Heart rate recovery during repeated high-intensity efforts is not related to the recovery of systemic oxygen uptake and muscle oxygenation, and can't be used to predict performance decrement

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Headline

Practitioners are always seeking for non-invasive, easy-to-use indices of exercise tolerance to adjust training contents (e.g., work intensity and volume, number of repetitions, recovery interval duration before the next effort) during a training session. Hear rate (HR) recovery (HRR) during high-intensity interval training (HIIT) sessions has been empirically used to regulate training. For example, the return of HR to a fixed value or percentage of maximal HR is sometime used in the field and in the scientific literature in an attempt to individualize between-effort recovery duration (1, 2). The present understanding of the determinants of HRR suggest, however, that this practice may not be very relevant (3). During the post-exercise recovery period, HR is believed to neither be related to systemic O_2 demands nor muscular energy turnover (4), but rather to the magnitude of the central command and metaboreflex stimulations (6-8). However, whether HRR can effectively be used to track between-efforts metabolic recovery and performance capacity during an actual HIIT session has surprisingly received little attention in the literature (9).

Aim. The first aim of the present study was to examine the association between changes in HRR during a HIIT session and the changes in two measures of metabolic recovery (i.e., oxygen uptake and muscle oxygenation recovery, likely reflective of muscle PCr recovery (10, 11)). The second aim of the study was to examine the capacity of changes in HRR during a HIIT session to predict performance capacity.

Methods

Athletes. Data from ten male cyclists (28.3 ± 5.1 yrs, 181.4 ± 4.1 cm, 77.5 ± 7.3 kg, training 8 ± 2 hours a week and with a maximal oxygen uptake of 60.1 ± 5.9 ml/min/kg), who participated in a previoulsy published study (5) were analyzed. The study conformed to the recommendations of the Declaration of Helsinki.

Design. Observational, lab-based research.

Methodology. All methods have been presented previously (5). The HIIT session consisted of 6 x 30-s all out cycling sprints, interspersed with 2 min of passive recovery (5). Participants were asked to sprint as fast as possible from the start. Average power output was determined for each sprint. Percentage of power decrement ($\%Dec_{Power}$) was calculated as: 100 - [(Sum of average power output during each sprint/Ideal power output) x 100]; where Ideal power output = the number of sprints x best average power output. Oxygen uptake (i.e., $\dot{V}O_2$) and HR were collected during all tests using an automated breath-by-breath system (Medgraphics CPX Gas Analysis System; St. Paul, MN) coupled with a polar transmitter belt (T31, Polar Electro, Kempele, Finland). The NIRS apparatus (Niromonitor NIRO-200, Hamamatsu Photonics, Japan) used in this study was a 3-wavelength continuous wave system, which simultaneously uses the modified Beer-Lambert and spatially resolved spectroscopy methods. Because of uncertainty in the differential path length factor (DPF) for the quadriceps muscle, DPF was not used in the present study. The NIRO-200 provides a direct measure of tissue hemoglobin O_2 saturation [tissue oxygenation index, TOI = $[HbO_2]/([HbO_2] + [HHb])$ x 100, expressed in %], calculated independently using the spatially resolved spectroscopy method and multidistance source detector approach. TOI reflects the dynamic balance between O_2 supply and O_2 consumption and is independent of near-infrared photon pathlength in muscle tissue – its changes post exercise have been used as a marker of muscle PCr recovery (10, 11). HRR, $\dot{V}O_2$ recovery and TOI recovery were calculated over the 2-min post-exercise period (i.e., the difference between the value observed immediately after exercise cessation and 2 min later). For the correlation analyses, individual sprint values of HRR, $\dot{V}O_2$ recovery and TOI recovery were expressed as deltas (e.g., ΔHRR) from the mean value observed across the 6 repetitions within each subject. The percentage of HRR decrement ($\%Dec_{HRR}$) was calculated as $\%Dec_{Power}$.

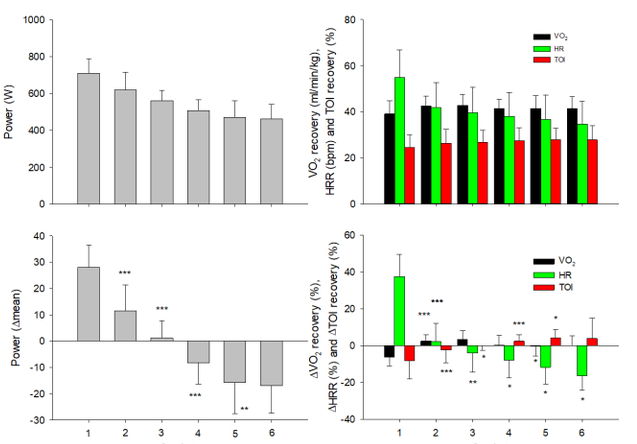

Fig. 1. Change in cycling power, oxygen uptake ($\dot{V}O_2$) recovery, heart rate recovery (HRR) and tissue oxygenation index (TOI) recovery throughout the six sprints shown both in absolute values (upper graphs) and as difference from the mean response across the six sprints. Values are mean ± standard deviation. ***: large difference from the preceding sprint, **: moderate difference from the preceding sprint, *: small difference from the preceding sprint.

Analyses

Data in the figures are presented as means with standard deviation or 90% confidence limits (CL). Between-sprint repetitions standarized differences in cycling power, $\dot{V}O_2$ recovery, HRR and TOI recovery were assed using common thresholds: >0.2 (small), >0.6 (moderate), >1.2 (large) and very large (>2) (13). The magnitude of the correlations between all variables were also assessed using the following thresholds: < 0.1, trivial; <0.1-0.3, small; <0.3-0.5, moderate; <0.5-0.7, large; <0.7-0.9, very large; and <0.9-1.0, almost perfect. If the 90% confidence intervals overlapped small positive and negative values, the magnitude was deemed unclear; otherwise the magnitude was deemed to be the observed magnitude (13).

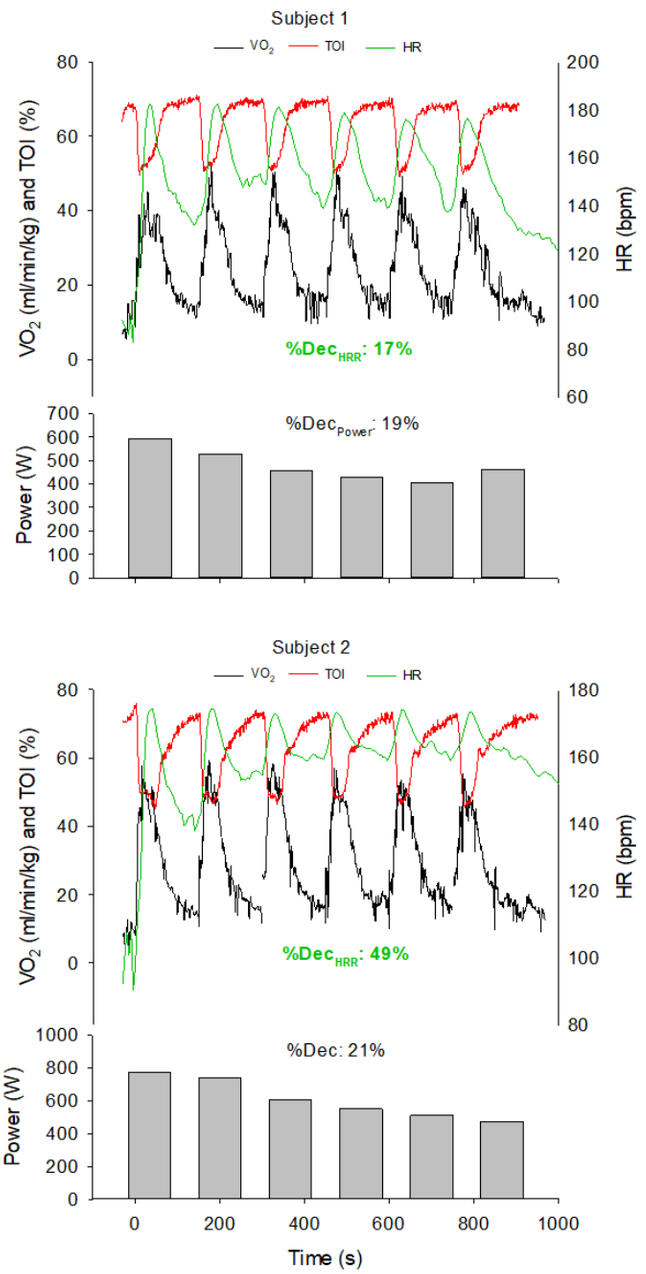

Fig. 2. Changes in oxygen uptake ($\dot{V}O_2$), heart rate (HR), tissue oxygenation index (TOI) and cycling power throughout the six sprints in two representative subjects. Note the difference between the 2 subjects in the progressive slowdown of HR recovery throughout the sprint repetitions (%Dec$_{HRR}$), despite a similar percentage of power decrement (%Dec$_{Power}$) – and with both subjects showing a preserved $\dot{V}O_2$ and TOI recovery.

Results

The average %Dec$_{Power}$ was 22% ± 5, %Dec$_{HRR}$ was 24% ± 11. In parallel to the progressive decrease in cycling power output throughout the sprint repetitions, there was a tendency for an improved $\dot{V}O_2$ and TOI recovery – which was at odds with the progressive slowdown of HRR (Figure 1). Interestingly also, the magnitude of the slowdown of HRR (%Dec$_{HRR}$) was subject-dependent (SD = 11%), despite very consistent %Dec$_{Power}$ (Figure 2). There was no clear correlation between %Dec$_{Power}$ and %Dec$_{HRR}$ (0.14 (-0.67;0.8)). The correlations between changes in HRR and changes in either $\dot{V}O_2$ or TOI were highly individual and small in magnitude (Figure 3, left panels). Changes in HRR and changes in cycling power were largely correlated (Figure 3, upper right panel). The correlations between changes in cycling power and changes in either $\dot{V}O_2$ or TOI were highly individual and small-to-moderate in magnitude (Figure 3, lower right panel).

Discussion

The poor association between the changes in HRR and changes in the measures of metabolic recovery (i.e., oxygen uptake and muscle oxygenation recovery, reflective of muscle PCr recovery (10, 11)), together with the fact that participants showed various magnitudes of HRR impairments throughout the sprints despite similar %Dec$_{Power}$ (Figure 2), confirms that using HRR as an index of readiness to perform the following interval during HIIT doesn't seem to have a clear physiological rationale. The lack of association between HRR and metabolic recovery markers (Figure 1 and Figure 3, left panels) is likely related to the fact that during the post-exercise recovery period, HRR is rather related to the magnitude of the central

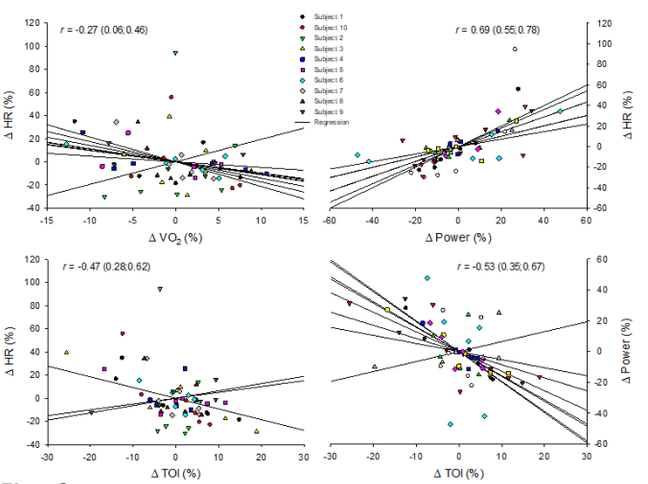

Fig. 3. Relationship between changes in heart rate (HR) recovery and oxygen uptake ($\dot{V}O_2$) recovery throughout the six sprints (upper left panel), changes in HR recovery and tissue oxygenation index (TOI) recovery (lower left panel), changes in HR recovery and cycling power (upper right panel), and changes in TOI recovery and cycling power (lower right panel). While regression lines are shown for each individual, correlation coefficient (90% confidence intervals) are provided for all subjects pooled together. The negative correlation between the changes in cycling performance and $\dot{V}O_2$ recovery (-0.26 (0.05;0.45)) is not shown.

command and metaboreflex stimulation (6-8), independently of the actual metabolic (oxidative) recovery. This understanding is also consistent with the large correlation (Figure 3, upper right panel) found between the reductions in both HRR and cycling power throughout the sprints. In fact, this correlation doesn't reflect a cause-to-effect mechanism (i.e., there was no clear correlation between $\%Dec_{Power}$ and $\%Dec_{HRR}$), but is likely explained by the fact that the accumulation of metabolites that are associated with peripheral fatigue (14) directly stimulate the metaboreflex in the blood (6-8), and in turn, affect HRR. Finally, the negative associations between the changes in cycling performance and $\dot{V}O_2$ or TOI recovery (Figure 3, lower right panel, i.e., worse sprinting performances observed with greater TOI recovery!) confirms also that mechanisms other than metabolic recovery are of greater importance when it comes to repeating high-intensity efforts interspersed with "long" (>90s) resting periods (e.g., impairments in neural drive and motor unit activation, or metabolites accumulation may better explain the performance decrement commonly witnessed during repeated-high intensity efforts - for review see (15)).

Practical Applications

- HRR is not related to metabolic (oxidative) recovery during HIIT.
- The use of changes in HRR as an index of readiness to perform the following interval during HIIT doesn't seem to have a clear physiological rationale (at least in terms of oxidative metabolic recovery, which is itself unlikely to be the most important factor when it comes to repeating high-intensity efforts with prolonged recovery).
- The monitoring of HRR during HIIT should be seen as an indirect means to assess the anaerobic glycolytic contribution to each interval bout (with the slower the HRR, the lower the blood pH) – but not obligatorily, performance capacity.
- If efforts are maximum, the monitoring of performance decrement is enough to directly assess the level of neuromuscular fatigue.

Limitations

- Neuromuscular performance was limited to cycling power, which precluded the examination of the mechanisms of the fatigue observed.

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Dataset

Dataset available on SportPerfSci.com

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