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Perceptual and Physiological Responses to Recovery from a Maximal 30 s Sprint

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Brief running head: Perceptual responses in recovery

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ABSTRACT

The aims of this study were to evaluate perceptions of post-exercise recovery and to compare patterns of perceived recovery with those of several potential mediating physiological variables. 17 well-trained men (age: 22 ± 4 years; height: 1.83 ± 0.05 m; body mass: 78.9 ± 7.6 kg; and body fat: $11.1 \pm 2.2\%$) completed 10 sprint trials on an electromagnetically-braked cycle ergometer. Trial 1 evaluated peak power via a 5 s sprint. The remaining trials evaluated: a) the recovery of peak power following a maximal 30 s sprint using rest intervals of 5, 10, 20, 40, 80, and 160 s; b) perceived recovery via visual analogue scales; and c) physiological responses during recovery. The time point in recovery at which individuals perceived they had fully recovered was 163.3 ± 57.5 s. Power output at that same time point was $83.6 \pm 5.2\%$ of peak power. There were no significant differences between perceived recovery and the recovery processes of $\dot{V}O_2$ or minute ventilation (\dot{V}_E). Despite differences in the time-courses of perceived recovery and the recovery of power output, individuals are able to closely predict full recovery without the need for external timepieces. Moreover, the time-course of perceived recovery is similar to that of $\dot{V}O_2$ and \dot{V}_E .

Key words: Perceived exertion, Wingate, sprinting, power output.

INTRODUCTION

Over the last 50 years a considerable amount of research has been conducted into the development of a ratio scale for evaluating perceived exertion during exercise. The 15-point scale developed by Borg (1970) has received the greatest attention in various forms of exercise and across a variety of populations. Whilst the scale was developed to additionally provide an indication of heart rate, the association between perception of effort and exercise intensity follows an exponential pattern, the mechanisms of which involve the complex integration of various central and peripheral signals (Borg, 1982). Although the relative importance of those central and peripheral components of perceived exertion remain largely elusive, if those perceptions can be used reliably to indicate the physical strain associated with exercise, then it is possible that those same signals could also be used to evaluate recovery after exercise. If so, perceived recovery could be an invaluable tool for regulating interval training where the magnitude of recovery between work bouts determines the overall training stimulus and the subsequent adaptive responses.

Whilst the evaluation of perceptual responses during short-term post-exercise recovery has already received some attention (Allen & Pandolf, 1977; Noble, 1979; Robertson, Nixon, Caspersen, Metz, Abbott, et al., 1992; Swank & Robertson, 2002), the evaluation of those perceptions has been determined using perceived exertion scales designed to evaluate perceptions of effort during exercise. Moreover, none of the aforementioned investigations linked perceptual responses to the recovery of exercise performance. The aims of the present study were therefore: a) to evaluate the pattern of perceived recovery and to compare perceived recovery with the recovery of power output; and b) to compare the pattern of perceived recovery with those of

several physiological variables which have been implicated as potential mediating factors in perceptual responses (Hampson, St Clair Gibson, Lambert, & Noakes, 2001).

METHODS

Experimental Approach to the Problem

During the 10 week period of investigation, participants completed 10 physiological trials, at approximately the same time of day, with an average of 7 ± 4 days between each. All trials were completed in an air-conditioned laboratory maintained at a constant temperature of 18°C. Participants were instructed to maintain their normal diet throughout the testing period, to avoid food and drink in the hour before testing, and to avoid strenuous exercise 24 hours prior to each trial. Trial 1 was a baseline trial to establish peak power output over 5 s and to familiarise participants with the equipment and the demands of a 30 s maximal cycle sprint test. Trials 2 – 7 involved participants performing a 30 s maximal sprint followed by a predetermined stationary rest period and a subsequent 5 s sprint to determine the recovery of peak power output. Trials 8 and 9 were used to establish individual perceptions of recovery following a 30 s maximal sprint, and the extent to which individuals had recovered when they perceived they were fully recovered. Trial 10 involved the evaluation of various physiological variables during recovery from a 30 s sprint in an attempt to explain individual perceptions of recovery. Trials 2 – 7 were randomized, with trials 8 and 9 included in the randomisation process after participants had experienced two of the experimental trials. In effect, it was felt important for participants to experience the test before evaluating perceptions of recovery. Trial 10 was the final trial of the investigation.

Subjects

Seventeen well-trained male strength & conditioning and sport science students volunteered for the study which was approved by Ethics Committee. Prior to testing, participants received written and verbal instructions regarding the nature of the investigation and completed a training history questionnaire, which indicated that all had been actively involved in sport for approximately 14 years. Times spent training and competing each week were reported as 8.8 ± 4.9 hours and 5.0 ± 3.8 hours, respectively. Prior to commencement of the study, all participants completed a health-screening questionnaire and provided written informed consent. Means \pm standard deviation for age, height, body mass, and estimated body fat (Durnin & Womersley, 1974) of the participants were: 22 ± 4 years, 1.83 ± 0.05 m, 78.9 ± 7.6 kg, and $11.1 \pm 2.2\%$ respectively.

Equipment

All sprints were performed on an electro-magnetically braked cycle ergometer (Lode Excalibur Sport, Groningen, Holland), which was fitted with standard pedals, toe-clips, and straps, and interfaced with a computer to enable high-frequency logging of the flywheel angular velocity. Perceptions of recovery were recorded using a 20 cm visual analogue scale (VAS) ranging from 'not at all recovered' to 'completely recovered'. Core temperature was monitored using a tympanic thermistor probe (Model CD, Edale Instruments (Cambridge) Ltd., Longstanton, UK). Blood lactate was evaluated from capillary puncture using an automated analyser (Biosen C-Line, EKF Diagnostic, Ebendorfer Chaussee 3, Germany). The analyser was calibrated before all trials in accordance with the manufacturer's instructions. Heart rates were monitored at 5 s intervals using heart rate monitors (Polar S610, Polar Electro Oy,

Kempele, Finland). All respiratory measures were made from expired air (breath-by-breath) using an on-line gas analyser (Jaeger Oxycon Pro, Hoechberg, Germany). The analyser was calibrated before each test using oxygen and carbon dioxide gases of known concentrations (Cryoservice, Worcester, UK) and the flowmeter was calibrated using a 3-litre syringe (Viasys Healthcare GmbH, Hoechberg, Germany). During the tests participants breathed room air through a facemask (Hans Rudolph, Kansas City, MO, USA) that was secured in place by a head-cap assembly (Hans Rudolph, Kansas City, MO, USA).

Procedures

Trial 1

On arrival at the laboratory, height, body mass, and estimated body fat (determined from the sum of four skinfolds) were recorded for each subject. Participants then performed a four-minute warm-up on the cycle ergometer at a power output of 100 W. The same warm-up procedure was used for all trials. The saddle height and handlebar position for each subject were determined before the first trial and remained constant for all subsequent trials. On completion of the warm up and starting from a stationary position, participants performed a series of 3×5 s maximal cycle sprints interspersed with three-minute stationary rest periods to determine individual measures of peak power output. A torque factor of $0.7 \text{ Nm}\cdot\text{kg}^{-1}$ was used for all sprint trials and participants were verbally encouraged to give maximal effort. On completion of the third sprint, participants cycled for a further three minutes at a power output of 100 W before performing a 30 s maximal cycle sprint for familiarisation purposes. After all trials, participants completed a cool-down by cycling at 100 W for a minimum of five minutes.

Trials 2 – 7

After the warm-up, and from a rolling starting power output of 100 W, participants completed a 30 s maximal sprint. On completion of the sprint, participants were instructed to remain stationary on the ergometer for a period of between 5 s and 160 s before performing a 5 s maximal sprint. Information on the duration of the recovery period was withheld from the subject in every trial and the computer screen was obscured from view. Since it was anticipated that the recovery of peak power output would likely follow a bi-phasic pattern (Bogdanis, Nevill, Boobis, Lakomy, & Nevill, 1995), the following recovery periods were used: 5 s, 10 s, 20 s, 40 s, 80 s, and 160 s.

Trials 8 and 9

In trials 8 and 9, participants followed the same procedure as in trials 2 – 7 up to the point at which they completed the 30 s sprint. In trial 8, on completion of the 30 s sprint, participants remained stationary on the ergometer and were asked to indicate, by placing a mark on the VAS at the same time points used for trials 2 – 7, the extent to which they felt they had recovered their ability to perform a subsequent 5 s sprint. To prevent visual feedback from influencing the results, a fresh VAS was used for each time point in the recovery process. In addition, participants were asked to indicate at what point in the recovery process they felt they had fully recovered. In trial 9, participants completed the same procedure as in trial 8, with the additional element of performing a maximal 5 s sprint at the time point in recovery at which they had previously indicated that they felt they had fully recovered.

Trial 10

In trial 10, following the fitting of the face mask and headgear, the tympanic thermistor, and the heart rate monitor, participants were asked to remain stationary on the ergometer for a period of three minutes to enable baseline physiological measurements to be recorded. After a further four-minute warm-up period, participants performed a 30 s maximal sprint followed by a five-minute recovery period during which the following physiological measurements were recorded: heart rate, blood lactate, core temperature, oxygen uptake ($\dot{V}O_2$), minute ventilation (\dot{V}_E), and breathing frequency. Blood lactate and core temperature measurements were made at 40 s intervals during the recovery period.

Statistical Analysis

All statistical analyses were conducted using the Statistical Package for the Social Sciences (SPSS for Windows, SPSS Inc., Chicago, IL). Measures of centrality and spread are presented as means \pm standard deviation. The possibility of learning or training effects influencing the outcome of the experiment was evaluated by conducting a one-way ANOVA on peak and mean power output in the 30 s sprints, in trial order. Synchronisation of the gas analysis data between participants was achieved using linear interpolation at 5 s intervals throughout recovery after eliminating values that were outside four standard deviations of the midpoint of a rolling 20 breath mean (attributed to 'noise') (Rossiter, Ward, Kowalchuk, Howe, Griffiths, et al., 2002). Differences in perceptions of recovery between trials 8 and 9 were evaluated using a two-way ANOVA, with mean values from each time point subsequently used to investigate the pattern of the recovery process. The recovery data from all the physiological variables were converted to percentages, with values at the end of the

30 s sprint used as the reference point for zero recovery, and with mean resting values from the start of Trial 10 used as the reference for full recovery. The recovery of peak power was also determined as percentage data, with peak power from the 5 s sprints in Trial 1 considered as the reference for full recovery. Differences between perceptions of full recovery and the recovery of power output at the same time point were evaluated using a Wilcoxon matched-pairs test. Differences between perceived recovery and both power output and physiological recovery were evaluated using two-way ANOVA tests with repeated measures on both factors. α was set at 0.05 for all analyses. Significant effects were followed up using Bonferroni-adjusted *post hoc* analyses. Non-significant effects were followed up by applying monoexponential models to characterise the kinetics of the corresponding recovery response for each individual using a non-linear least-squares fitting procedure (XLfit, IDBS Ltd, Guildford, UK). Models were developed using the same approach previously used for off-transient phosphocreatine (PCr) and $\dot{V}O_2$ recovery kinetics (Rossiter et al., 2002): $\Delta X_{(t)} = X_0 + \Delta X_{(ss)}(1 - e^{-t/\tau})$; where X is the physiological variable concerned, t = time, $\Delta X_{(ss)}$ is the asymptotic value to which X projects, and τ is the time constant of the response (note: since in all cases, recovery at time point zero was zero, the first term on the right hand side of the equation was redundant). Resultant time constants were subsequently compared using Pearson correlations.

RESULTS

Sprint performance

There was no significant effect of trial order on values of peak ($F_{(4,34,69,51)} = 1.572, p = 0.187$) or mean ($F_{(8,128)} = 1.453, p = 0.181$) power output in the 30 s sprints (grand means: 960 ± 146 W and 729 ± 86 W, respectively).

Perceived recovery versus the recovery of power output

There were no significant differences between trials 8 and 9 on perceptions of recovery following the 30 s sprint ($F_{(1,16)} = 4.350$, $p = 0.056$). The patterns of perceived recovery and the recovery of power output, including the results of the *post hoc* analysis, are presented in Figure 1. The time in recovery at which individuals perceived they had fully recovered was 163.3 ± 57.5 s, at which point, power output was $83.6 \pm 5.2\%$ of peak power. In effect, individuals significantly ($p < 0.001$) underestimated full recovery by 16.4% (95% likely range: 13.7 to 19.0%). Analysis of the data revealed a significant effect of variable ($F_{(1,16)} = 16.99$, $p < 0.001$), time ($F_{(2.5,40.3)} = 299.75$, $p < 0.001$), and variable \times time ($F_{(2.7,42.8)} = 11.68$, $p < 0.001$).

Perceived versus cardiopulmonary recovery

The recovery patterns of the various cardiopulmonary factors are presented in Figure 2, with patterns of actual recovery presented in Figure 3. There was a significant effect of time ($p < 0.001$) on each variable. There were also significant variable \times time interactions for $\dot{V}O_2$ ($F_{(3.07,49.19)} = 4.55$, $p = 0.006$), \dot{V}_E ($F_{(5,80)} = 20.24$, $p < 0.001$), breathing frequency ($F_{(2.21,35.32)} = 56.43$, $p < 0.001$), and heart rate ($F_{(2.27,36.32)} = 17.539$, $p < 0.001$). Significant differences between variables were only observed in analyses involving breathing frequency ($F_{(1,16)} = 120.90$, $p < 0.001$) and heart rate ($F_{(1,16)} = 50.14$, $p < 0.001$). Moreover, *post hoc* analyses were only able to detect differences in contrasts involving breathing frequency and heart rate. Time constants for perceived recovery, $\dot{V}O_2$, and \dot{V}_E were 86.2 ± 33.2 s, 61.5 ± 15.3 s, and 92.3 ± 36.3 s, respectively. Correlations between the time constants of perceived

recovery and both $\dot{V}O_2$ and \dot{V}_E were -0.10 (95% likely range: -0.56 to 0.40) and 0.23 (95% likely range: -0.28 to 0.64), respectively.

Perceived versus peripheral recovery

The patterns of perceived recovery versus the recovery patterns of blood lactate, and core temperature are presented in Figure 4, with actual blood lactate and core temperature responses presented in Figure 5. The analysis revealed significant differences between the process of perceived recovery, and those of blood lactate ($F_{(1,16)} = 14.22, p = 0.002$) and core temperature ($F_{(1,16)} = 121.74, p < 0.001$). There was a significant effect of time for perceptual responses and core temperature in recovery ($F_{(1,49,23.76)} = 15.91, p < 0.001$). Significant interactions were observed between the patterns of perceived recovery and those of blood lactate ($F_{(1.01,16.21)} = 13.81, p = 0.002$), and core temperature ($F_{(2,32)} = 12.44, p < 0.001$). *Post hoc* analyses revealed significant differences between all contrasts (See Figure 4).

DISCUSSION

The aims of this study were to evaluate post-exercise perceptions of recovery and to compare the pattern of those perceptions with the recovery patterns of several potential mediating physiological variables. The results revealed significant differences between the patterns of perceived recovery and the recovery of peak power output. In effect, individuals significantly underestimated recovery in the early stages of the process, with the two patterns converging as time progressed. Nevertheless, the results revealed a relatively small (given the absence of any external reference of elapsed time), but significant underestimation of the time to full recovery. In a recent investigation it was established that individuals were able to maintain

performance in a multiple sprint test (12 × 30 m) when left to choose their own between-sprint recovery durations (Glaister, Witmer, Clarke, Guers, Heller, et al., 2010). Moreover, after completion of the first two sprints, the duration of those self-selected recovery periods was not significantly different within individuals. Whilst the underestimation of full recovery in the present study appears to conflict with these findings, the 30 s sprint in the present study was designed to largely deplete PCr stores (Walter, Vandenborne, McCully, & Leigh, 1997). In contrast, the 5 s sprints used by Glaister et al. (2010) would only partially reduce PCr stores and as such, any slight underestimation of full recovery would be unlikely to affect peak power output, at least in the early stages of the protocol. Indeed, the idea of a slight underestimation of full recovery in the Glaister et al. (2010) investigation may explain why the duration of perceived recovery was adjusted (lengthened) by the participants following completion of the first two sprints.

The pattern of perceived recovery was similar to that observed in studies which have investigated perceptual responses during recovery using perceived exertion scales (Allen & Pandolf, 1977; Noble, 1979; Swank & Robertson, 2002). Although Robertson et al. (1992) noted a more linear response, the authors also found a similar non-linear pattern when perceptual responses were constrained to feelings of strain associated with ventilatory effort. Whilst it is difficult to say whether perceptions of recovery are the same as those derived using perceived exertion scales, the similarities between the two processes combined with the fact that ratings of perceived exertion do not return to baseline immediately upon cessation of exercise suggests that they may be.

Despite the absence of any significant differences between the recovery kinetics of perceptual responses and those of $\dot{V}O_2$ and \dot{V}_E , the time constants of the corresponding monoexponential recovery kinetics were poorly correlated. Moreover, although *post hoc* tests were unable to detect any significant differences, the results suggest that time was affecting the degree of similarity between perceptual and both $\dot{V}O_2$ and \dot{V}_E kinetics. Previous research into the relationship between perceptual responses during recovery and $\dot{V}O_2$ have shown that, despite similarities in recovery patterns, the two processes appear to be unrelated since their kinetics become dissociated under conditions of induced-alkalosis (Swank & Robertson, 2002) and hyperoxia (Allen & Pandolf, 1977). Indeed, research into the link between $\dot{V}O_2$ and perceptual responses during exercise suggests that, despite evidence of a positive relationship ($r = 0.76$ to 0.97), particularly when $\dot{V}O_2$ is expressed as a percentage of $\dot{V}O_{2max}$ (Sargeant & Davies, 1973; Skinner, Hutsler, Bergsteinova, & Buskirk, 1973), $\dot{V}O_2$ is unlikely to directly influence perceptual responses since its kinetics cannot, it appears, be consciously monitored (Mihevic, 1981). In contrast, \dot{V}_E , which has also been shown to strongly correlate ($r = 0.61$ to 0.94) with perceived exertion during high (greater than approximately 70% $\dot{V}O_{2max}$) (Robertson, 1982), rather than low-intensity exercise (Edwards, Melcher, Hesser, Wigertz, & Ekelund, 1972; Cafarelli & Noble, 1976) and, in some instances, with perceptions of strain in recovery (Robertson et al., 1992; Swank & Robertson, 2002), may well explain recovery perceptions. Previous research comparing perceptions of exercise intensity with \dot{V}_E has suggested that the strong correlation between the two variables at high exercise intensities is due to afferent feedback from mechanoreceptors associated with the recruitment of ancillary muscles of respiration (Robertson, 1982). Since these

same muscles remain highly activated during the early stages of recovery, the same process may also explain the link between \dot{V}_E and perceived recovery. Moreover, the reduced activation of the aforementioned ancillary muscles as recovery progresses may explain the non-significant widening gap between \dot{V}_E and perceptual responses over time and as such explain the variable \times time interaction. However, if \dot{V}_E can explain perceptual responses, aside from a possible homogeneity effect and a limited number of data points with which to model the perceptual responses, it is difficult to reason why their respective time constants were poorly correlated.

Although the link between perceptual responses and \dot{V}_E appears to hold for both exercise and recovery, the same does not appear to be true for breathing frequency. Previous research examining the relationship between perceptual responses and respiratory variables during exercise has reported similar correlations between perceptions of exertion and breathing frequency as those reported for perceptual responses and \dot{V}_E (Pandolf, Cafarelli, Noble, & Metz, 1972; Noble, Metz, Pandolf, & Cafarelli, 1973; Kamon Pandolf, & Cafarelli, 1974). Conversely, whilst similar patterns of breathing frequency and \dot{V}_E have been observed in recovery after exercise (Allen & Pandolf, 1977; Robertson et al., 1992; Swank & Robertson, 2002), none of the studies observed a significant relationship between perceptual responses and breathing frequency. However, previous research into perceptual responses during recovery utilized end-exercise intensities $\leq \dot{V}O_{2\max}$ as the starting point for recovery. In contrast, the present study used a much higher exercise intensity in order to provide a more complete description of the recovery process. As a result, the rapid decline in breathing frequency in the early stages of recovery, in comparison to the much

steadier decline in \dot{V}_E , shows a clear disparity between the kinetics of the two processes.

It is difficult to say whether the cues involved in perceptions of recovery are different from those used in perceptions of effort, although the results from the breathing frequency and heart rate data suggest that this may be the case. However, the relationship between heart rate and perceived exertion during exercise is far from as certain as the original work by Borg (1970) and subsequent others (Sargeant & Davies, 1973; Skinner et al., 1973; Stamford, 1976) have suggested. For instance, the association between heart rate and perceived exertion has been shown to break down as a result of various environmental and pharmacological interventions (Ekblom & Goldbarg, 1971; Pandolf et al., 1972; Kamon et al., 1974; Davies & Sargeant, 1979). In effect, since the relationships between perceptions of effort and both breathing frequency and heart rate are far from clearly established, the perceptual cues that determine the extent of recovery could very well be the same as those used to determine levels of exertion.

The disparity between the recovery kinetics of perceptual responses compared with those of blood lactate, contrasts with the large number of studies which support a significant positive relationship ($r = 0.61$ to 0.77) between perceived effort and blood lactate during both exercise (Edwards et al., 1972; Gamberale, 1972; Morgan & Pollock, 1977) and recovery (Allen & Pandolf, 1977; Robertson et al., 1992; Swank & Robertson, 2002). However, the lower exercise intensities used, coupled with the lack of frequent sampling (≤ 3 samples per investigation), raises concerns regarding the validity of the assumptions drawn in previous recovery-based investigations. Indeed,

Swank and Robertson (2002) highlight that the single measure of blood lactate obtained 5 minutes into their recovery protocol represents a limitation to their conclusions. Concerns also exist regarding the link between perceived effort and blood lactate during exercise since, as with \dot{V}_E , the lack of any appreciable accumulation of blood lactate below lactate threshold means that the relationship only appears to hold true for higher exercise intensities. Once again, whilst the results of various experimental interventions add support to a blood lactate/perceived effort relationship (Ekblom & Goldbarg, 1971; Gamberale, 1972; Allen & Pandolf, 1977; Boutcher, Seip, Hetzler, Pierce, Snead, et al., 1989; Hetzler, Seip, Boutcher, Pierce, Snead, et al., 1991; Haskvitz, Seip, Weltman, Rogol, & Weltman, 1992; Swank & Robertson, 2002), others report contradictory findings (Stamford & Noble, 1974; Löllgen, Graham, & Sjogaard, 1980; Staab, Agnew, & Siconolfi, 1992). If the cues for perceptual responses are the same in exercise and recovery, then the results of the present study clearly show that blood lactate is not a causal factor. However, the contrast between the above findings may simply be a reflection of the fact that muscle, rather than blood, lactate is the influential cue in the perceptual response. In effect, the increase in blood lactate during the recovery period represents the time lag between production, efflux, diffusion, and sampling. In contrast, since anaerobic glycolysis shuts down on cessation of exercise, the corresponding decline in muscle lactate could be a mediating factor in the perceptual response. Then again, the much slower decline of muscle lactate relative to that of PCr (Sahlin, Harris, & Hultman, 1979; Walter et al., 1997) suggests that neither muscle nor blood lactate is related to the perceptual response.

Finally, whilst previous research has suggested a possible influence of core temperature on perceptual responses, it appears that any such influence only occurs under extreme environmental conditions when the ability to defend any rise in core temperature is compromised (Noble et al., 1973; Kamon et al., 1974). In the present study, the absence of any notable change in core temperature during recovery, in contrast to the relatively major changes in perceived recovery, supports the view that core temperature has no influence on perceptual responses under normal (neutral) environmental conditions (Mihevic, 1981; Hampson et al., 2001).

PRACTICAL APPLICATIONS

The results of the present study show a clear disparity between perceptions of recovery and the recovery of power output. Although those same patterns of recovery converge as time progresses, individuals tend to underestimate the time to full recovery. From a practical perspective, coaches and athletes need to be aware of the above and adjust recovery periods accordingly if perceived recovery is to be used to regulate interval training performance. In the end, as with perceptions of effort, it is difficult to reconcile the physiological cues which regulate perceptions of recovery. However, if perceptual cues are the same for exercise and recovery then, based on the findings of the present study, it is difficult to make a case for influential factors other than $\dot{V}O_2$ and \dot{V}_E .

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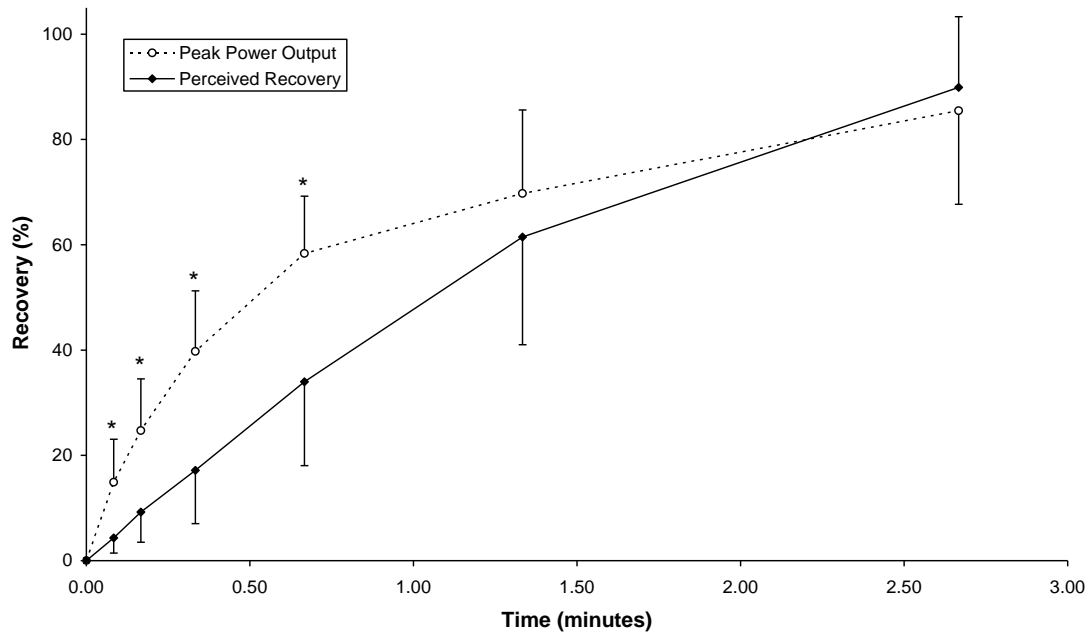


Figure 1. The recovery patterns of peak power output and perceived recovery following a maximal 30 s cycle sprint ($n = 17$). Values are means; bars are standard deviations. *Significantly different from data at the same time point ($p < 0.05$).

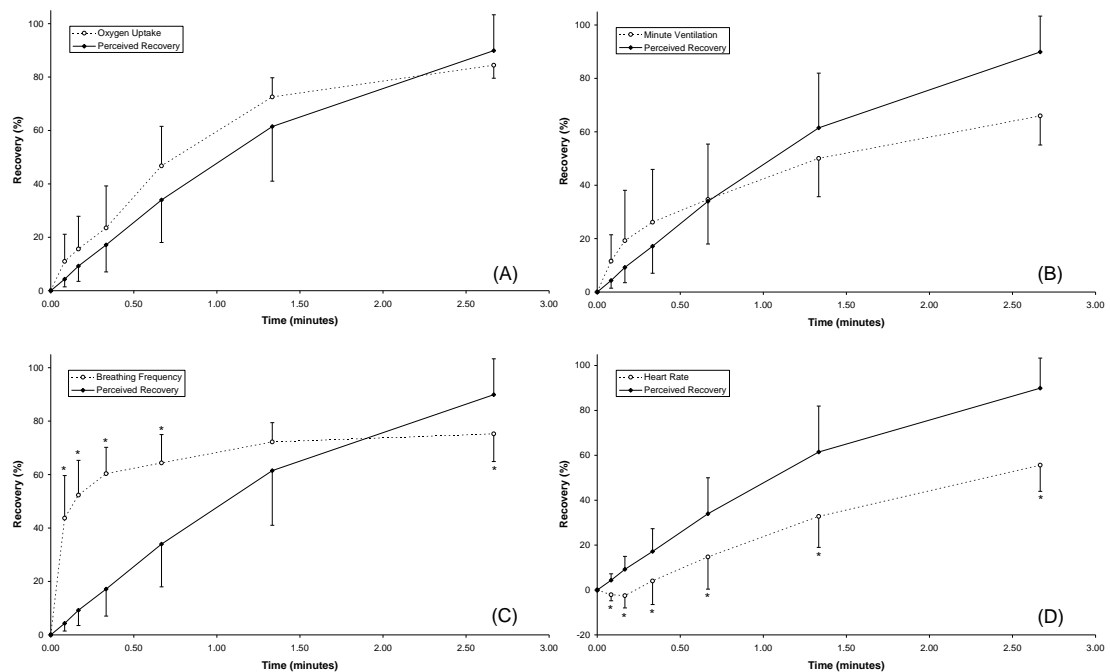


Figure 2. Comparisons between perceived recovery and the recovery processes of oxygen uptake (A), minute ventilation (B), breathing frequency (C) and heart rate (D)

following a maximal 30 s cycle sprint ($n = 17$). Values are shown as percentages of full recovery (derived from resting data) to allow direct comparisons between variables. Values are means; bars are standard deviations. *Significantly different from data at the same time point ($p < 0.05$).

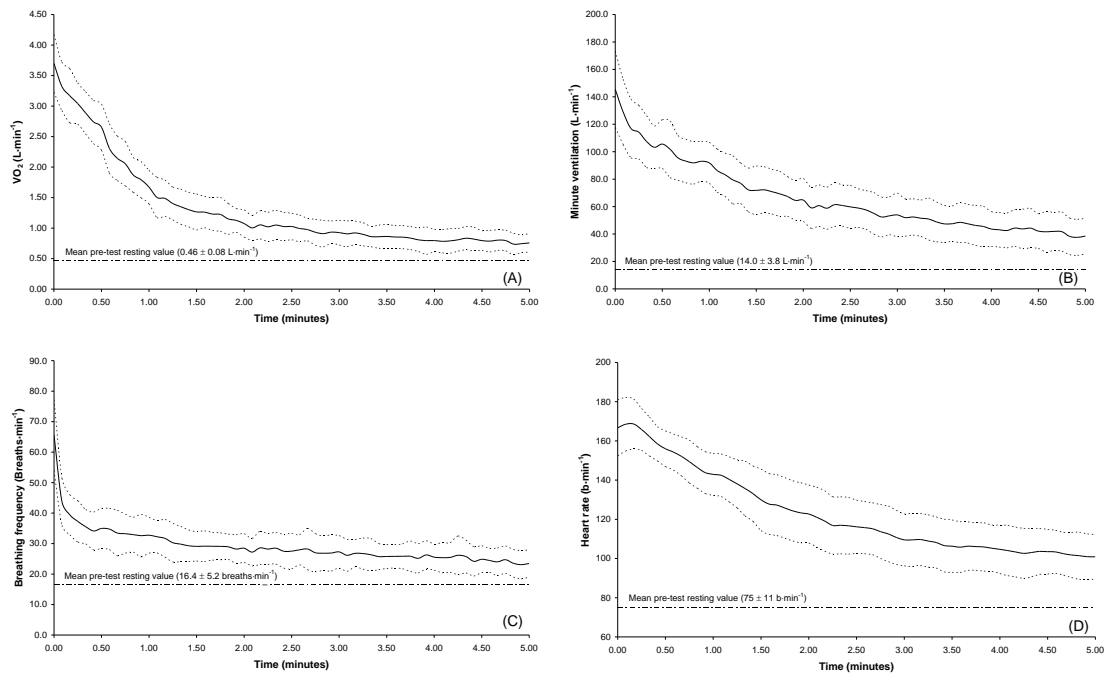


Figure 3. Oxygen uptake (A), minute ventilation (B), breathing frequency (C) and heart rate (D) during five minutes of recovery following a maximal 30 s cycle sprint ($n = 17$). Solid lines are means; dashed lines are standard deviations.

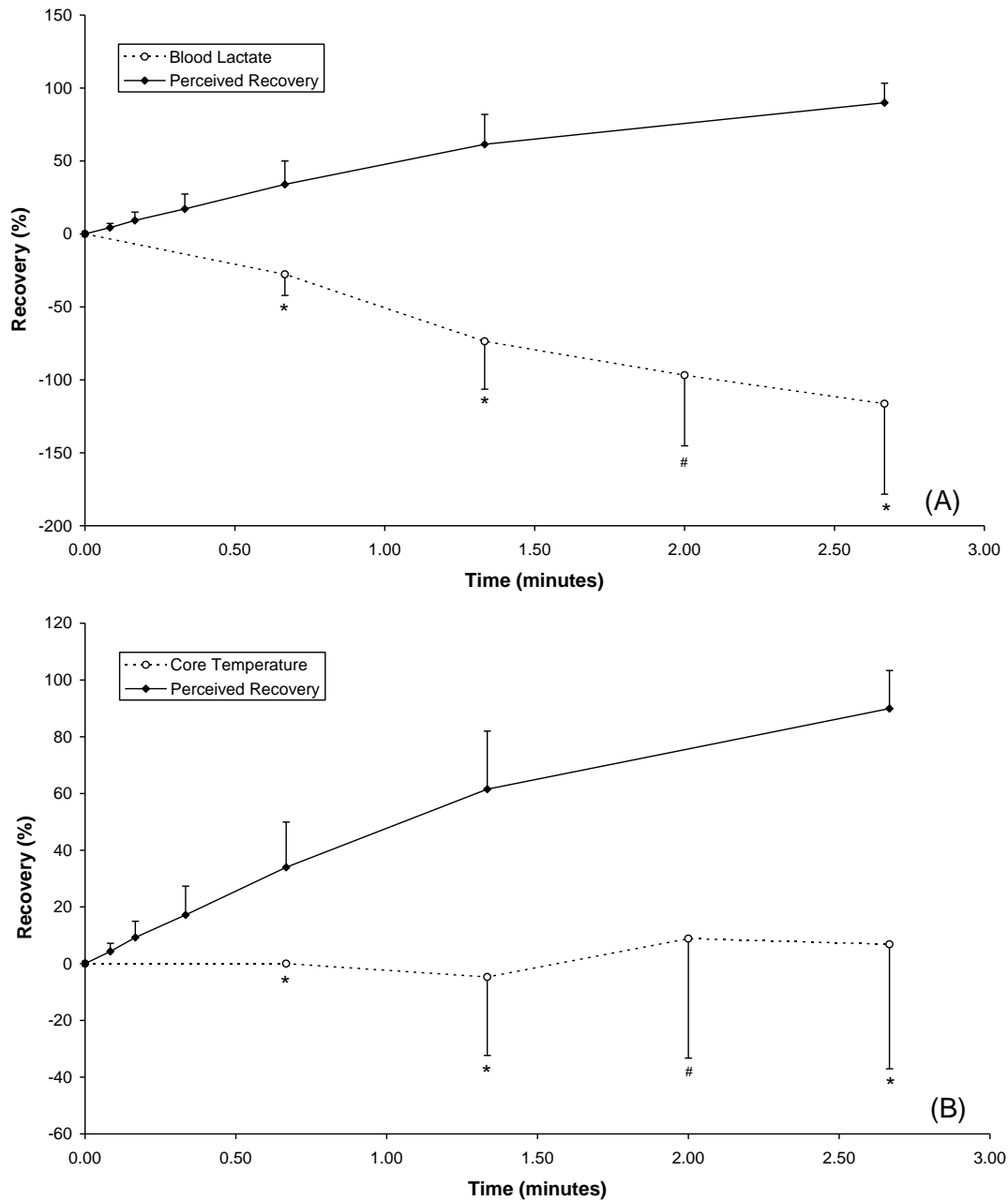


Figure 4. The patterns of perceived recovery and those of blood lactate (A) and core temperature (B) during recovery following a maximal 30 s cycle sprint ($n = 17$). Values are means; bars are standard deviations. *Significantly different from data at corresponding time point ($p < 0.05$). #No direct comparison made.

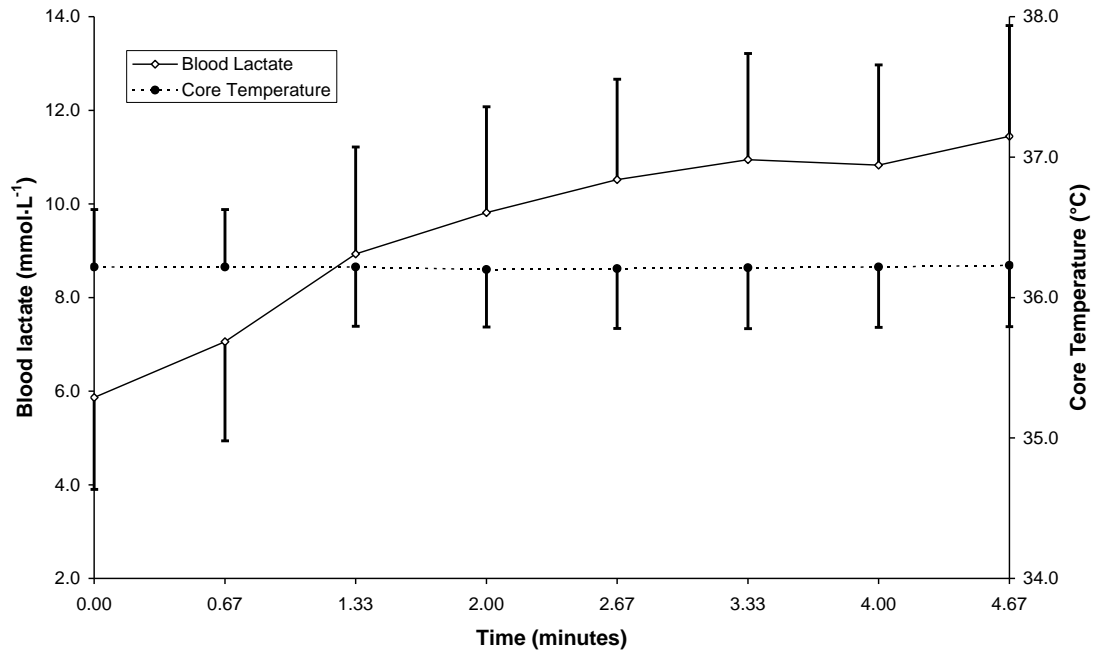


Figure 5. The recovery patterns of blood lactate and core temperature following a maximal 30 s cycle sprint ($n = 17$). Values are means; bars are standard deviations.