Complex Interplay Between Determinants of Pacing and Performance During 20-km Cycle Time Trials

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Purpose: This study examined the determinants of pacing strategy and performance during self-paced maximal exercise. Methods: Eight well-trained cyclists completed two 20-km time trials. Power output, rating of perceived exertion (RPE), positive and negative affect, and iEMG activity of the active musculature were recorded every 0.5 km, confidence in achieving preexercise goals was assessed every 5 km, and blood lactate and pH were measured postexercise. Differences in all parameters were assessed between fastest (FAST) and slowest (SLOW) trials performed. Results: Mean power output was significantly higher during the initial 90% of FAST, but not the final 10%, and blood lactate concentration was significantly higher and pH significantly lower following FAST. Mean iEMG activity was significantly higher throughout SLOW. Rating of perceived exertion was similar throughout both trials, but participants had significantly more positive affect and less negative affect throughout FAST. Participants grew less confident in their ability to achieve their goals throughout SLOW. Conclusions: The results suggest that affect may be the primary psychological regulator of pacing strategy and that higher levels of positivity and lower levels of negativity may have been associated with a more aggressive strategy during FAST. Although the exact mechanisms through which affect acts to influence performance are unclear, it may determine the degree of physiological disruption that can be tolerated, or be reflective of peripheral physiological status in relation to the still to be completed exercise task.

Keywords: electromyography, affect, RPE, cycling

During self-paced exercise, participants display nonmonotonic changes in heart rate and power output, and frequently display an “endspurt” commencing at ~90% of trial duration. St Clair Gibson and Noakes proposed these phenomena are evidence of a control system that regulates performance in order to maintain physiological homeostasis. Exercise has been suggested to be regulated through variation of skeletal muscle recruitment in a manner that prevents physiological failure based on feedback from the peripheral systems and knowledge of the end point of exercise, a mechanism previously described as teleoanticipation. During self-paced exercise the brain continually recalculates the work rate it perceives as optimal, meaning it cycles between periods of certainty, when power output changes are initiated based on assessment of afferent signals, and uncertainty, during which there is no knowledge of how these changes have affected peripheral system function. As exercise progresses, it is suggested that periods of certainty and uncertainty become progressively shorter until eventually the brain may allow an endspurt when the distance remaining is short enough to pose no risk of loss of physiological homeostasis.

A range of physiological, psychological, and tactical factors appear relevant in determining the selection and maintenance of pacing strategies during prolonged exercise. Hulleman et al have demonstrated that “aggressive” strategies, whereby initial power outputs are higher than those predicted by previous performances, result in improved 1500-m cycle time trial performance. Similarly, Jones et al observed that an initial power output above the average that could be maintained for ~120 s, but then declined to 10% below the average, increased time to failure compared with an even pace strategy. The authors proposed this “fast-start” strategy enhanced performance by accelerating VO₂ kinetics at the onset of exercise, thereby increasing the oxidative contribution to energy expenditure and “sparing” some anaerobic capacity. Although these studies have used relatively short duration bouts of exercise, there is some evidence that more aggressive strategies can also result in improved performances in longer events. Swart et al demonstrated that over a series of 40-km cycle time trials participants adopted more “aggressive” strategies with successive trials, and these strategies involving a higher power output in the initial stages were associated with improved performances. In contrast, Mattern et al demonstrated that even-paced and “fast start” strategies during a 20-km cycle time trial were not as effective as strategies whereby power output was initially below
the average for the entire trial but then increased in the final stages. A strategy characterized by a higher power output in the earlier stages of a trial may be expected to result in greater metabolic disruption in the peripheral physiology, thereby compromising the ability to increase muscular work rate in the final 10%. This would appear to be confirmed by the study of Hausswirth et al., who found that triathletes who started a 10-km run at a pace 5% faster than that achieved in a control run produced slower overall performances than when the run was commenced at a pace 5% slower. The 5% faster strategy was also accompanied by higher values for oxygen uptake, ventilation, heart rate, and blood lactate at the end of the first kilometer. However, although starting 5% slower than control run pace resulted in superior performance compared with starting 5% faster, an initial pace that was 10% slower resulted in performances similar to those of the 5% faster strategy. Therefore, in prolonged exercise bouts, it is clear that a high degree of regulation of muscular work is required in order to maximize performance and avoid premature fatigue.

In addition to physiological parameters, the rating of perceived exertion (RPE) has been suggested to be the principle regulating mechanism of physiological function during exercise. Tucker suggests athletes alter power output throughout a trial to achieve a set RPE at intermediate points, culminating in achievement of maximal attainable RPE at the end. If this is true, it would seem that RPE at intermediate stages of a trial may predict ability to achieve an endspurt. For example, if RPE is maximal at 90% of total distance, it should be impossible to further increase work rate. Conversely, acceleration would seem possible if RPE at this point is submaximal, and the rate of increase in RPE is one that will not result in achievement of maximal values before task completion.

Although RPE is influenced by feedback from various physiological systems, there is evidence that psychological state can dissociate changes in RPE from peripheral physiology, thereby compromising the ability to increase muscular work rate in the final 10%. This would appear to be confirmed by the study of Hausswirth et al., who found that triathletes who started a 10-km run at a pace 5% faster than that achieved in a control run produced slower overall performances than when the run was commenced at a pace 5% slower. The 5% faster strategy was also accompanied by higher values for oxygen uptake, ventilation, heart rate, and blood lactate at the end of the first kilometer. However, although starting 5% slower than control run pace resulted in superior performance compared with starting 5% faster, an initial pace that was 10% slower resulted in performances similar to those of the 5% faster strategy. Therefore, in prolonged exercise bouts, it is clear that a high degree of regulation of muscular work is required in order to maximize performance and avoid premature fatigue.

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Methods

Participants

Eight well-trained individuals (6 male, 2 female, 32.6 ± 11.5 years) who were currently active in competitive endurance cycling participated in the study. All had at least 4 years of continuous training history and at the time of the study performed at least 5 training sessions per week. Full written informed consent was provided and preexercise health questionnaires were completed before participation in the procedures, which had Institutional Ethics Committee approval. Participants were asked...
to prepare for each trial as for a minor competition, by training lightly in the preceding 48 h and following usual precompetition dietary regimes.

Design

A repeated-measures experimental design was employed involving assessment of physiological and psychological variables during and following two 20-km time trials within a laboratory environment.

Procedure

Participants visited the laboratory on 2 occasions separated by 2 to 7 days (mean 5.4 ± 1.9). On each visit and following an individualized warm-up, they performed a 20-km time trial after being instructed to complete the task as quickly as possible. Participants used their own bicycles mounted onto the Kingcycle ergometry system (Kingcycle Ltd, High Wycombe, UK). The front wheel was removed and the cycle attached to the Kingcycle by the front forks and a pillar under the bottom bracket. The rear wheel was positioned on the air-braked flywheel and the velocity of its revolution monitored by a photo-optic sensor. The Kingcycle rig was interfaced to a PC equipped with Kingcycle v6.7 software, which calculated the power output (watts) the cyclist would have generated at that cadence on level ground. The system was calibrated by asking participants to reach a power output of approximately 250 W while seated in the same position as they would be during the subsequent trial. They then stopped pedaling and the height of the pillar supporting the bottom bracket was adjusted so that deceleration of the flywheel was equal to a reference power decay curve. Throughout each trial a visual display provided information relating to distance completed, elapsed time, current speed, and power output. Instantaneous power output was recorded every 0.5 km throughout each trial, along with surface EMG from three lower limb muscles, which was recorded over a 10-s period. Rating of perceived exertion, positive and negative affect, and hear rate (HR) were also assessed over the first 2 km.

No familiarization trial was included within this study protocol. As the intention was to compare differences between the faster and slower performances, we deemed it appropriate to omit a familiarization trial, which had the potential to reduce the magnitude of any differences in performance or psychological factors between trials.

Measures During Exercise

RPE. The Borg Category 20 Scale was used to record RPE throughout the trials. Participants provided a whole number response, and the scale was anchored by explaining that a score of 20 should equate to a previous memory of absolute exhaustion.

Positive and Negative Affect. Participants rated how they felt “right now” using the Worcester Affect Scale. This consists of two scales: a 10-point Likert scale for positive affect (1 = not at all positive; 10 = extremely positive) and a 10-point Likert scale for negative affect (1 = not at all negative; 10 = extremely negative).

Confidence in Achieving Goal. Participants articulated their goals before each trial. During exercise they were asked, “How confident are you that you can achieve your goal?” They then rated confidence using a 10-point Likert scale (1 = not at all; 10 = very much so).

Heart Rate. Heart rate was continually recorded throughout all trials via radiotelemetry (Polar Vanguard, Polar Electro, Finland). Heart rate values were subsequently converted to percentage of theoretical maximum heart rate.

Muscle Activity. Surface electromyography was used to monitor neuromuscular activity in the vastus lateralis, biceps femoris, and tibialis anterior muscles. Skin was prepared by shaving and cleaning with light abrasion and an alcohol swab. Electrodes (Ag/AgCl) were placed in a bipolar configuration on muscle bellies. Electrodes and wires were taped to the skin to reduce potential movement artifacts. The EMG signals were recorded using an MIE MT8 telemetry system (MIE Medical Research Ltd, Leeds, UK) of preamplifiers with a fixed gain of ×1000 at a sample rate of 2 kHz. A magnet and reed switch provided a pulse signal referencing the beginning of each pedal revolution.

Data analysis was performed using MATLAB software (Mathworks, Natick, USA). The EMG signals were rectified, and movement artifacts were removed with a high-pass second-order 15-Hz Butterworth filter and then smoothed with a low-pass second-order 5-Hz Butterworth filter.

An average integrated-EMG (iEMG) signal was calculated for each muscle at each time point during the trial based on 10 consecutive pedal revolutions. This was normalized by dividing by the average iEMG recorded over the first 2 km.

Postexercise Measures

Blood Analysis. Postexercise fingertip capillary blood lactate concentration was measured using the Analox GB7 analyzer (Analox Instruments Ltd., London UK), and blood acid–base status was assessed using the Radiometer NPT7 blood gas analyzer (Radiometer Medical, Bronshoj, Denmark). Blood was sampled using capillary tubes containing 6 IU of Na-heparin and 9 IU of Li-heparin per 100-μL tube volume. It has been demonstrated that arterialized capillary blood provides an accurate reflection of acid–base status. To ensure that peripheral capillary beds were arterialized, participant’s hands were immersed in a water bath at ~50°C for 1 min before sampling. The first drop of
blood was wiped away, and the tube was held flush with the wound so that blood traveled directly from the tissue to the capillary.

**Data Analysis.** Paired-samples t tests for repeated measures were used to assess differences in all parameters, other than confidence in achievement of preexercise goals, between fastest and slowest trials. Two-way ANOVA for repeated measures was used to assess differences in confidence in goal achievement at intermediate points. In order to identify differences in ability to produce an endspurt, mean power output was calculated for the initial 18 km and final 2 km of each trial. The magnitude of endspurt was defined as the mean power output during the final 2 km expressed as a percentage of mean power output during the initial 18 km. Data is presented as mean ± standard deviation, and significance was accepted at the $P < .05$ level.

**Results**

Mean performance time was not significantly different between Trial 1 (1841 ± 223 s) and Trial 2 (1812 ± 141 s), with both trials displaying a similar overall pacing strategy characterized by an obvious endspurt in the final 10%. Three participants achieved their best performance in Trial 1, with the remainder achieving their best in Trial 2. When comparing the fastest (FAST) and slowest (SLOW) trials, mean performance in FAST was 1796 ± 154 s and in SLOW was 1872 ± 208 s ($P < .01$).

Comparison between FAST and SLOW trials indicated that better performances were achieved through production of a higher power output during the initial 90% of the trial. Mean power during the first 18 km was 248 ± 46 W in FAST and 229 ± 49 W in SLOW ($P < .01$). While power output was higher during the final 2 km, the difference between FAST (317 ± 73 W) and SLOW (298 ± 54 W) was not significant (Figure 1).

The magnitude of endspurt was 129 ± 15% in FAST and 133 ± 15% in SLOW (NS). Five participants achieved their greatest endspurt in their slowest trial, and three in their fastest trial.

In both FAST and SLOW, heart rate continually increased throughout exercise (Figure 2). Participants recorded similar relative values at each intermediate point, and maximal recorded values were also similar (FAST 100 ± 7.0 vs SLOW 101 ± 4.4% predicted maximum heart rate).

Despite differing power outputs during FAST and SLOW, RPE profiles were almost identical, with no significant differences observed at any intermediate point (Figure 3). In both cases, the increase was generally linear from the outset with maximal values attained upon trial completion (FAST 20 ± 0.00 vs SLOW 19.88 ± 0.35). In all but one individual trial, reported RPE at 20 km was 20.

The iEMG tracked power output throughout trials (Figure 4), and this was the case for all individual muscle groups. Comparison of mean iEMG at each 0.5-km interval indicates that during SLOW neuromuscular activity was higher than during FAST in both the initial 90% and final 10% ($P < .01$).

Blood analysis indicates blood lactate concentration was higher following FAST (11.06 ± 3.90 mmol/L) than SLOW (9.01 ± 2.63 mmol/L) ($P = .04$) and pH was lower following FAST (7.25 ± 0.05) than SLOW (7.30 ± 0.04) ($P = .04$) (Figure 5).

Positive affect was significantly higher ($P < .01$) throughout FAST, whereas negative affect was significantly higher ($P < .01$) throughout SLOW. These differences were present from the outset and did not develop as trials progressed (Figure 6).

Although differences did not achieve statistical significance, the trend was for participants to become progressively more confident of achieving their goal throughout FAST, and less confident throughout SLOW (Figure 7).

![Figure 1](image-url) — Power output during the fastest (FAST) and slowest (SLOW) trials (*$P < .01$, **NS).
Figure 2 — Heart rate during the fastest (FAST) and slowest (SLOW) trials.

Figure 3 — Rating of perceived exertion (RPE) during the fastest (FAST) and slowest (SLOW) trials.

Figure 4 — Integrated EMG (iEMG) during the fastest (FAST) and slowest (SLOW) trials (*$P < .01$).
Figure 5 — Postexercise blood lactate and pH after the fastest (FAST) and slowest (SLOW) trials (*$P < .05$).

Figure 6 — Positive and negative affect during the fastest (FAST) and slowest (SLOW) trials (*$P < .01$).
Discussion

The general pacing strategy employed was typical of that described previously, and the achievement of an endspurt was present in every individual trial. In this study it was apparent that superior performances were associated with a significantly higher power output through the initial 90% of the trial rather than a larger endspurt in the final 10%. This aligns with the findings of Jones et al, who demonstrated improved exercise tolerance with “fast-start” strategies, and Swart et al, who demonstrated that over a series of trials participants adopted progressively more “aggressive” strategies resulting in improved performances. Similar performances in the first and second trials performed in this study do, however, suggest that participants did not improve simply through learning to adopt more aggressive strategies.

It is interesting that there were no significant differences in magnitude of endspurt achieved between fastest and slowest trials. It may be expected that the higher work rate in the initial 90% of faster trials would result in greater metabolic disruption, thereby compromising ability to further increase work rate. However, this does not appear to be the case. Although the higher blood lactate concentration and lower pH following faster trials may indicate greater metabolic disruption being allowed by the pacing regulatory center, it does not appear that the ability to produce an endspurt is simply indicative of an overly conservative strategy. Rather, the relationship between power output in the first 90% and final 10% is similar in both fastest and slowest trials, suggesting a high degree of regulation of muscular work rate regardless of actual performance achieved.

In the present study, iEMG activity tracked power output in both fastest and slowest trials, indicating pacing strategies were regulated by central mechanisms rather than being indicative of absolute muscular fatigue. However, as iEMG activity was lower yet power output was higher in faster trials, then effectively the power/EMG ratio was greater on these occasions, suggesting differences in performance resulted from some variation in peripheral physiological status. It may therefore be that higher iEMG activity in the slower trials resulted from neuromuscular compensation for reduced force production capability in the skeletal muscles. Although the exact nature of any differences in peripheral physiological status at the beginning of the trials was unknown, this phenomenon has been demonstrated by Bundle et al, who found compensatory neuromuscular activity resulted from increased reliance on anaerobic metabolism for force production. These authors also suggested that, regardless of specific physiological mechanisms, the principle of compensatory neuromuscular activity for impaired muscle contractile function seems likely to be a general response.

In this study, and in line with previous research, poorer overall performance was associated with higher negative affect and lower positive affect. The similar RPE and higher iEMG activity throughout slower trials suggests participants were maintaining effort in an attempt to achieve a goal performance. The assessment of rate of goal progress has been suggested to encompass two elements, namely, effort toward the goal and goal commitment. Importantly, when individuals remain committed to their goal but performance capacity is reduced due to either reduced effort or peripheral physiological fatigue, then negative feelings occur. Power output was lower throughout slower trials, meaning that although individuals’ perception of exertion was maintained and they were apparently attempting to compensate for reduced force production capacity through activation of additional muscle mass, their rate of goal progress became negative. This was accompanied by lower positive affect and higher negative affect, thereby supporting the findings of Gaudreau et al, who demonstrated a relationship between negative affect and high performance–goal discrepancies. Although differences did not achieve statistical significance, reduced confidence in achieving preexercise goals throughout slow trials may suggest an increasing discrepancy between goal and actual performance as exercise progressed.
It is important to emphasize that participants reported higher positive affect and lower negative affect from the outset during faster trials, with the reverse being the case during slower trials. As superior performances were associated with a less conservative strategy, and RPE was similar throughout fastest and slowest trials then, in this study, it seems that affect may have been a more important regulator of pacing strategy than RPE. Rating of perceived exertion has previously been demonstrated to be influenced by acid–base status, with lower pH being associated with higher RPE at a fixed workload. In the present study, and despite similar RPE values throughout exercise, postexercise pH was lower and blood lactate concentration was higher following fastest trials. Therefore, a more positive affect may have allowed the greater degree of metabolic activity required to achieve superior performances without altering reported RPE values. On their own, the findings of this study cannot determine whether a more positive and less negative affect resulted from positive feedback regarding goal progress during exercise; from afferent feedback from peripheral physiological systems, which is interpreted in the context of the up-coming exercise task; or are independent of these factors. However, given that iEMG data demonstrates that skeletal muscle recruitment increased during slower trials, then it is clear that the ability of skeletal muscle to generate force was compromised during the slower trials, regardless of whether this was responsible for differences in affect. This may suggest that on these occasions peripheral physiological status was not conducive to a high level of performance, and this would have been the case from the outset of exercise.

It is interesting that participants still seemed to be striving to achieve a challenging level of performance throughout both trials. Walsh et al propose that five psychological criteria are required for development of helpless patterns of behavior: (i) the task is extremely important, (ii) self-awareness is high, (iii) the individual believes that further practice will be of minimal benefit, (iv) perception of competence is low, and (v) the person is prevented from reaching a self- or externally defined goal. Even if we add a sixth condition, that of insufficient physical resources for the demands of the activity, then it appears participants continued to strive to achieve their goal even though they grew progressively less confident in their ability to achieve it throughout their slowest trials. It may be that some “threshold” level for each of these criteria must be achieved before participants cease striving for a particular goal. When Walsh et al assessed affect and task persistence within activity, they found increased negative affect when participants failed the task compared with when they were successful. However, no difference was found in task persistence between successful and unsuccessful trials and it was postulated that this was because task persistence was influenced by a complex interaction of gender, goal orientation, and the success/failure context. Within the present study, it may be the case that that even though participants grew less confident in their ability to achieve their goal throughout the slower trials, they persisted in striving to achieve a high level of performance.

**Practical Applications**

This study demonstrates that the psychological construct of affect is a key regulator of pacing strategy during exercise, and that affect may be reflective of peripheral physiological status. Further research is therefore warranted in order to clarify the precise nature of the link between physiological status and affect before and during exercise. Specifically, the investigation of the effects of manipulation of both physiological and psychological parameters will assist in explanation of the manner in which performance and physiological function are regulated during a maximal exercise task. This may eventually result in the ability to prescribe novel interventions that allow athletes to maximize their available physiological capacity during activity or the ability to predict performance based upon pre-performance affect levels.

**Conclusion**

Superior performances during a self-paced exercise task were associated with more aggressive strategies, resulting in greater metabolic disruption. Integrated-EMG activity was greater throughout slower trials, suggesting inferior performance did not result from reduced skeletal muscle recruitment. Rather, it appears peripheral factors related to force generation capabilities account for differences in performance. As RPE profiles were similar throughout fastest and slowest trials, it may also be suggested that affect is the more important regulator of pacing strategy, as this differed between trials from the outset. The exact reasons for this relationship between affect and performance are unclear, although it seems possible that either a more positive and less negative affect allowed toleration of greater physiological disruption, or was reflective of a peripheral physiological status more conducive to the still-to-be-performed exercise task.

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**References**
