UNIVERSITY OF WISCONSIN-LA CROSSE

Graduate Studies

"BREAKING AWAY": THE EFFECT OF NON-UNIFORM PACING ON POWER OUTPUT AND RPE GROWTH

A Manuscript Style Thesis Submitted in Partial Fulfillment of the Requirements for the Degree of Master of Science

Jacob Cohen

College of Science and Allied Health
Clinical Exercise Physiology

December 2010

"BREAKING AWAY": THE EFFECT OF NON-UNIFORM PACING ON POWER OUTPUT AND RPE GROWTH

By Jacob Cohen

We recommend acceptance of this thesis in partial fulfillment of the candidate's requirements for the degree of Master of Science in Clinical Exercise Physiology

The candidate has completed the oral defense of the thesis.

Carl Foster, Ph.D. Thesis Committee Chairperson	4/28/10 Date
Glenn Wright, Ph.D. Thesis Committee Member	4-28-10 Date
Scott Doberstein, MS, ATC, LAT Thesis Committee Member	4/28/10 Date

Thesis accepted

Vijendra K. Agarwal, Ph.D. Associate Vice Chancellor for Academic Affairs	5/6/10 Date

ABSTRACT

Cohen, J. W. "Breaking away": The effect of non-uniform pacing on power output and rpe growth. MS in Clinical Exercise Physiology, December 2010, 31pp. (C Foster)

The purpose of this study was to determine the effects of a short burst in power output midway through a 10 km cycle time trial and its relationship to RPE growth. We hypothesized that a break-away effort in non-normal pacing will cause a disruption in normal pacing strategy. However, a return to the cyclists pre-determined template would occur prior to completion of the time trial. Ten subjects performed a maximal incremental cycling test followed by a total of four 10 km cycling time trials. The first two trials were to habituate the subjects and to establish a baseline performance. The final two trials were randomized and consisted of a spontaneously paced 10 km cycling time trial and a 10 km cycling time trial during which subjects were required to increase velocity (maximum effort) for 1 km . The results of the study indicate that a burst in power output during a 10 km time trial does have an effect on overall power output and RPE growth. During a breakaway effort, power output was significantly increased compared to the no burst time trial. RPE growth was significantly faster during the burst time trial compared to the no burst time trial.

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INTRODUCTION

The sport of cycling is fueled by the desire to push a rider's performance to the limit. Cyclists are often seen in competition attempting to break away from the peleton (main group). On an elite level, riders can be seen in The Tour de France, Giro d' Italia, or La Vuelta a Espana attempting such difficult efforts simply for the sole purpose of winning the day's stage. Research on physiological responses and how athletes "pace" themselves toward the finish has helped with understanding the rationale behind these breakaway efforts. The cyclist embarks upon a journey down the road trying to complete the course in the shortest amount of time possible; a pacing strategy. Providing that the cyclist is of an elite/professional level competitor, it can be safe to assume that this is not the first attempt at completing the given task. Most likely the rider has a certain pattern for expending energy in mind which follows a pre-determined template or plan [3]. This "template" has been created by the athlete from past experiences and may be expressed at both conscious and subconscious levels. There have been several research studies completed on topics such as performance templates, pacing strategies, Rating of Perceived Exertion (RPE), and physiological regulation during exercise. [2,3,6-9] Sensory feedback and physiological changes influence the pacing template by creating the perception of fatigue. Tucker and Noakes describe pacing strategy as "the efficient use of energetic resources during athletic competitions, so that all available energy stores are used before finishing a race, but not so far from the end of a race that a meaningful slowdown can occur". [9] Foster et al [3] investigated the pattern of developing the performance template during high intensity time trials. Participants completed self-paced time trials of varying distances in either rowing or cycling. The results showed that there was a learning effect when the subject performed multiple time trials. Through the first 4 of 6 time trials, each trial was significantly faster than the previous. The learning effect was attributed to the fact that performance of each trial increased with confidence that the trial could be completed with minimal risk of exhaustion or fatigue. This was accomplished by having higher levels of exertion earlier in the time trial.

Ulmer [10] proposed a feedback control system for controlling an athlete's performance and metabolic expenditure. Ulmer [10] used the term "teleoanticipation" to describe the control system's ability to take into account the finishing point. Recently other physiological events such as the rate of heat storage and body temperature have been suggested as important controllers of effort. Thus, when an athlete engages in a self-paced event, they have the ability to regulate performance so as to prevent physiological changes that might be detrimental to performance and to avoid homeostatic disturbances large enough to be dangerous.

Tucker [8,9] demonstrated that an athlete's RPE plays an important role as mediator in the regulation of work rate. The RPE scale developed by Borg is a well-established and highly used device to measure perceived exertion. [10] Tucker demonstrated that RPE has been shown to increase in proportion to distance completed and/or distance remaining during an event. [2] The results of Faulkner found that RPE was set as a function of either how much of the exercise bout had been performed, or how much of the exercise bout remained. These findings demonstrate that RPE is scalar to time and suggests that the brain regulates performance in an anticipatory manner based on the knowledge of metabolic energy reserves and biomechanical performance from the start of a given exercise.

As an investigator of performance, it is necessary to review how a performance template develops through a series of feedback systems within the body. These feedback systems play an intricate role in the development of an appropriate pacing strategy for completing a race. [9] Research about physiological regulation of pacing has suggested that the pacing strategy is a main component of an internal control system that is responsible for anticipating exertion and providing feedback to other body systems. Pacing strategies require continual regulation by the brain during exercise. [5] This proposed system is often referred to as the teleoanticipatory system, where the brain anticipates the finishing point of exercise. [1,6-10] The concept of teleoanticipation (telos = end) involves the brain regulating the intensity of exercise accordingly to ensure the athlete can complete competition without unreasonable disturbances to homeostasis.

Given that athletes do develop a pacing strategy based on a feedback mechanism, it becomes necessary to ask what happens when the strategy is disrupted? Foster et al [3] demonstrated that after repeated attempts of a particular exercise task such as a cycling time trial, athletes develop a performance template with growing confidence that the task at hand can be completed with little harm to homeostasis. Investigation regarding changes to athletes perceived exertion and overall work completed when the "template" is disrupted. Reiner [5] asked a similar question regarding changes to perceived exertion changes in cycling time trials when the performance template was disrupted. However, the disruption in the pacing strategy was not intense enough, thus little change was seen in the athletes perception of performance and work completed.

PURPOSE/HYPOTHESIS

The purpose of this study was to determine the effects of a short burst in power output midway through a 10 km cycling time trial (non-uniform pacing) on the perception of fatigue and muscular power output (e.g. pacing strategy) and its relationship to RPE growth. It was hypothesized that a break-away effort (short duration burst) in non-normal pacing will cause a disruption in normal pacing strategy. However, a return to the cyclist's pre-determined template will occur prior to completion of the time trial as depicted by figure 1.

METHODS

Subjects

The subjects were ten healthy, well-trained cyclists and tri-athletes. All subjects provided written informed consent prior to participation in the study, and the study protocol was approved by the Institutional Review Board for the Protection of Human Subjects at the University of Wisconsin- La Crosse.

Table 1. Characteristics of Subjects (mean±SD)

	Males (n=6)	**Females (n=4)**
Age (yr)	31.0±10.88	21.3± 2.06
Height (in)	69.97± 1.70	65.00±1.15
Mass (kg)	74.60±5.75	62.10±6.93
VO2 max (L/min)	4.58±0.58	3.24±0.46
Peak Power at VO2max (W)	354.±10	269±43
Power Output (W/kg)	4.8±0.4	4.4±0.8

Procedures

The subjects performed a maximal incremental cycling test to determine their VO2max followed by a total of four 10 km time trials on a racing road bicycle synchronized with a computer driven simulator (Velotron, Racermate Inc.). Subjects were given a minimum of 48 hours of rest between trials. A self-selected warm-up was performed prior to all trials. The first two 10 km cycling time trials were performed to habituate the subjects to the task and to establish a baseline performance. The final two trials were randomized and consisted of a spontaneously paced 10 km cycling time trial and a 10 km cycling time trial during which subjects were required to increase velocity (maximum effort) for 1 km at the 4 km point in the ride (e.g similar to a break away effort in competitive road cycling). After the break away effort, the subject was instructed to finish the remainder of the time trial at the best possible speed. In the break away trials as well as the normal pace trials, subjects were instructed to try to beat their baseline time. Prior to either the normal pace or breakaway trials, subjects were informed that they may have to perform a burst in power output. A small monetary incentive was given to encourage subjects to beat their baseline time in each of these two trials. They were not informed regarding whether the trial was freely paced or would require a break away until just prior to the 4 km point. This was done with the intention of encouraging the subjects to ride at approximately the same pace during the first 4 km of both the experimental trials. RPE (Rating of Perceived Exertion) was measured using the Category Ratio (Borg) 0-10 scale at the end of every 1 km. Heart rate (radiotelemetry, Polar), power output (Velotron, Racermate Inc.), and time were measured continuously and recorded every ½-km (500 meters). Fingertip blood samples were obtained every two kilometers and analyzed for lactate accumulation using dry chemistry (Lactate Plus, Nova Biomedical)

Data Analysis

Data analysis was carried out using repeated measures ANOVA to test the hypothesis that a break-away effort (short duration) of non-normal pacing would cause a disruption in a cyclists normal pacing strategy. Post-hoc testing was carried out using paired t-tests to analyze the significance of data points between no burst and burst conditions. An alpha of 0.05 was used to determine the statistical significance within data points of difference between burst and no burst conditions.

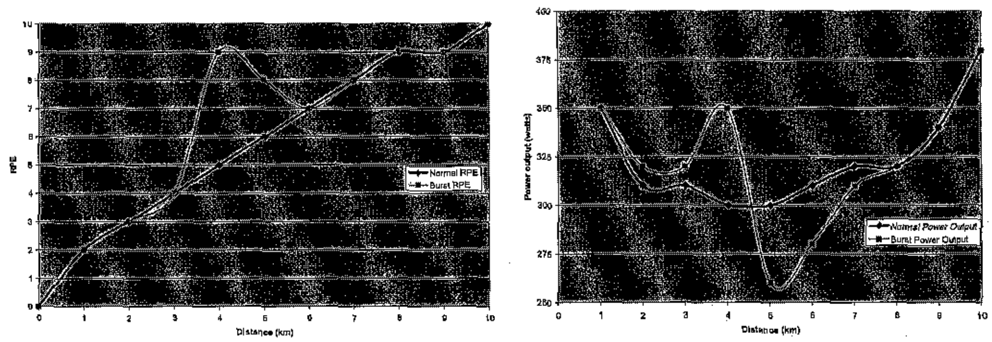

Figure 1 – Hypothesis of reciprocal changes between RPE growth and power output

RESULTS

The time required for completion of the burst (17:00±0.05 min) and no burst (16:37±0.07 min) trials were faster compared to baseline (17:15±0.06 min) trial. Power output and RPE were the same during the first 4 km during the steady state and burst time trials. During the burst, power output was significantly higher than the steady state time trial, as designed by the protocol. Average power output midway through the burst (4.5km) was 288.36±58.65 Watts. At the same point during the steady state trial, power output was 238.35±46.40 Watts. Depicted by Figure 2, power output following the burst was 221.42±44.46 Watts at 5.5-km and 218±44.53 watts at 6-km compared to the no burst trial 243.06±47.17 Watts at 5.5-km and 243.72±48.31 Watts at 6-km, respectively. Power output continued to be slightly lower, although not significantly, during the remainder of the burst trial compared to the no burst trial.

Figure 3 shows RPE during the burst trial was significantly higher immediately following the burst at 5-km was 8.7±1.52 compared to the same point during the steady state trial (6.00±1.05). RPE remained slightly elevated during the burst trial at 6 km compared to the no burst trial, but this difference was not statistically significant. By 7-km, RPE had returned to the normal pattern of increase seen during the no burst trial.

Blood lactate concentration was not different between burst and no burst trials during the first 4 km. Blood lactate increased to 12.8±1.9 mmol at 6 km during the burst trial compared to 9.3±1.6 mmol during the no burst trial. Lactate concentration continued to be elevated during the burst trial compared to the no burst trial for the remainder of the trial.

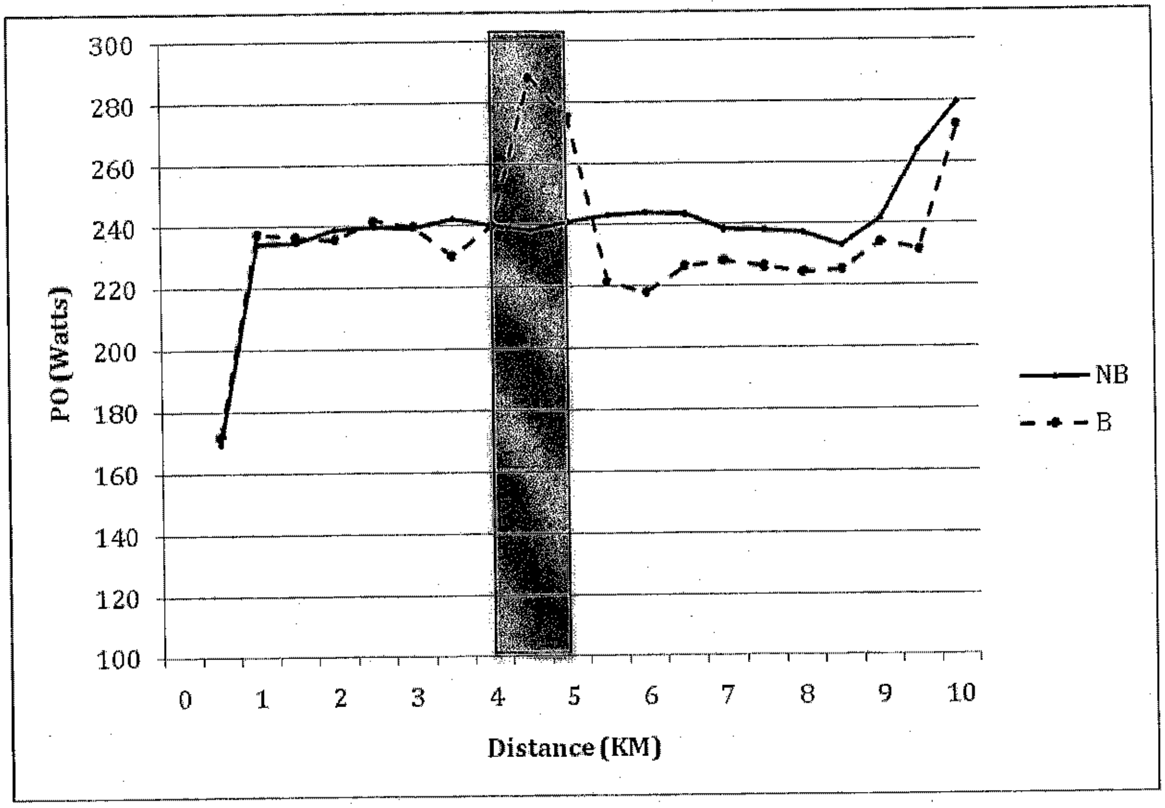

Figure 2 – Average power output during burst and no burst trials.

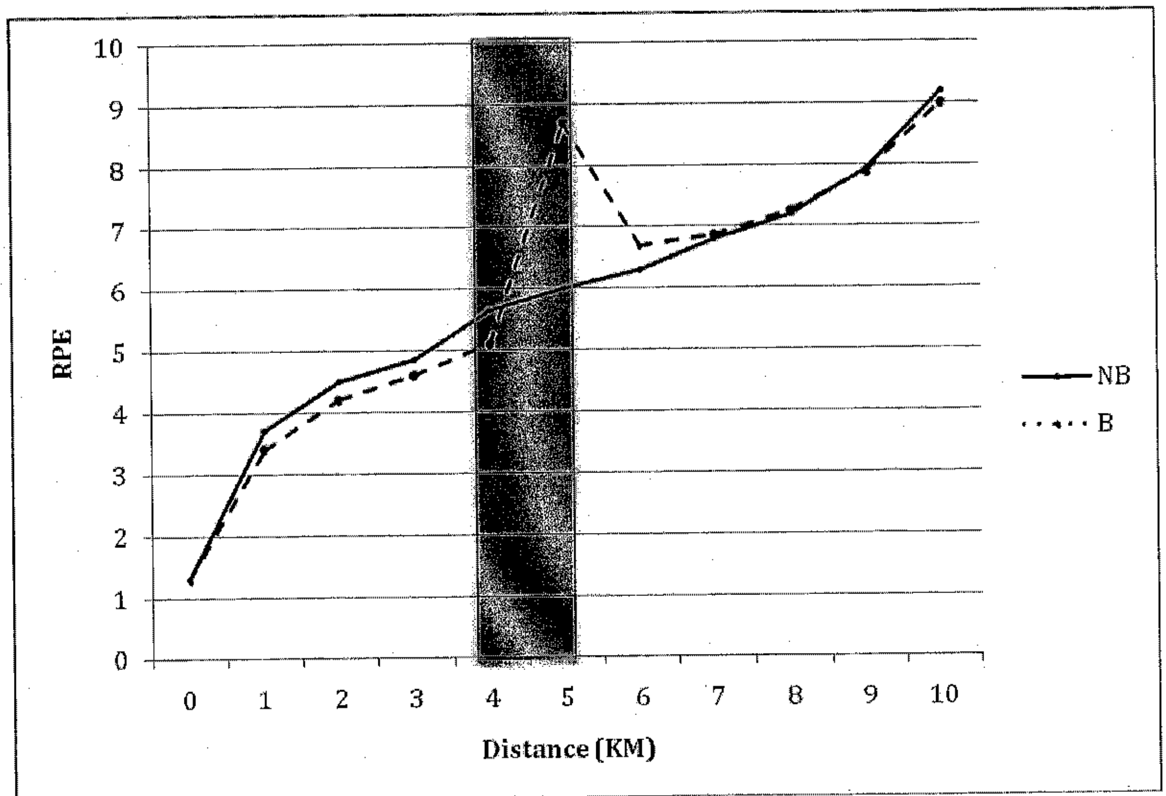

Figure 3- Average RPE growth during burst and no burst trials.

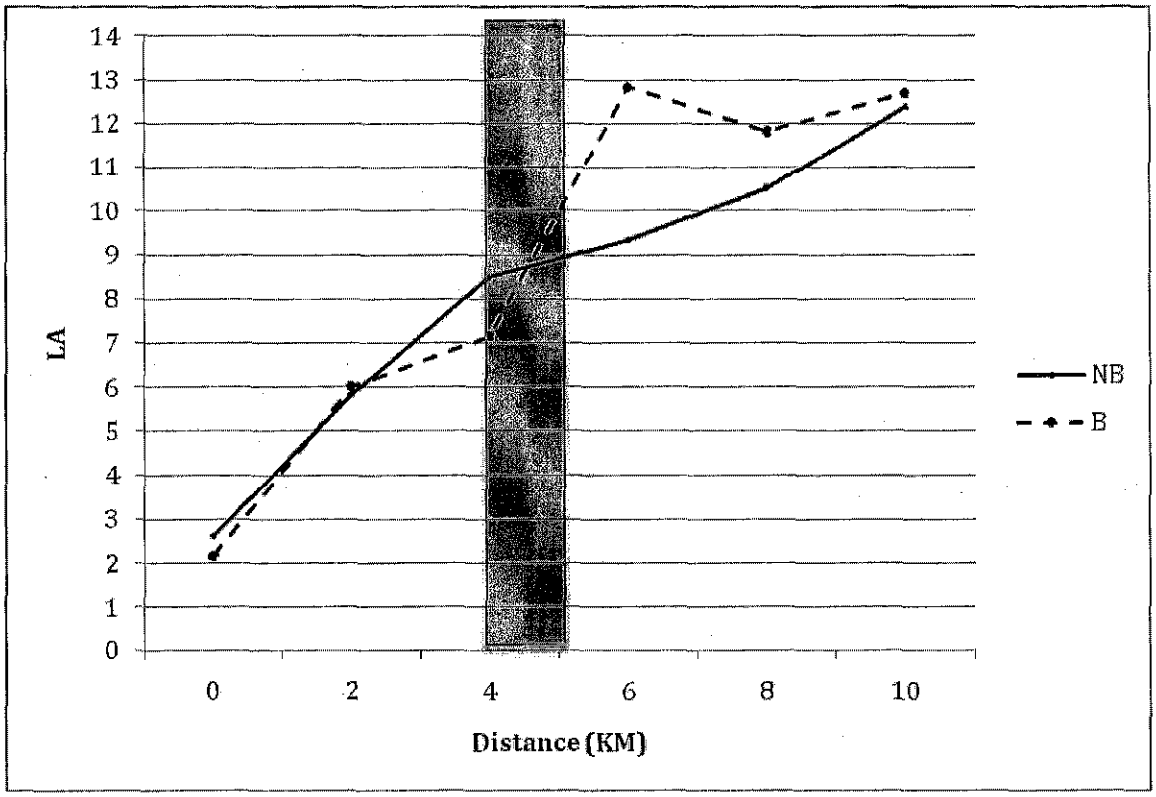

Figure 4- Blood lactate concentrations during burst and no burst trials.

DISCUSSION

The results of the study indicate that a burst in power output during a 10 km time trial has an effect on overall power output and RPE growth. During a breakaway effort, power output was significantly increased compared to the no burst time trial. However, power output following the burst was decreased for the remainder of the trial compared to the no burst trial (Fig. 2). This supports the idea that a burst in power midway through the 10 km time trial disrupts the subject's pre-determined pacing strategy after the intense effort.

RPE growth was significantly faster during the burst time trial compared to the no burst time trial. Depicted by figure 3, it can be seen that despite the increase in RPE following a burst, after only a short recovery period RPE had returned to the scalar growth pattern expected during a normal time trial. Thus, although there was the expected reciprocal relationship between RPE growth and power output, the power output never fully normalized despite a return of RPE to its normal growth profile. Development of a pacing strategy can be seen when reviewing average completion times of three different time trial conditions: habituation, no burst, and burst. The trend of faster completion time supports the idea of teleoanticipation and pacing strategy development through repetition of similar time trials or efforts. Both Ulmer and Foster's research about the development of pacing strategies support these findings.

Figure 4 shows that blood lactate accumulation was elevated to higher levels during the burst trial compared to the no burst condition after the 4-km point. This rise in lactate levels is attributed to the burst in power output at 4-km. Coincidently, lactate accumulate during the burst trial was lower compared to the no burst condition right before the breakaway effort. Anticipation of the task ahead is the contributing factor for the slight decrease.

Results of this study are supported by previous research regarding the scalar properties of RPE growth. Joseph's [4] research regarding various distances of time trials showed similar scalar RPE growth regardless of the distance. Reiner et al [5] also studied RPE progression in cycling time trials. Once again, the results support the findings of the presented study suggesting that RPE growth does rise in a scalar fashion. Despite the burst in power and the disruption of the pacing strategy, RPE did return to its normal linear growth. Reiner's [5] study also supports the findings in the reciprocal changes seen in RPE growth and power output.

REFERENCES

1. Abbiss CR, Laursen PB. Describing and understanding pacing strategies during athletic competition. *Sports Med.* 2008;38:239-252

2. Faulkerner J, Parfitt G, Eston R. The rating of perceived exertion during competitive running scales with time. *School of Sport and Health Sciences, University of Exeter*

3. Foster C, Hendrickson K, Peyer K, Reiner B, DeKoning JJ, Luia A, Battista R, Hettinga F, Porcari JP, Wright G. Pattern of developing the performance template. *British Journal of Sports Medicine* 2009; 5

4. Jospeh T, Johnson B, Battista RA, Wright G, Dodge C, Porcari JP, De Koning JJ, Foster C. Perception of fatigue during simulated competition *American College of Sports Medicine* 2007; 381-386

5. Reiner BA , "Breaking away": The effect of non-uniform pacing on RPE growth. *Unpublished* 2008

6. St. Clair Gibosn A, Lambert EV, H.G Rauch L, Tucker R, Baden DA, Foster C, Noakes T. The Role of information processing between the brain and peripheral physiological systems in pacing and perception of effort. *Sports Medicine.* 2006; 36;8: 705-722

7. St Clair Gibson A, Lambert EV, Lambert MI, Hampson DB, Noakes TD. Exercise and fatigue-control mechanisms. *Int Sports Med J* 2001;3;2

8. Tucker R. The anticipatory regulation of performance: the physiological basis for pacing strategies and the development of perception-based model for exercise performance. *British Journal of Sports Medicine* 2009;43:392-400

9. Tucker R, Noakes TD. The physiological regulation of pacing strategy during exercise: a critical review. *British Journal of Sports Medicine* 2009;43:e1

10. Ulmer V. Concept of an extracellular regulation of muscular metabolic rate during heavy exercise in humans by psychophysiological feedback. *Experientia* 1996;52:416-420

APPENDIX A

INFORMED CONSENT

INFORMED CONSENT

"Breaking Away': The effect of Non-Uniform Pacing on RPE Growth"

Principal Investigator: Jacob Cohen
423 11th St N
La Crosse, WI 54601
612-701-4598
jakewcohen@gmail.com

Emergency Contact: Name: ______________________________

Phone Number: ______________________________

Purpose and Procedures

- The purpose of this study is to determine the effects of a short burst in power output midway through a 10 km cycling time trial on the perception of fatigue and on muscular power output (e.g. pacing strategy)
- Participation in this study will require ~5 Hours of my time over the course of approximately 2 weeks.
- 1st session: This will be a maximal exertion test. This will begin at low intensity workload and progressively increase in intensity until maximal exertion has been achieved, typically lasting 10-20 minutes. I will be wearing a mouthpiece that is hooked up to a respiration-analyzing device throughout the duration of the test.
- 2nd -5th sessions: Perform four 10 km cycling time trials, of which two trials are practice trials to familiarize me with the lab and equipment and establish my baseline performance, one trial at my normal pace but with the goal of beating my baseline time to completion, and one 20 km time trial with a 2 km burst at the 10 km point. I will wear a heart rate monitor and have small blood samples taken from my finger for all time trials.
- Testing will take place in Mitchell Hall room 225, UWL

Potential Risks

- The risk of serious complications in healthy individuals is almost zero.
- The side effects of the study are similar to those of a bicycle race. I am aware that I may experience shortness of breath, fatigue, muscle soreness, and some discomfort from the breathing apparatus and finger stick blood samples. Individuals trained in CPR and first aid will be present during testing sessions.

Rights and Confidentiality

- Participation is voluntary
- I have been informed that I am free to withdraw from the study at any point in time without penalty.
- If this study is published in scientific literature or presented at professional meetings, the results will be presented as aggregate data with no names associated with the data.

Possible Benefits

Benefits to me and the scientific community is that I may help determine the effect of non-uniform pacing on RPE growth and muscular power output and its effects on cycling time trials.

Questions regarding any procedure in this study can be directed to Jacob Cohen (612-701-4598) or Carl Foster (608-785-8687). Question regarding the protection of human subjects may be addressed to the UW-La Crosse Institutional Review Board for the Protection of Human Subjects, (608-785-8124 or irb@uwlax.edu).

Jacob Cohen
UWL Grad Student/Researcher
jakewcohen@gmail.com
612-701-4598

Dr. Carl Foster
UWL Student Advisor
Foster.Carl@uwlax.edu
608-785-8687

Participant ______________________________ Date ____________

Researcher ______________________________ Date ____________

APPENDIX B

REVIEW OF LITERATURE

REVIEW OF RELATED LITERATURE

The purpose of this paper is to review related literature concerning rate of perceived exertion (RPE), fatigue, pacing strategies, and performance template development.

Rate of Perceived Exertion (RPE)

Using the rating of perceived exertion (RPE) has been accepted as a valid method for measuring exertion during physical activity. A subjective RPE can be found using the Borg's 1-10 scale.[1] Conclusions have been made that RPE provides an adequate reference for regulating high intensity exercise. The RPE scales has, in an informal way, been linked to physiological variables such as muscle force output, heart rate, ventilation, respiratory rate, oxygen uptake, and blood lactate concentration.[2] There have been studies that have shown that RPE is can also be influenced by psychological factors as well. Baden at al [2] suggested that RPE was influenced by affect and knowledge of exercise duration, and attempted to display this in a study involving subjects running variable distances on a treadmill. Subjects ran three trials at 75% of their peak treadmill running speed. The first trial each runner was told to run for 20 minutes and stop, the second trial they were told to run for 10 minutes, but at 10 minutes were told to run for an additional 10 minutes. The third trial subjects we not told how long they were going to run, but were however stopped at 20 minutes. Results showed a bigger increase in RPE during the second trial compared to the other two. Supporting the original hypothesis, they suggested that RPE is not purely a measure of physical exertion. The changes witnessed in RPE resulted from an affective process; the changes in RPE after being deceived about the distance to be completed were not merely just a change in physiological influences.[2]

A study done by Eston et al [3] looked at fatigue on the relationship between perceived exertions during constant exercise. They wanted to test whether the scalar time model of RPE was affected by antecedent fatiguing activity. Participants completed four laboratory exercise tests using an electronically braked cycle ergometer. Subjects were tested under fatigued and non-fatigued conditions based in their individual VO2 max measurements. Each test required the subject to ride at 75% of their VO2max during the constant load cycle test and the fatigued test. Supporting Eston et al's [3] hypothesis results showed that time to exhaustion were significantly less in the constant load test compared to the fatigued test. RPE values increased significantly in each condition over time as well. The increase in RPE was greater after the fatiguing bout of exercise even though the time to exhaustion occurred earlier. The results of RPE were plotted as a percentage of time taken for each exercise trial in fatigued and non-fatigued conditions. The rate of increase in RPE was found to be similar for both conditions, supporting similar findings that RPE appears to have scalar time properties. The results also support other studies, similar to those completed by Faulker [4] and Joseph et al [5] suggesting that RPE could be used to predict the endpoint or duration of an exercise bout. These results also support the idea that RPE during exercise demonstrates the effects of antecedent fatiguing and that an internal timing device (central control theory) regulates RPE. [3] Faulkner et al. were able to display this scalar increase in RPE over time. In Faulkner's study, he studied RPE and heart rate along with pacing strategies over various running distances. Although the rate of increase in RPE was greater in a 7-mile race compared to the 13.1-mile race, there were no differences when RPE was expressed over time. This further supports the notion that RPE scales in proportion to the duration of exercise.[4,5]

St. Clair Gibson et al [6] concluded that it is more likely to sense an increase in a sensation compared to a decrease. That being said, they suggest that an athlete is more likely to perceive increases in RPE compared to a decrease in RPE during competition, thus making an increase in RPE occur as a linear event, scaling relative to time.[5,6] However, changes in RPE can increase in a non-linear function too. For instance, if cyclist attempts to break-away from a fellow group of riders (or peleton); or if an athlete/cyclist receives sudden unexpected crowd support.[6]

Analysis of Fatigue

Fatigue has often been referred to as a result of interactions between peripheral physiological systems and cognitive systems of the body.[6,8] It can also be defined as a decrease in contractile function as a result of high intensity exercise.[9,10]

It can now be seen that an athlete, or cyclist for purposes of this review, has an internal anticipatory system for regulating energy expenditure. This regulation system is responsible for the development of the pacing strategy, which is merely a distribution of work and energy expenditure. [11-13] Compared to team sports where athletes compete against one another in a head-to-head fashion, endurance sports such as running, cycling, and rowing are thought to be "closed-loop" designed, where the athlete is attempting to finish a known distance in the shortest possible amount of time. [11]

A cyclist's anticipatory pacing system is designed to prevent catastrophic events in homeostasis. St. Clair Gibson et al [6] reported that the concept of fatigue is still controversial in respects on its relationship to skeletal muscle (peripheral) and brain (central) mechanisms. In an attempt to discover mechanisms causing fatigue, three different models have been proposed as an explanation. The peripheral model of fatigue views the phenomenon as a decrease in intensity of exercise level as a direct result of peripheral influences on the body. These influences derived from intense exercise include decreases in pH, decreased ATP (energy stores), inorganic phosphate accumulation, changes in electrolytes, etc. The second model, the central-teleoanticipation model, views fatigue as a safety mechanism which in return, maintains metabolic activity at a safe range to control homeostasis. This concept is derived from Ulmer's [14] studies about teleoanticipation as a central control mechanism for physical activity. This model suggests that efferent neural commands signal skeletal muscle to maintain a certain metabolic output (power), taking into consideration the duration and intensity of the exercise, in order to assure completion of activity without damage to homeostasis and within the certain time frame and/or desired distance. A third model of fatigue known as the Cognitive-Discussion Model states that the sensation of fatigue, itself, will moderate the level of intensity based an perceived effort acting as the "regulator" of energy expenditure. Once fatigue is experienced at the conscious level, signals are sent to the sub-conscious area of the brain where anticipatory processing would take place, much like the process described in the previous model of fatigue. In other words, there is a constant "discussion" continuing on between the conscious and sub-conscious parts of the brain. [6]

Lambert et al [7] have proposed that a complex model of fatigue, where there is thought to be a central programmer as a regulator for the process of determining fatigue in exercise exists. It is suggested that the central controller of exercise takes neural input from the peripheral system. St. Clair Gibson et al [8] have further argued that fatigue is a result of sensory perception, rather than physical events such as decreases in pH, acidosis, etc. They believe that muscular fatigue does not reside soley in muscle.[8] The central governor model explains the regulation of exercise intensity comes from the central nervous system (CNS). For this to be the case, fatigue would no longer be considered a physical event, instead it may be regarded as an emotional event. The way the central governor system works is the brain reduces the number of muscular motor units recruited during prolonged exercise.[8, 15] Any additional motor unit recruitment above what has been calculated would result in unreasonable disturbances in homeostasis, which in turn could lead to an early termination of exercise. This model also uses RPE progressively in order to discourage the conscious mind from attempting to recruit more motor units. If the system were overridden, the athlete would attempt to recruit more units than the body can manage in a safe (homeostatic) way.

Pacing Strategies

As an investigator of performance, it is necessary to review how a performance template develops through a series of feedback systems within the body. These feedback systems play an intricate role in the development of an appropriate pacing strategy for completing a race.[13] Research about physiological regulation of pacing has suggested that the pacing strategy is a main component of an internal control system that is responsible for anticipating exertion and providing feedback to other body systems. Pacing strategies require continual regulation by the brain during exercise.[6] This proposed system is often referred to as the teleoanticipatory system, where the brain anticipates the finishing point of exercise.[6,11,13, 14, 16] The concept of teleoanticipation (telos = end), involves the brain regulating the intensity of exercise accordingly to ensure the athlete can complete competition without unreasonable disturbances to homeostasis.[13] Energy expenditure and work completed (power output) are thus regulated by this central governor within the brain.[11] "Exercise intensity is regulated within the brain based on a complex "algorithm" involving a peripheral sensory feedback system and the anticipated workload remaining.[11] Since the algorithm is selected for an appropriate endpoint and expected distance/duration of an event, knowledge of the endpoint must be one of the main controllers of metabolic activity in the peripheral physiological systems.[6] The feedback system is influenced by previous exercise bouts experienced by an athlete. Environmental variables such as temperature, gradient, and course have been shown to influence the development of the anticipatory system as well as physiological and psycholocial influences such as previous metabolic expenditure, fatigue, RPE, HR, and power output. [4]

St. Clair Gibson et al [6-8] described how pacing strategies in exercise are controlled by information processing between the brain and peripheral physiological systems. They proposed that there are many different types of pacing strategies that can be developed in the brain depending on the type of activity being attempted.[6] However, there has not been a sufficient amount of research completed regarding the "optimal" pacing strategy. St. Clair Gibson et al [6] proposed four broad pacing strategies into which a given exercise bout can fall:

- An all-out pacing strategy involves the athlete beginning the particular event at their maximal possible pace and continues to attempt to continue this maximal pace until the event ends. Subsequently, there is a decrease in pace towards the end of the event.

- A slow start pacing strategy requires the athlete to start off exercise at a submaximal pace and increases pace at a steady rate throughout the event.
- Even paced strategy has an athlete maintain a constant submaximal rate through the entire event.
- Variable pacing strategy involves an initial maximal pace in the first stage of the event, then pace is moderated during the middle portion of the event, then lastly pace is increased towards the end of the event.

Reiner [17] completed a study evaluating non-uniform pacing on RPE growth. Non-uniform pacing would be considered a small interval of even higher intensity than the predetermined pace; a "break away" effort. The study examined effects of an increase in power output midway through a 10 km cycling time trial and the related effects on pacing strategy and RPE. Subjects were 8-road cyclists who performed three, 10 km cycling time trials. The first trial was used to allow subjects to habituate to the equipment and establish a baseline, another trial was a "best pace" steady state ride, and the other time trial included a burst in pace by 2 km/hr for 1 km at the 3 km point of the ride. After the "break away" effort, subjects were required to complete the remainder of the time trial in the best time possible, trying to beat their baseline times. Results of the study supported the hypothesis that RPE would be higher following the "break away" effort. However, power output was not down regulated significantly suggesting that the burst in velocity and effort may not have been hard enough.[17]

Development of a Performance Template

It has been established that the sport of cycling requires great physical and mental endurance. The rigors of training and competition place great stress on the physiological and psychological systems of the body. A cyclist of the elite level has had plenty of experience and input relating to how they can complete a race putting forth the best performance possible. The athlete has constructed a mental "template" on the best possible way to finish a particular course; they can anticipate what lies ahead. Foster et al [8] conducted a study about the development of a performance template. The purpose of their study was to make systematic observations about how the performance template is developed in fit individuals. The individuals being tested in the study had limited experience with high intensity time trials. Foster et al [8] wanted to observe power output in individuals during a variety of cycling and rowing exercise time trials. Results suggested that there is indeed a "learning effect during the performance of successive high intensity time trials." There was an increase in learning during earlier time trials compared to later trials. Subjects became more confident in their ability to complete each time trial as the study continued on. Foster et al [18] found that subjects were "holding back" during early trials, then progressively increased effort throughout trials as they developed a specific pacing strategy without negative consequences.

Summary

An athlete constructs an internal performance template based on previous experiences physiological inputs about a given event. Pacing strategies are then designed and controlled by a central governor within the brain. This control center regulates pacing which in turn regulates metabolic output in an attempt to prevent fatigue and other metabolic disturbances to the body's natural homeostasis. Rate of perceived exertion can be used as an appropriate measure of exercise intensity. Using Borg's 1-10 scale of RPE offers a simple yet subjective approach to categorizing exercise efforts. RPE has suggested being predetermined too in regards to a particular pacing strategy being used to complete an athletic event.

REFERENCES

1. Wergel- Kolmert U, Wisen A, Wohlfart B. Repeatability if measurements of oxygen consumption, heart rate, and Borg's scale in en during ergometer cycling. *Clinical Physiology and Functional Imaging.* 2002;22:261-265

2. Baden DA, McLean TL, Tucker R, Noakes TD, St. Clair Gibson A. Effect of anticipation during unknown or unexpected exercise duration on rating perceived exertion, affect, and physiological function. *British Journal of Sports Medicine.* 2005;39:742-746

3. Eston R, Faulkner J, St Clair Gibson A, Noakes T, Parfitt G. The effect of antecedent fatiguing activity and physiological activity during constant load exercise. *Psychophysiology* . 2007;1-8

4. Faulkerner J, Parfitt G, Eston R. The rating of perceived exertion during competitive running scales with time *School of Sport and Health Sciences, University of Exeter.* 2008

5. Jospeh T, Johnson B, Battista R, Wright G, Dodge C, Porcari JP, De Koning JJ, Foster C. Perception of Fatigue during Simulated Competition *American College of Sports Medicine.* 2007;381-386

6. St Clair Gibson A, Lambert EV, Lambert M, Hampson D, Noakes T. Exercise and Fatigue-Control Mechanisms. *Int Sports Med J* 2001;2(3)

7. Lambert EV, St. Clair Gibson A, Noakes TD. Complex systems model of fatigue: integrative homeostatic control of peripheral physiological systems during exercise in humans. *British Journal of Sports Medicine.* 2005;39:52-62

8. Noakes TD, St. Clair Gibson A, Lambert EV. From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans: summary and conclusions. *British Journal of Sports Medicine.* 2005;39;120-124

9. Rauch HGL, St. Clair Gibson A, Lambert EV, Noakes TD. A signaling role for muscle glycogen in the regulation of pace during prolonged exercise *British Journal of Sports Medicine.* 2005;39:34-38

10. Westerblad H, Allen D, Lannergren J. Muscle Fatigue: Lactid Acid or Inorganic Phosphate the Major Cause? *News Physiological Science*. 2002;17:17-21

11. Abbiss CR, Laursen PB. Describing and Understanding Pacing Strategies during Athletic Competition *Sports Med.* 2008;38:239-252

12. Tucker R. The anticipatory regulation of performance: the physiological basis for pacing strategies and the development of perception-based model for exercise performance *British Journal of Sports Medicine.* 2009;43:392-400

13. Tucker R, Noakes TD. The physiological regulation of pacing strategy during exercise: a critical review. *British Journal of Sports Medicine.* 2009;43: e1

14. Ulmer V. Concept of an extracellular regulation of muscular metabolic rate during heavy exercise in humans by psychophysiological feedback. *Experientia.* 1996;52:416-420

15. Weir JP, Beck TW, Cramer JT, T Housh J. Is fatigue all in your head? A critical review of the central governor model. *British Journal of Sports Medicine*. 2006; 40:573-586.

16. St. Clair Gibosn A, Lambert EV, Rauch L, Tucker R, Baden DA, Foster C, Noakes T. The Role of Information Processing Between the Brain and Peripheral Physiological Systems in Pacing and Perception of Effort. *Sports Medicine*. 2006;36(8):705-722.

17. Reiner B "Breaking away": The effect of non-uniform pacing on RPE growth. *Unpublished* 2008

18. Foster C, Hendrickson K, Peyer K, Reiner B, DeKoning JJ, Luia A, Battista R, Hettinga F, Porcari JP, Wright G (2009) Pattern of Developing the Performance Template. *Medicine and Science in Sports & Exercise.* 2009; 2-10.