td>22-39</td>
<td>64.0-92.0</td>
<td>3.62-5.54</td>
</tr>
</tbody>
</table>

n = 9. V̇O₂ peak, peak O₂ consumption.

Fig. 1. Mean blood lactate concentration (±SE) plotted as a function of time under control and occluded (OCC) conditions. *Significant differences between tests (P < 0.05).

Fig. 2. Mean O₂ consumption (V̇O₂) plotted as a function of time under control and occluded (OCC) conditions. *Significant differences between tests (P < 0.05).
lactate concentration, which persisted to min 40 (18 min of recovery). At cessation of exercise, blood lactate during the OCC condition was 356% greater than during the CON condition (4.56 ± 0.53 vs. 1.0 ± 0.17 mM). Peak OCC lactate at min 19 was 379% greater than CON (5.17 ± 0.81 vs. 1.08 ± 0.34 mM, respectively). The last 10 min of recovery showed no significant difference between the two conditions.

\( \dot{V}O_2 \). The resting rates of \( \dot{V}O_2 \) were not different between CON and OCC conditions (Fig. 2, Table 2). OCC values increased in parallel with CON values at the onset of exercise, where both reached a steady-rate plateau by min 12, and were not significantly different (Fig. 2). However, with the cuff inflation at min 16, the subsequent 2 min of OCC exercise showed a significant (19%) depression in \( \dot{V}O_2 \) to 1.62 ± 0.06 l/min. The sum of these 2 min of depression from the steady-rate exercise \( \dot{V}O_2 \) plateau in the OCC condition is referred to as the occlusion-induced undershoot volume (Table 3).

Cuff release resulted in a large positive inflection of the postoccluded exercise \( \dot{V}O_2 \) to a peak of 2.65 ± 0.13 l/min at min 19; \( \dot{V}O_2 \) then declined steadily to 2.1 ± 0.8 l/min by the end of exercise (min 22). Each of the minute values for the 4-min overshoot period was statistically greater than the CON values for the corresponding time points (Fig. 2). Overshoot volumes were positively correlated with the undershoot volume: overshoot volume = undershoot volume · 1.61 ± 0.25, \( r = 0.80 \) (Table 3). The overshoot volume averaged 96.9 ± 12.4% greater than the undershoot volume.

At the cessation of exercise in the OCC condition (min 22), \( \dot{V}O_2 \) was still 12% above CON values (2.10 ± 0.08 vs. 1.87 ± 0.07 l/min) and was still elevated 6% above preclosure steady-rate exercise plateau values. It was predicted from previous studies (19) that these end-exercise \( \dot{V}O_2 \) values in OCC would return to CON or at least OCC plateau values so that nonsignificant differences in \( \dot{V}O_2 \) would exist at the onset of the recovery period. This, however, did not happen, resulting in a dissimilarity in \( \dot{V}O_2 \) values at the onset of recovery. Therefore the overshoot volumes were underestimated. The total exercise \( \dot{V}O_2 \) volume for OCC conditions was 23.88 ± 0.75 liters for the 12-min exercise bout, which was statistically greater than CON conditions (22.57 ± 0.66 liters).

During recovery from CON exercise a rapid (68%) decrease in \( \dot{V}O_2 \) from end-exercise values occurred within 2 min. Thereafter \( \dot{V}O_2 \) fell slowly toward its mean asymptotic value of 0.33 ± 0.11 l/min at 42.3 ± 1.50 min. In OCC conditions, cessation of exercise also resulted in a rapid (65%) decrease in \( \dot{V}O_2 \) from end-exercise values within 2 min. Because \( \dot{V}O_2 \) fell in this rapid component at roughly the same rate and magnitude from dissimilar start points, the first 4 min of recovery \( \dot{V}O_2 \) are significantly higher in OCC than CON. By min 27 (recovery min 5), however, there was no statistical difference between the two conditions, and, in fact, recovery min 15 showed a significantly lower \( \dot{V}O_2 \) in the OCC condition than the CON (Fig. 2), despite significantly different blood lactate concentrations at that same time point. The OCC recovery \( \dot{V}O_2 \) continued to fall slowly toward its mean asymptotic value of 0.31 ± 0.10 l/min at 43.56 ± 1.23 min. This OCC base-line \( \dot{V}O_2 \) is not significantly lower than the CON base-line \( \dot{V}O_2 \), nor are the times to asymptote significantly different.

**Total recovery \( O_2 \) consumption.** The areas under the CON and OCC \( \dot{V}O_2 \) curves of Fig. 2 from min 22 to 50 were used to calculate the TROC volumes, which were not significantly different (13.44 ± 0.61 vs. 13.71 ± 0.45 liters, respectively) (Table 2).

**EPOC\(_1\).** The EPOC\(_1\) base-line volumes were not significantly different between the CON and OCC conditions (13.71 ± 0.87 vs. 13.27 ± 0.99 liters, respectively). However, because of the high preexercise resting \( \dot{V}O_2 \) (which suggested presence of an anticipatory response), four of nine subjects in both experimental conditions exhibited negative EPOC\(_1\) volumes (Table 2).

**EPOC\(_2\).** To establish each individual's postexercise base-line \( \dot{V}O_2 \), the lowest \( \dot{V}O_2 \) during the recovery period was multiplied by the 28 min of that period, and these volumes were then subtracted from their corresponding TROC volumes. Neither the base-line volumes (9.27 ± 0.63 vs. 8.77 ± 0.35 liters) nor the EPOC\(_2\) volumes (4.17 ± 0.35 vs. 4.93 ± 0.26 liters) were significantly

<table>
<thead>
<tr>
<th>Subj No.</th>
<th>Rest, l/min</th>
<th>TROC, liters</th>
<th>EPOC(_1), liters</th>
<th>EPOC(_2), liters</th>
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</thead>
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<tr>
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<td>OCC</td>
<td>CON</td>
<td>OCC</td>
<td>CON</td>
</tr>
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<td>12.68</td>
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<td>0.418</td>
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<td>0.333</td>
<td>11.13</td>
<td>11.96</td>
</tr>
<tr>
<td>6</td>
<td>0.586</td>
<td>0.521</td>
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<td>7</td>
<td>0.505</td>
<td>0.610</td>
<td>14.15</td>
<td>13.37</td>
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<tr>
<td>8</td>
<td>0.508</td>
<td>0.426</td>
<td>12.22</td>
<td>12.59</td>
</tr>
<tr>
<td>9</td>
<td>0.652</td>
<td>0.700</td>
<td>16.91</td>
<td>16.58</td>
</tr>
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</table>

Mean ± SE 0.490±0.030 0.474±0.04 13.44±0.61 13.71±0.45 4.17±0.35 4.93±0.26

No significant differences were found between control (CON) and occlusion (OCC) conditions. \( \dot{V}O_2 \), \( O_2 \) consumption; TROC, total gross volume of \( O_2 \) consumed after exercise during seated resting recovery with no base-line subtraction; EPOC\(_1\), net volume of \( O_2 \) consumed after exercise during seated resting recovery above resting preexercise base line; EPOC\(_2\), net volume of \( O_2 \) consumed after exercise during seated resting recovery above asymptote reached during recovery.
different between the CON and OCC conditions, respectively (Table 2). The mean time to asymptote during recovery in CON conditions was 20.33 ± 1.50 min and in OCC conditions was 21.56 ± 1.23 min; these were not significantly different.

DISCUSSION

This study demonstrates a time dissociation between blood lactate concentration and \( V_{O_2} \) during recovery from exercise in human subjects. After circulatory occlusion, blood lactate concentration remained significantly (356%) elevated above CON values at exercise cessation. This large lactate load was significantly elevated to min 40 (Fig. 1). At this same time point, however, mean \( V_{O_2} \) values had become equal (Fig. 2), and mean TROC volumes to this point were not significantly different (9.30 ± 0.43 vs. 9.75 ± 0.31 liters for CON and OCC, respectively). This dissociation between blood lactate concentration and TROC was not predicted by the traditional “lactacid \( O_2 \) debt” hypothesis; i.e., if the slow lactacid component of \( O_2 \) recovery is governed by the lactate load, then larger recovery \( O_2 \) volumes should have been observed in these OCC experiments.

The induced lactacidemia and the subsequent fall in lactate concentration during the postexercise period showed no significant effect on recovery \( V_{O_2} \). Despite the significantly elevated blood lactate concentration throughout the recovery period, neither the TROC nor the EPOC volumes were significantly different between the CON and OCC conditions (Table 2). In humans (11, 18, 21), as well as other mammals (5, 13, 14, 22), reptiles, and amphibians (3, 10), a dissociation of the time course of changes in blood lactate concentration and the recovery of \( O_2 \) has been demonstrated. Results of the present investigation suggest that removal of a moderately large mass of lactate during exercise recovery, presumably through oxidation and gluconeogenesis (9), does not result in an elevation of \( V_{O_2} \).

TROC and EPOC are the summed volumes of all \( O_2 \)-derived processes of restoring metabolic homeostasis from disturbances not only in the working muscle but all organs and tissues of the body. Because \( O_2 \) is ultimately consumed in the mitochondria, direct and indirect controls of cellular respiration are inherently responsible for the EPOC. Although postexercise \( V_{O_2} \) will likely be controlled directly by intramitochondrial ADP concentration, a variety of additional factors have been shown to indirectly influence mitochondrial respiration: catecholamines, thyroxine, glucocorticoids, fatty acids, calcium ion, and temperature. In addition, calorigenic costs are seen in the elevated work of breathing and circulation (17, 21), metabolite turnover and synthesis, ion redistribution, membrane and tissue repair, and oxidation of reduced coenzymes will add to the EPOC volume over time (9). The most influential of these calorigenic processes is temperature; it directly affects mitochondrial [both skeletal muscle (7) and liver (6)] and whole-animal [both rat (5) and human (8)] \( V_{O_2} \). Additionally, Brooks et al. (5) demonstrated that after exercise, the rate of \( V_{O_2} \) in the intact animal and tissue temperatures decline in parallel. The fact that exercise utilizing large muscle mass results in a prolonged elevation in tissue temperature necessitates, as a consequence of the \( Q_{10} \) effect, that \( V_{O_2} \) be significantly elevated both during and after exercise. Hagberg et al. (11) calculated from the van't Hoff-Arrhenius equation that 60–70% of the magnitude of the slow component of their TROC volumes for 15 min was the result of this \( Q_{10} \) effect.

During exercise, every subject displayed a significantly (96.9%) larger overshoot volume after occlusion than undershoot volume during occlusion (Table 3). \( O_2 \) lack during circulatory occlusion can be interpreted at the cellular level, where \( O_2 \) transport to, and metabolite as well as heat removal from the exercising limbs are impeded. To maintain the same power output, more type II fibers may be recruited, whereas perhaps some of the slower oxidative fibers are inhibited or at least demonstrate an increasing dependence on glycolytic carbohydrate flux. Cellular \( V_{O_2} \) (\( Q_{O_2} \)) is therefore reduced, compounded by elevated venous \( P_{O_2} \), proton concentration, and tissue temperature. On cuff release, hypoxic blood with metabolites are “washed” from the contracting cells, but the effects appear to be lasting: a heightened \( Q_{O_2} \) and \( V_{O_2} \), an elevated tissue temperature, a lowered \( pH \), and a decreased mitochondrial coupling efficiency are attributable to heat accumulation (9). The inflated overshoot volumes therefore likely represent the overall recovery from physiological, mechanical, and biochemical stresses imposed at the cellular level, which are acrobically restored over several minutes after cuff release. The overshoot volumes are clearly larger than a replenishment of the \( O_2 \) lack (undershoot volume) caused by circulatory occlusion and together should not be viewed as a simple deficit-debt repayment scheme for aerobic energy credits.

At exercise cessation, the \( V_{O_2} \) values for the two conditions were not yet equal, and yet neither the subsequent TROC nor EPOC volumes were significantly different in the two conditions (Table 2). Into resting recovery, mean \( V_{O_2} \) values fell to a lower asymptotic value of 0.31 ± 0.10 l/min by min 21.56 in OCC than in CON (0.33 ± 0.11 l/min by min 20.33). Although not statistically significant, these base-line differences are magnified 28 times when

### Table 3. Undershoot \( V_{O_2} \) volumes during exercise with circulatory occlusion and overshoot volumes during exercise after occlusion

<table>
<thead>
<tr>
<th>Subj No.</th>
<th>Undershoot Volume, liters</th>
<th>Overshoot Volume, liters</th>
<th>%Difference</th>
</tr>
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<tbody>
<tr>
<td>1</td>
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<td>2</td>
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<td>79.2</td>
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<td>3</td>
<td>0.83</td>
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<td>55.4</td>
</tr>
<tr>
<td>4</td>
<td>1.05</td>
<td>2.18</td>
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</tr>
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<td>5</td>
<td>0.51</td>
<td>1.26</td>
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<td>6</td>
<td>1.00</td>
<td>1.70</td>
<td>70.0</td>
</tr>
<tr>
<td>7</td>
<td>0.53</td>
<td>1.20</td>
<td>126.4</td>
</tr>
<tr>
<td>8</td>
<td>0.51</td>
<td>1.07</td>
<td>109.8</td>
</tr>
<tr>
<td>9</td>
<td>0.82</td>
<td>1.93</td>
<td>155.3</td>
</tr>
</tbody>
</table>

Mean ± SE 0.74±0.07 1.44±0.14* 96.9±12.4

Values are given in absolute and relative terms; n = 9. Linear regression: overshoot volume = (undershoot volume) (1.61) + 0.25, r = 0.80. *Significantly different from undershoot volumes (P < 0.05).

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calculating EPOC₂ volume, which again, despite this apparent disparity, were not significantly different between the two conditions. These data cast further doubt on the traditional lactacid O₂ debt hypothesis.

The values for EPOC₁ and EPOC₂ are largely dependent on the base-line volume. In calculating EPOC₁ base lines, mean preexercise resting volumes were not significantly different for CON and OCC conditions. Similarly, in calculating EPOC₂ base lines, mean asymptotic recovery volumes were not significantly different between the two conditions. However, comparing EPOC₁ and EPOC₂ base-line volumes within each of the two experimental conditions, it is found that the preexercise resting base-line volumes are significantly greater than recovery base-line volumes (48 and 53% larger for CON and OCC, respectively). This disparity may be explained by anticipatory responses to exercise. Negative EPOC₁ volumes imply by definition that there was no "excess O₂" required in recovery; clearly the premise of EPOC₁ is suspect when applied to the experimental conditions used in this study. Therefore a preexercise resting base line should be used with caution.

Although resting base line volumes have been used in a variety of O₂ debt measurements (11, 20, 21), a variety of other base lines have also been employed (11, 14, 16, 18, 23). However, there appears to be no satisfactory way to apportion the exercise-induced biochemical and physiological disturbances to resting homeostasis. By convention, metabolic adjustments made during exercise are evaluated by means of pre- or postexercise base-line subtractions. In experiments such as the present work, where an experimental variable is changed in an otherwise identical test-retest protocol with the same subjects, the best choice for comparison of the two conditions is probably no base-line subtraction at all, i.e., our TROC volumes. Moreover, there is no reason to believe that the energy transductions supported by any base-line V₀₂ subtractions do not support recovery processes.

In conclusion, the results of this study demonstrate that 1) manipulation of exercise blood lactate concentration has no significant effect on the slow ("lactacid") component of the recovery V₀₂; 2) the practice of subtracting a resting, preexercise base line from the total recovery V₀₂ volumes should be approached judiciously; and 3) occlusion-induced undershoot and overshoot V₀₂ volumes are unequal, so that overshoot volumes represent much more than inflated "anaerobic energy credits." The time course and magnitude of oxidative recovery from exercise is therefore not governed by blood lactate concentration.

The authors thank those who served as subjects, W. R. Lee for his skillful technical assistance, and J. E. Balcom for her secretarial assistance.

This research was supported by the University of California, Berkeley, Fitness Evaluation Program. Present address of W. C. Stanley: Cardiovascular Research Institute, Univ. of California, San Francisco, and Veterans Administration Medical Center (111C1), 4150 Clement St., San Francisco, CA 94121.

Received 21 December 1987; accepted in final form 18 March 1988.

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