Breaking Away: Effects of Nonuniform Pacing on Power Output and Growth of Rating of Perceived Exertion

Jacob Cohen, Bridgette Reiner, Carl Foster, Jos J. de Koning, Glenn Wright, Scott T. Doberstein, and John P. Porcari

The rating of perceived exertion (RPE) normally grows as a scalar function of relative competitive distance, suggesting that it may translate between the brain and body relative to managing fatigue during time-trial exercise. In nonstandard pacing situations, a reciprocal relationship between RPE and power output (PO) would be predicted. *Purpose:* To determine whether PO would decrease when RPE was forced above the normal growth curve during a cycle time trial. *Methods:* Well-trained cyclists performed randomly ordered 10-km cycle time trials. In CONTROL they rode at their own best pace throughout. In BURST, they made a 1-km “burst” at the 4-km mark and then finished as rapidly as possible. *Results:* CONTROL was significantly (*P* < .05) faster than BURST (16:36 vs 17:00 min). During CONTROL, responses between 4 and 5 km were PO, 240 W; RPE, 5–6; and blood lactate ([HLa]), 8–9 mmol/L. During BURST PO increased to 282 W, then fell to 220 W after the burst and remained below CONTROL until the end spurt (9 km). RPE increased to 9 during the burst but returned to the normal RPE growth pattern by 6 km; [HLa] increased to ~13 mmol/L after the burst and remained elevated throughout the remainder of the trial. *Conclusions:* The reciprocal behavior of RPE and PO after BURST supports the hypothesis that RPE translates between the brain and the body during heavy exercise. However, the continuing reduction of PO after the burst, even after RPE returned to its normal growth pattern, suggests that PO is regulated in a more complex manner than reflected solely by RPE.

**Keywords:** fatigue, cycling, burst, breakaway

Success in pursuit-type sports largely depends on minimizing the time required to complete a certain task. Within the last generation, a rich literature has emerged relative to how athletes distribute their energetic reserves to successfully minimize the time requirements for a variety of events.1–5 This literature has collectively been referred to as pacing strategy and is generally understood in terms of the anticipatory-feedback model of regulating energy expenditure put forth from the Cape Town group,6–9 which is based on the concept of teleoanticipation suggested by Ulmer.10 This concept suggests that exercise tasks are organized by athletes to allow completion of an event in minimal time while simultaneously avoiding critical homeostatic disturbances. In support of this concept, recent evidence suggests that different events with approximately the same total energetic requirement (800-m running, 200-m swimming, and 1500-m speed skating) are paced differently to account for differences in kinetic energy and power losses to the environment.11 Much of the pacing literature has focused on events with an “optimal” pacing strategy,1–5 particularly in events where the athlete is racing against the clock. Although there is clear evidence of an overall pacing strategy in longer events,12 many sporting events have a highly stochastic pattern of power output (PO) within the event,13–17 with decisions about how to pace the event also determined by the terrain, the weather, and the behavior of other competitors.

However, we know comparatively little about the consequences of large within-event variations in PO. In concert with a fundamental understanding of the acute18 and chronic19,20 responses to interval exercise, we know that stochastic exercise produces similar net energetic, cardiovascular, and metabolic responses21 and that fatigue associated with stochastic exercise may impair performance more than fatigue during steady-state exercise.22 Given the belief that the rate of growth of the rating of perceived exertion (RPE) is a strongly regulated variable during time-trial exercise,3,4,6,7,23 it seems reasonable to hypothesize that there would be a reciprocal relationship between the increase in RPE after a midrace “burst” and the decrease in PO after the conclusion of the burst. Similarly, it seems reasonable to predict that as RPE returns toward the normal growth curve after the burst, PO would return toward the baseline value observed during time-trial exercise. Accordingly, the purpose of this study was to observe the relationship between changes in RPE and
PO during and after a burst performed during a cycling time trial (ie, similar to an attempt to “break away” from the peloton during a cycling competition).

**Methods**

The subjects for this study were well-trained competitive cyclists (6 men, 4 women). All were competitive at a local level in either road cycling (USCF Category 4, or Veteran) or triathlon. The study was completed in the winter, during a period of relatively light training (3–6 h/wk). Before participating, the subjects provided written informed consent, and the study was approved by the university human subjects committee. Descriptive characteristics of the subjects are presented in Table 1.

The subjects performed a maximal incremental test (3 min at 25 W + 25 W/min) on an electronically braked cycle ergometer (Lode Excalibur, Groningen, NL) to define their exercise capacity. Respiratory-gas exchange was measured using open-circuit spirometry (AEI, Pittsburgh, PA), integrated over 30 seconds. Heart rate (HR) was measured using radiotelemetry.

Each subject performed a total of four 10-km time trials on an electronically braked racing bicycle synchronized with a computer-driven simulator (Velotron, Racermate Inc, Seattle, WA). There were at least 72 hours between trials, and subjects were asked not to perform heavy exercise for 48 hours before each trial. No real or virtual grade or headwind or tail wind was imposed. The first 2 trials were for habituation (trial 1) and to set a target velocity (trial 2). Trials 3 and 4 were performed in random order. The first 4 km of each of trials 3 and 4 were performed at the same average velocity as in trial 2, with the subject using visual feedback regarding momentary PO and velocity from the ergometer console. In trial 3 (CONTROL), after the first 4 km the subject was instructed to finish the 10-km distance as quickly as possible, using a self-selected pacing strategy. In trial 4 (BURST), after the first 4 km the subject was instructed (without prior warning) to perform a 1-km burst at the highest power output he or she could achieve and then to finish the trial as quickly as possible. Obviously, once either trial 3 or 4 was completed, the subject knew which pacing pattern would be used during the last trial. We did not attempt to correct for this by deceiving the subject or by suggesting the presence of future trials. In our experience, performance during competitive simulations such as this trial are so consistent that we felt that the experimental manipulation did not have to be fully blinded, particularly given that pacing was controlled during the first 4 km of each time trial. During the burst, strong verbal encouragement was provided to encourage the subjects to ride at very high intensity, as if they were trying to break away from the peloton in a cycling competition. In an attempt to mimic competitive motivation, the subjects were instructed before both trials 3 and 4 that if they bettered their time from trial 2, they would receive an economic incentive ($10). The elapsed distance, momentary velocity, and PO were visible to the subjects, just as would normally be available in competition with contemporary cyclocomputers.

PO was measured by an electronically braked racing cycle (Velotron, Racermate Inc, Seattle, WA), integrated over each 0.5 km, as was HR. The RPE was measured every 1 km using the Category Ratio RPE scale. Blood lactate [HLa] was measured in capillary blood obtained immediately before the beginning of the time trial and at the end of every 2 km, using dry chemistry (Lactate Plus, Nova Biomedical).

Data analysis was carried out using repeated-measures ANOVA for a multiple-trials (burst vs no-burst trials) × sequential measures within-trial design. Post hoc tests were conducted, when justified by ANOVA, using Bonferroni corrected paired t tests. Statistical significance was accepted when $P < .05$.

**Results**

The times required to complete the BURST (1020 ± 6 s) and CONTROL (997 ± 0.7 s) trials were significantly faster than the baseline trial (trial 2; 1035 ± 6 s). The CONTROL trial was significantly faster than the BURST trial. As per design, the PO, HR, RPE, and [HLa] were the same during the first 4 km of the CONTROL and BURST trials.

During the BURST, between 4 and 5 km, PO (282 ± 58 W) increased significantly compared with CONTROL (240 ± 46 W; Figure 1). After the conclusion of the BURST, PO decreased significantly compared with CONTROL and remained significantly lower through 9.5 km. During the last 0.5 km, during the end spurt, there was no difference in PO between BURST and CONTROL trials. Before 4 km, there was no difference in the rate of growth of RPE between BURST and CONTROL. At the conclusion of the BURST at 5 km, RPE (8.7 ± 1.4) was significantly greater than at 5 km during CONTROL (6.0 ± 1.1). However, after the reduction in PO after the burst, RPE was not different at 6 km in BURST versus CONTROL (6.7 ± 1.4 vs 6.3 ± 0.9) and did not differ between trials through the remainder of the time trial (Figure 1).

HR and [HLa] both increased progressively during the time trials (Figure 2). Both HR and [HLa] were

<table>
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<tr>
<th>Table 1</th>
<th>Descriptive Characteristics (Mean ± SD) of the Subjects</th>
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<tbody>
<tr>
<td></td>
<td>Men (n = 6)</td>
</tr>
<tr>
<td>Age (y)</td>
<td>31.0 ± 10.9</td>
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<tr>
<td>Height (cm)</td>
<td>177.7 ± 4.3</td>
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<tr>
<td>Body mass (kg)</td>
<td>74.6 ± 5.8</td>
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<tr>
<td>VO_{2max} (ml · kg⁻¹ · min⁻¹)</td>
<td>61.4 ± 0.8</td>
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<tr>
<td>Peak power output (W)</td>
<td>354 ± 10</td>
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<td>Peak power output (W/kg)</td>
<td>4.8 ± 0.4</td>
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greater after the BURST than at the same point during CONTROL. During BURST, [HLa] remained above CONTROL at both 6 km and 8 km.

**Discussion**

The first primary finding of this study was that PO decreased after a 1-km burst (designed to be similar to a breakaway attempt during a cycling race), in a manner that was reciprocal with the increase in RPE and beyond its normal rate of growth (ie, template) during a 10-km cycling time trial. The decrease in PO was also reciprocally paired with the increase in objective measures of homeostatic disturbance (HR and [HLa]). These results were consistent with the experimental hypothesis. The second primary finding of this study was that, contrary to the hypothesis, PO failed to completely normalize during the period of recovery after the burst, despite the rapid normalization of both RPE and HR, to values consistent with the normal template of growth during 10-km time trials. To some degree, the depression of PO after the burst mirrored the continued elevation of [HLa]. These data suggest that the linkage between discrepancies in the growth of RPE and deviations from the template PO are less well coupled than proposed in the model of Tucker. However, there is recent evidence from both Renfree et al and Swart et al that other psychophysiologic

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**Figure 1** — Responses of power output (top) and rating of perceived exertion during the CONTROL (filled circles) and BURST (open circles) time trials. The period of the burst, between 4 and 5 km, during which the subjects tried to behave as if they were “breaking away” from the peloton during a cycling race is indicated by the boxed segment in the center. *Power output and rating of perceived exertion are significantly (P < .05) different.

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markers may “fine-tune” the more coarse relationship between the growth of RPE and momentary PO during self-paced exercise.

Renfree et al demonstrated that in addition to RPE, a psychobiologic marker, the affect was associated with changes in PO even when RPE was relatively constant. Comparing slow and fast performances within the same group of subjects, Renfree noted that reductions in PO seemed to be associated with a negative affect. This suggested that affect may decrease when the progress toward the goal (ie, finishing a race in a target time) is not proceeding as rapidly as anticipated. In the setting of this study, the reduction in PO after the burst may reasonably be expected to cause the subject to perceive that the finish is not approaching rapidly enough, and this may have changed affect (a parameter that we did not measure). Thus, in spite of a return of both the absolute value and the rate of growth of RPE to target values shortly after the burst, our results are consistent with the results of Renfree et al to suggest that affect was more negative after the burst. Remarkably, in the report of Renfree et al, even at equivalent values for RPE, slower performances were associated with increased [HLa], which was the only persistent objective physiologic disturbance evident in the current data. Alternatively, there are data suggesting that arousal may be decreased as fatigue develops during heavy exercise. This would likely have the effect of down-regulating PO, even if the RPE at the 6-km point had returned to control values. In addition,
there is active debate over whether homeostatic changes induced by severe exercise (such as the breakaway effort in this study) are communicated by afferent nerves to both the power-producing and the effort-sensing areas of the brain.28,29 Although from a commonsense standpoint one would expect integration of afferent centers, with expression of fatigue sensations defined by the RPE, the process of how fatigue is developed and experienced remains a topic of active investigation.

In a real competitive situation, the effect of the burst may likely be different than observed in this trial. Although it seems highly likely that cyclists in competition reduce their PO after breaking away from the field, as was observed in this data set, the peloton has the luxury of chasing as a group, with a resultant ~30% decrease in PO to ride at the same velocity and the ability to take short turns at very high power output when trying to overtake a rider who has broken away. This explains why breakaway efforts are so rarely successful in cycling competition. However, from the perspective of understanding how athletes regulate their effort during competition, the current results are very clear in their indication that a momentary increase in PO creates a “price to pay” in terms of perceived effort, and this likely leads to some sort of reciprocal reduction in PO. As such, it supports the concept that we have previously put forth, that PO is regulated by an active calculation of the perceptual cost of exercise at a particular moment versus the relative percentage of the competition remaining to be completed.3

Conclusions

The current data suggest that, beyond the immediate down-regulation of PO that occurs after a burst during a cycling time trial, compatible with the concept of regulation of PO by an RPE-driven template, there is a persistent reduction in PO despite a return of RPE to the normal template of growth. This suggests that there may be other psychophysiological elements that are integrated during a task that define how PO is regulated.

References


