Impact of low-intensity isocapnic hyperpnoea on blood lactate disappearance after exhaustive arm exercise

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Objective: To investigate the impact of low-intensity isocapnic hyperpnoea (IH) on blood lactate disappearance after exhaustive arm exercise in comparison with passive and active recovery using the previously loaded muscle group.

Design: Randomised, crossover trial.

Setting: Institute for Sports Medicine.

Participants: 18 healthy non-smoking and physically active male subjects.

Interventions: Subjects performed three arm cranking tests to volitional exhaustion on 3 different days at least 48 h apart. Arm exercise was randomly followed by 30 min of passive recovery (PR), active arm cranking (AC) at 30% of peak power output or ventilatory recovery (VR) by means of IH at 30% of 12 s maximal voluntary ventilation. Blood lactate concentrations were measured every 2 min during recovery.

Main outcome measurements: Blood lactate disappearance during the three different recovery strategies.

Results: No significant differences in blood lactate concentrations were found between interventions PR, AC and VR during the whole measurement period. Mean (SD) peak lactate concentrations were 11.09 (1.98) mmol/l for PR, 11.13 (1.44) mmol/l for AC and 11.25 (1.93) mmol/l for VR. At the end of the recovery period measured lactate concentrations were 4.35 (1.56) mmol/l for PR, 3.77 (1.60) mmol/l for AC and 4.09 (1.35) mmol/l for VR. Moreover, all other variables measured were not significantly different, with the exception of higher average recovery heart rates during AC (116 (9) bpm) and VR (111 (17) bpm) compared with PR (93 (11) bpm).

Conclusion: Low-intensity IH seems not to enhance blood lactate disappearance after exhaustive arm exercise compared with passive or active recovery using the previously loaded muscle group. The magnitude of the involved muscle mass appears critical to effective active recovery.

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Exhaustive, anaerobic exercise leads to a large production of lactate and hydrogen ions in the exercising muscles, causing a concomitant decrease in intracellular pH.^{1,2} This acidosis impairs the ability for muscle contraction and glycolytic enzyme activity, leading to reduced exercise performance.² To regain optimal performance as soon as possible, fast lactic acid elimination is crucial for athletes, particularly if there are repeated, high-intensity bouts of exercise within a short period of time, such as competitions in swimming, athletics or wheelchair racing.^{3,4}

In general, blood lactate elimination is accelerated by active recovery of the previously loaded muscle group at moderate intensities.^{4–10} In practice, owing to restricted infrastructural possibilities or a densely packed schedule during a competition, active recovery is not always possible. Moreover, active recovery using the previously loaded muscle group may impair subsequent exercise performance as energy stores become additionally depleted,¹¹ early resynthesis of muscle glycogen is compromised, and time for an optimal refilling of glycogen stores is shortened.¹² This might have negative consequences for subsequent exercise performance, especially in sports or situations where it is not possible to use another muscle group for active recovery purposes (eg, leg exercise after upper body exercise) as is the case in athletes with an injured spinal cord.

Type I fibres can metabolise lactate,^{13–15} and as respiratory muscles mainly consist of type I fibres,^{16–18} we hypothesised that respiratory muscles may have the potential to enhance blood lactate elimination. If this is the case, respiratory muscles might be used as an easy to handle tool to accelerate blood lactate elimination without affecting glycogen resynthesis in previously exhausted limb muscles. This study aimed at investigating the

impact of low-intensity isocapnic hyperpnoea (IH) on blood lactate disappearance after exhaustive arm exercise and at comparing these data with results of conventional active and passive recovery protocols. An acceleration of blood lactate elimination by active recovery strategies (moderate arm cranking exercise and IH) was expected compared with that of passive recovery.

SUBJECTS AND METHODS

Subjects

Eighteen trained, male, non-smoking subjects participated in the study. Their average (SD) age was 30 (5) years, height 178 (7) cm, weight 72 (9) kg, weekly physical training volume 5.6 (3.3) h and peak oxygen uptake ($\text{VO}_{2\text{peak}}$) for arm cranking 42 (7) ml/min/kg. Subjects were asked to perform no strenuous exercise and eat food rich in carbohydrates the day before testing and to abstain from caffeine intake on the test days. The study was approved by the ethics committee of Canton Luzern, Switzerland. Written informed consent of the subjects was obtained before the start of the study.

Experimental procedure

Preliminary testing

Two preliminary sessions preceded the three main test trials. During the first preliminary session, standard spirometric data,

Abbreviations: AC, arm cranking; FVC, forced vital capacity; FEV_1 , forced expiratory volume in 1 s; HR, heart rate; IH, isocapnic hyperpnoea; MVV, maximal voluntary ventilation; PEF, peak expiratory flow; PR, passive recovery; $\text{VO}_{2\text{peak}}$, peak oxygen uptake; VR, ventilatory recovery; VT, ventilatory threshold

including forced vital capacity (FVC), forced expiratory volume in 1 s (FEV₁), peak expiratory flow rate (PEF) and maximal voluntary ventilation (MVV) over 12 s were obtained by an ergospirometric device (Oxycon Alpha, Jaeger, Hoechberg, Germany). Afterwards, a familiarisation trial for IH by partial rebreathing from a bag was completed (Spirotiger, Idiad, Volketswil, Switzerland). Bag size corresponded to about one-third of subjects' FVC. Subjects were breathing at a preset respiratory frequency to reach a target minute ventilation (see below).

The second preliminary session consisted of 30 min continuous IH at a minute ventilation corresponding to 30% MVV and ensured that subjects could sustain this load over 30 min. To assure the predetermined target ventilation, tidal volume, breathing frequency and end tidal partial pressure for carbon dioxide were monitored by means of the above-mentioned metabolic cart. If necessary, subjects were guided to keep preset tidal volume and breathing frequency to achieve the target ventilation.

Main experiments

All three main tests started with a 2 min resting period for baseline data determination. Subjects were seated in a chair connected to an arm cranking (AC) ergometer (Ergometrics 800 SH, Ergoline, Bitz, Germany). After this resting period, subjects started AC at 20 W. Then, the workload was increased 5 W every 20 s until subjects' volitional exhaustion. Respiratory variables at rest and during the exercise tests were sampled breath by breath through a face mask by an Oxycon Pro (Jaeger, Würzburg, Germany). This device was calibrated for gas and volume according to the manufacturer's recommendations immediately before each test. The three main tests were at least 48 h apart.

During AC, the pedal axis of the ergometer was aligned with the shoulder joint axis and subjects were positioned such that the elbow was slightly flexed at maximal reach. Five minutes after the end of the test, subjects followed one of three recovery strategies in a randomised order. These strategies were passive recovery (PR), active recovery by AC or ventilatory recovery (VR) by means of IH for 30 min. While subjects sat relaxing on a chair for PR, they were either arm cranking at 30% of the previously reached maximal workload (AC) or breathing at 30% MVV with the Spirotiger device (VR) as an active recovery strategy.

Capillary blood for enzymatic lactate analysis (Super GL Ambulance, Ruhrtal Labor Technik, Möhnesee, Germany) was sampled from an earlobe at rest, immediately after cessation of the exercise test and every 2 min until the end of the recovery phase. Heart rate (HR) was determined by an HR monitor (Polar S610, Polar, Kempele, Finland) throughout the whole test and recovery period. Immediately after cessation of the AC exercise test, subjects were asked to rate their perceived exertion by means of a Borg scale, with 6 indicating "no" and 20 "maximal" exhaustion.¹⁹

Statistics

Data points of the measured lactate concentrations of the three different recovery strategies were fitted to the following bi-exponential curve, as described in detail elsewhere²⁰:

$$La(t) = La(0) + A_1 \cdot (1 - e^{-\gamma_1 t}) + A_2 \cdot (1 - e^{-\gamma_2 t})$$

La(t) denotes the time-dependent lactate concentration with La(0) being the lactate concentration at the start of recovery. The form of this equation suggests that the lactate kinetics during recovery can be described by two main processes: one with a high-velocity constant (γ_1) describing the appearance ($A_1 > 0$) of lactate in the bloodstream and the other, with a low-velocity constant (γ_2) describing its disappearance ($A_2 < 0$).

The variables were calculated using a commercially available computer software package (SYSTAT, version 10, SPSS Inc, Richmond, California, USA) with the regression method of least mean squares.

An analysis of variance for repeated measures was applied to compare γ_1 and γ_2 between the different intervention strategies (PR, AC, VR). In addition, VO_{2peak} , peak power, rating of perceived exertion as well as maximal and average recovery HRs were analysed in the same way. If significance was found, a post hoc test with Bonferroni correction was used to locate significant differences. Results are given as mean (SD). Values were considered to be significantly different if $p < 0.05$.

The ventilatory threshold (VT) of the exercise test preceding AC was calculated from carbon dioxide output (VCO_2) and VO_2 values according to the V-slope method.²¹

RESULTS

The mean of the subjects' FVC was 5.8 (1.1) litre (115% predicted), FEV₁ 4.6 (0.7) litre (109% predicted), PEF 11.4 (1.4) l/s (119% predicted) and MVV 206.8 ± 30 l/min (140% predicted). Peak power output, VO_{2peak} , maximal HR and rating of perceived exertion did not differ significantly between tests (table 1).

Figure 1 shows blood lactate curves. The low-velocity constants (γ_2) describing blood lactate disappearance were 0.052 (0.031)/min, 0.072 (0.035)/min and 0.053 (0.032)/min for PR, AC and VR respectively ($p = 0.138$), indicating that blood lactate elimination rate constants after exhaustive arm ergometry were not significantly different among the three different recovery strategies. Average recovery HRs during AC (115.9 (9.2) bpm) and VR (111.3 (16.8) bpm) were significantly higher than the average HR during PR (92.8 (10.5) bpm).

Average workload during AC was 44.2 (7.3) W, corresponding to 29.9 (1.0)% of peak power output. This workload was clearly below the VT at 55.8 (14.5)% of VO_{2peak} , which corresponded to an absolute workload of 88.1 (30.0) W.

During VR subjects were ventilating 61.6 (9.3) l/min (29.8 (0.9)% of MVV) at an average breathing frequency of 26.2 (3.7)/

Table 1 Values measured at the end of exhaustive arm cranking tests before starting different recovery strategies

Values measured	Subsequent recovery strategy		
	Passive	Arm cranking	Isocapnic hyperpnoea
Peak power (W)	148.1 (24.7)	147.8 (22.7)	146.9 (23.0)
Peak oxygen uptake (ml/min/kg)	41.7 (7.3)	40.6 (4.8)	41.7 (8.0)
Maximal heart rate (bpm)	175.9 (11.7)	176.2 (9.8)	176.2 (10.5)
Rate of perceived exertion (Borg)	18.3 (1.4)	18.3 (1.4)	18.2 (1.2)

Results are shown as mean (SD).

Note that there were no significant differences between tests.

Figure 1 Mean blood lactate elimination curves for different recovery strategies after an exhaustive arm cranking exercise test (AET).

min. Average end tidal partial pressure for carbon dioxide was 32.8 (5.1) mm Hg.

DISCUSSION

The main finding of this study is that there were no significant differences in blood lactate disappearance after exhaustive arm cranking exercise due to the different recovery strategies. This was surprising, as a superior impact of active recovery strategies (AC and VR) on blood lactate elimination was expected compared with PR. Different reasons may explain this observation and are discussed below.

AC for active recovery

Active recovery at moderate intensities by means of the previously loaded muscle group is known to accelerate blood lactate elimination compared with PR.^{4–10} Although AC showed a nearly 40% faster low-velocity constant (γ_2) than PR and VR, no significant differences ($p = 0.138$) were found between the different recovery strategies. This might be owing to the relatively large interindividual differences between subjects (fig 1) but may be caused for other reasons discussed below.

In contrast to the above mentioned studies where leg exercise was applied, we used an arm cranking exercise for recovery. Slower blood lactate kinetics for arm exercise compared with leg exercise were also reported by Thiriet *et al.*²² Obviously, the magnitude of the affected muscle mass is critical for effective active recovery. Arm muscle mass in a group of 29-year-old men was found to be only 40% (7.1 kg vs 17.7 kg) of leg muscle mass.²³ Taking into account the higher proportion of type I fibres,^{16–24} combined with the bigger muscle mass involved during leg exercise, it seems not surprising that lactate elimination from arm muscles would be different from that of leg muscles. The large proportion of type II fibres in arm muscles^{16–24} limits their ability to oxidise lactate itself. In combination with the relatively small muscle mass of the arms, lactate elimination is additionally compromised compared with elimination from leg muscles.

Moreover, if the chosen intensity for AC had been too high, lactate elimination would have decelerated. However, considering the measured mean recovery HR of 116 bpm during AC compared with other active recovery HRs reported in the literature,^{4–7–22} it seems very unlikely that our recovery intensity of 30% of maximal power output was too high. Moreover, a recovery load of 30% of maximal power output was suggested to be within the optimal range for active recovery and has been

successfully applied in the past.^{22–25–26} Further, the chosen recovery workload was far below the VT, which supports the assumption of an optimal active recovery intensity.

However, the present active recovery design by means of AC (instead of using previously unloaded leg muscles) was chosen, as there are situations where it is not possible to use another muscle group for active recovery purposes as is the case, for example, in athletes with an injured spinal cord.

VR for active recovery

With regard to the type of muscle fibre in the arm versus respiratory muscles, one can hypothesise that VR is better than AC and PR in accelerating blood lactate disappearance. However, this assumption was not supported by the present findings. Although respiratory muscles mainly contain type I fibres,^{16–18} the total muscle mass affected in VR is quite small and thus oxidative capacity is limited. In fact, diaphragmatic muscle mass was found to be only 262 g²⁷ compared with the above-mentioned 7 kg of arm muscle mass.²³ Even if during VR the auxiliary respiratory muscles worked actively, this seems to be a very small active muscle mass to engage in lactate oxidation, which might be the main reason why the VR strategy failed to enhance blood lactate elimination.

A further reason might be that the chosen intensity of IH at 30% MVV was too low for a sufficient effect on lactate metabolism over 30 min. On the other hand, a higher intensity of IH might have led to respiratory muscle fatigue, which compromises subsequent exercise performance.^{28–29} In fact, Martin and coworkers showed that intense IH at 66% of the mean MVV reduces subsequent running performance.²⁹ Additionally, the possibility has to be taken into account that during upper body exercise, some of the respiratory muscles of the rib cage have to partake in non-ventilatory functions.³⁰ Thus, a recovery strategy like IH, integrating all respiratory muscles, should be performed at a moderate intensity, to avoid the development of rib cage muscle fatigue, which otherwise might compromise subsequent AC performance. The fact that average recovery HR during VR was close to the HR values during AC and significantly higher than during PR (table 1) suggests that the chosen intensity of IH at 30% MVV was adequate and comparable to the intensity during AC. Hence, it is questionable if a higher intensity of IH (eg, 40–50% MVV) would provide faster lactate removal without causing respiratory muscle fatigue.

Perhaps specifically trained respiratory muscles would have provided faster blood lactate elimination. This assumption is supported by the finding of Spengler *et al.*, who demonstrated lower blood lactate concentrations at the end of an exhaustive cycling exercise after 4 weeks of IH training.³¹ It was suggested by the authors that the decrease in blood lactate concentration was probably caused by enhanced lactate uptake of the trained respiratory muscles. In contrast, others observed no changes of blood lactate concentrations after respiratory muscle training.³² As the respiratory muscles of our study subjects were not specifically trained, it would be interesting to see if there were differences in the potential for lactate elimination after a respiratory training period. Further investigations are needed to answer this question.

Preliminary exercise and lactate elimination

It has been shown that the intensity of preliminary exercise influences the kinetics of subsequent lactate elimination³³ and thus might falsify results. Data of the three arm cranking tests to exhaustion preceding the three recovery interventions refute this assumption for the present study. No differences were found in peak power, $\dot{V}O_{2peak}$, maximal HR or peak lactate concentration between tests (table 1 and fig 1), which implies that the preceding exhaustive exercise was comparable for each testing session.

What is already known on this topic?

Blood lactate elimination is enhanced using a previously loaded large muscle group for active recovery.

What this study adds?

The magnitude of the affected muscle mass seems to be critical to effective active recovery as we found no significant acceleration of blood lactate elimination using either arm or respiratory muscles for active recovery.

CONCLUSIONS

Low-intensity IH seems not to enhance blood lactate disappearance after exhaustive arm exercise compared with passive or active recovery with the previously loaded muscle group. The magnitude of the affected muscle mass seems to be critical to an effective active recovery as shown by the fact that AC also failed to enhance blood lactate elimination significantly.

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Competing interests: None.

Some results of this study were presented at the 11th Annual Congress of the European College of Sport Science and therefore published in abstract form in *Schweiz Zeitschr Sportmed Sporttraum* 2006;**54**:69.

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COMMENTARY

Sport competitions often involve repeated high-intensity bouts followed by brief recovery periods. Active recovery using jogging or cycling is a typical recovery strategy repeatedly shown to accelerate blood lactate disappearance. However, low-intensity exercise using large muscles as in cycling or jogging may not be practical between competitions. The authors have therefore examined the effectiveness of using arm cranking as well as voluntary overventilation to activate additional muscle mass with the goal of taking up and metabolising blood lactate. In both cases, the working muscle mass employed to take up circulating lactate is smaller than that activated by traditional leg exercise. The findings here suggest that these alternative strategies do not activate sufficient muscle mass to achieve the goal of rapid lactate elimination. This information is important for athletes and coaches in competitive tournament settings. Large muscle mass is required for effective active recovery.

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