

UNIVERSITY OF WISCONSIN-LA CROSSE

Graduate Studies

RAPIDITY OF RESPONSE TO HYPOXIC CONDITIONS DURING EXERCISE

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

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College of Science and Health
Clinical Exercise Physiology

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RAPIDITY OF RESPONSE TO HYPOXIC CONDITIONS DURING EXERCISE

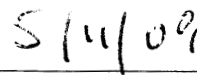
By Kayla Henslin

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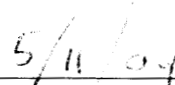
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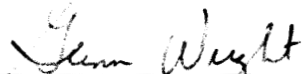
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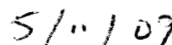
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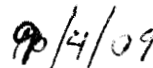


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ABSTRACT

Henslin, KB. Rapidity of response to hypoxic conditions during exercise. MS in Clinical Exercise Physiology, December 2009, pp.40. (C. Foster)

Previous studies have found decreases in arterial oxygen saturation to be linked to reductions in power output (PO). The purpose of this study is to determine how long is required to identify arterial desaturation and change pacing strategy while breathing low oxygen concentrations during simulated competition. We tested the hypothesis that the starting PO of a TT would be the same regardless of inspired $F_{I}O_2$, due to a strong pre-trial template. Trained cyclists performed randomly ordered 3 km time trials with differing patterns of low oxygen administration. The early desaturation time trial began 3 minutes after administration of hypoxic air; the late desaturation time trial began 30 seconds after administration. We saw no early effect on PO when the hypoxic condition was applied prior to the time trial, and there was no difference relative to when hypoxia was applied. The time required to decrease PO was approximately 40 seconds after the start of the trial. Despite reductions in PO, the rating of perceived exertion was greater throughout the hypoxic trial. The results support the strong effect of the pre-exercise template, and the inadequacy of matching PO to perceived exertion during the trial.

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INTRODUCTION

Pacing strategy is the individualized plan for completing a task in the shortest possible time. It has also been described as the efficient use of limited energetic sources, so that all energy stores are used prior to finishing an exercise bout, but not so early that a significant slowdown occurs [5]. Pacing strategy is determined by several factors including: individual capacities of the athlete, previous experience in training or racing, anticipation of event duration, physiological feedback and central feedback.

During self-paced exercise, the work rate is adjusted to prevent unreasonably large disturbances in homeostasis. The best contemporary model for pacing strategy is the Anticipatory Regulation of Self-Paced Exercise [12]. This model has both anticipatory and feedback components. The anticipatory component uses prior experience and preparation along with physiological and psychological inputs. Based on the anticipated distance or duration of the exercise, the athlete sets an initial intensity. Prior to exercise, an expectation of the projected rate of increase in fatigue is determined. The level of fatigue, expressed by the Rating of Perceived Exertion (RPE), is a valid way to quantify fatigue or intensity of exertion [2]. RPE is strongly influenced by the magnitude of homeostatic disturbances caused by exercise, such as body temperature, arterial saturation, heart rate, breathing rate and muscle glycogen content. During exercise afferent signals are sent to the brain and interpreted in perspective of those anticipated during the exercise bout. Throughout the bout the current or “conscious” RPE is compared with the template (anticipated) RPE. If the conscious RPE is

interpreted as matching the template, it is classified as being acceptable and the workload remains constant. However, if the conscious RPE does not match that anticipated, the workload is modified until the RPE matches the template. Duration remaining in the event is the key comparison factor in deciding the present RPE [12].

Recent research in our laboratory [6,7] has shown that growth of fatigue is proportional to the relative duration remaining in the event, regardless of time trial distance or hypoxic challenges. These studies support the concept that pacing strategies are altered to match the conscious RPE to the RPE template. These studies also support the concept that athletes continuously reevaluate their level of fatigue throughout an exercise bout.

Exercise performance is influenced by differences in the fraction of inspired oxygen ($F_{I}O_2$). Hypoxia has been shown to impair, while hyperoxia has been shown to augment, maximal oxygen uptake (VO_{2max}) and exercise performance [1,8,9,10,11]. Exposure to blinded hypoxic challenges in the midst of an exercise bout, between 2-4km of 5km time trial (TT), revealed that both power output and arterial O_2 saturation decrease within 30 seconds of administration [6]. This suggests that arterial O_2 saturation and power output are closely linked. The rapidity of response supports the concept that there are centrally mediated signaling mechanisms as well as peripheral regulation.

The regulation of pacing strategy still remains poorly understood. Much of the research has focused on open-loop trials at fixed workloads, which disregard the normal PO variation in a typical race situation. Recent research [6] has looked at the rapidity of response to an unobvious hypoxic gas mixture in the middle of a TT. It is unclear from previous research whether the decrease in arterial O_2 saturation is serving as a signaling

mechanism to modify the exercise template (e.g. decrease power output) or whether they are only coincidentally correlated. The purpose of this study is to determine how rapidly power output and pacing strategy change after hypoxic air ($F_{I}O_2 = \sim 15\%$) has been administered 3 minutes prior to beginning a TT versus at the beginning a TT. We hypothesize pacing strategy will begin the same as the control condition. PO will differ when the body senses the difference in S_aO_2 . If the early power output is not downregulated by pre-exercise hypoxia, it will suggest that decreases in power output are mediated by peripheral effects of hypoxia rather than by direct central mediation.

METHODS

Subjects

The protocol was approved by the University of Wisconsin-La Crosse Institutional Review Board for the Protection of Human Subjects. Eight adult well-trained cyclists provided written informed consent and participated. Both male and female cyclists were recruited. Descriptive statistics of the subjects are presented in Table 1. Subjects were asked to avoid strenuous exercise within 48 hours of testing. Each subject completed an incremental exercise test to determine responses to maximal exercise and threshold workloads, a habituation 3km time trial and blinded, randomly ordered control (room air) and two experimental (hypoxic) 3km time trials.

Table 1. Descriptive statistics

	Male n=4	Female n=4
Height (cm)	182.0 \pm 4.5	166.3 \pm 14.8
Weight (kg)	83.6 \pm 4.2	67.4 \pm 7.5
Age (years)	31.5 \pm 9.9	22.8 \pm 2.2
Peak Power (watts)	375 \pm 73	263 \pm 14
VO2max (ml/kg/min)	61.0 \pm 10.2	52.9 \pm 5.3
VO2 VT (L/min)	2.96 \pm 0.19	2.31 \pm 0.74

Incremental Exercise Test

The test was performed on an electrically braked cycle ergometer (Lode Excalibur, Groningen, The Netherlands). The incremental exercise test protocol began at 25 W for 3 minutes and then increased by 25 W every minute until volitional fatigue. Oxygen uptake (VO_2) was measured using open circuit spirometry (AEI Technologies, Pittsburgh, PA). The peak VO_2 observed for a continuous 30 s period was accepted as the $\text{VO}_{2\text{max}}$. Heart rate (HR) was continuously monitored using radiotelemetry (Polar Vantage XL, Polar Instruments, Port Washington, NY). Upon completion of each stage, RPE was recorded using the Category Ratio RPE scale [2].

Time Trials

The time trials were completed on an electrically braked cycle ergometer (Velotron, Racermate, Seattle, WA). A sample protocol is shown in Figure 1. Upon arrival to the lab, the subjects completed a warm-up for 15 minutes: minutes 0-5 at 100 W, minutes 5-10 at 50%, minutes 10-12 at 75%, and minutes 12-15 at 50% of peak power output. Upon completion of the warm-up, the subjects were fitted with a breathing apparatus, which controlled whether the subjects were breathing from the bags or from the room. The control trial ($F_{\text{I}}\text{O}_2 = 0.2093$, $P_{\text{I}}\text{O}_2 = 159\text{mmHg}$) was bagged normoxic air; the experimental trials ($F_{\text{I}}\text{O}_2 = 0.15$) represented an inspired $p\text{O}_2$ at an approximate altitude of 2300m ($P_{\text{I}}\text{O}_2 = 123\text{ mmHg}$). This elevation represents the highest altitude at which the Olympic competitions have been held (Mexico City). The control and experimental 3km time trials were randomly ordered. The subjects were blinded to the composition of air in the bags. One of the experimental trials (the early desaturation TT) began 3 minutes after administration of the bagged air to ensure arterial oxygen

saturation had already decreased. In the other experimental trial (the late desaturation TT), the subjects were switched to bagged hypoxic air 30 seconds prior to the start of the TT. This was calculated through the pilot study to allow the subject to clear the room air from the hoses, so that hypoxic exposure began with approximately the first breath of the TT. The administration of oxygen was unobvious, because the subjects were seated on the bike, which was not facing the bags. The timing of the administration was also consistent between similar trials. Various feedback variables were available to the subjects during the trials: distance completed, elapsed time, momentary velocity, HR and PO. RPE was recorded every 300m by use of hand signals using the Category Ratio Scale [2]. Arterial oxygen saturation (S_aO_2) was measured by a fingertip pulse oximeter (Allegiance Oxi-Reader 2000, Allegiance Health Care, McGraw Park, IL).

Prior to the control and experimental time trials, a habituation trial was completed by every subject to familiarize the subjects with the distance of 3km, the procedures, and the equipment, including the respiratory mask, and to allow the subjects to become familiar to the slight increase in inspiratory resistance from the inspired bag system.

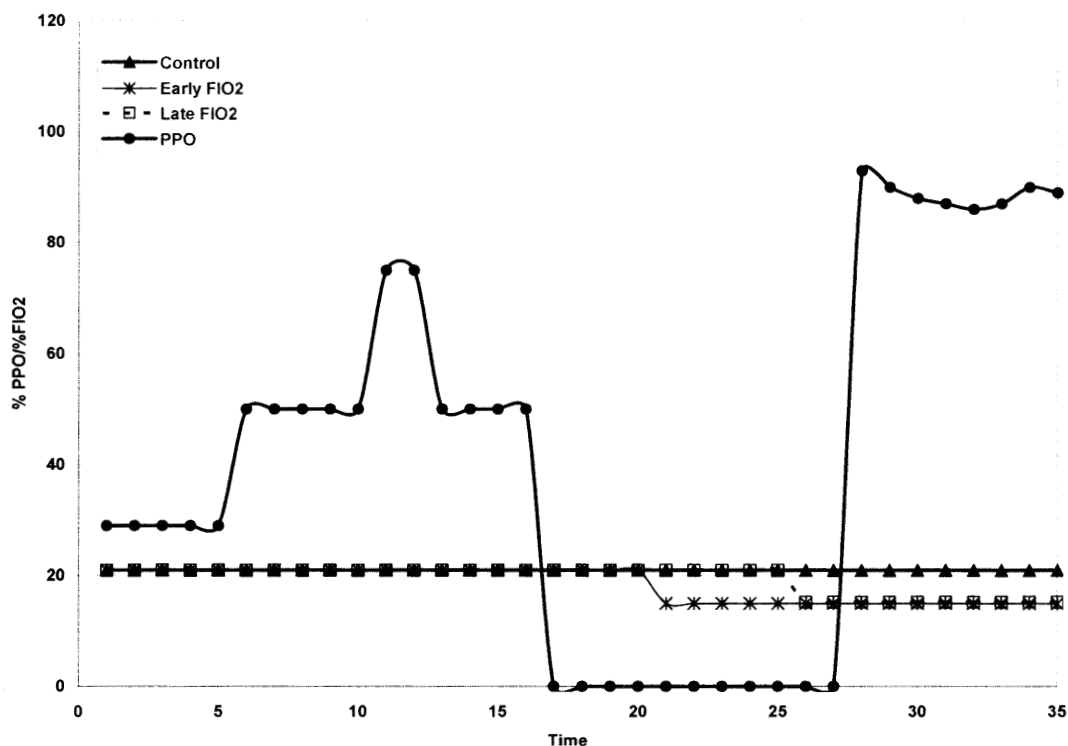


Figure 1. Protocol of Control and Experimental Conditions. The control, early F_{IO_2} and late F_{IO_2} lines represent the experimental manipulation of hypoxic condition. The PPO line represents the warm-up protocol, followed by the rest period and TT.

Statistical Analysis

The data were analyzed using repeated measures ANOVA to identify differences between the control, early desaturation, and late desaturation trials for PO, ventilation, RPE, S_aO_2 , and HR. The Tukey test was used to determine significantly different pairwise differences when justified by ANOVA. A P value of < 0.05 was accepted as a significant difference. We hypothesized that the initial power output would be constant in both trials due to the presence of a performance template.

RESULTS

The experimental manipulation caused a reduction in TT performance as seen by the increased duration of the 3km time trial, which supports the reductions seen in mean PO between the control and hypoxic trials (Table 2).

Table 2. Time trial mean results

	Control	Early	Late
Time (minutes)	4.96 \pm 0.50	5.06 \pm 0.48	5.09 \pm 0.45
Mean PO (watts)*	272.5 \pm 81.7	255.4 \pm 70.0	249.5 \pm 60.6
Max HR (bpm)	176.8 \pm 10.0	171.3 \pm 7.4	175.6 \pm 9.0
Max PO (watts)	318.8 \pm 77.3	315.6 \pm 81.3	311.6 \pm 67.8

* $p \leq 0.05$

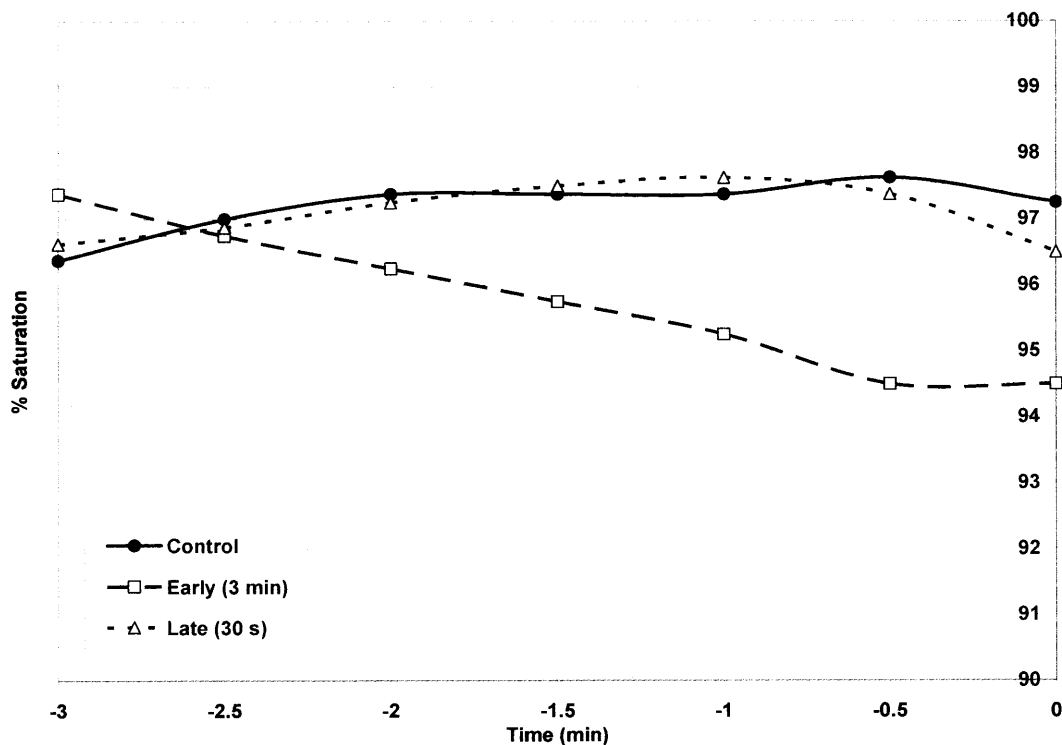


Figure 2. Pre-Exercise S_aO_2 .

The experimental manipulation of $F_I O_2$ applied in the early desaturation trial produced a significant decrease in pre-exercise S_aO_2 prior to the start of the time trial. There was no significant decrease in pre-exercise S_aO_2 in the late desaturation trial.

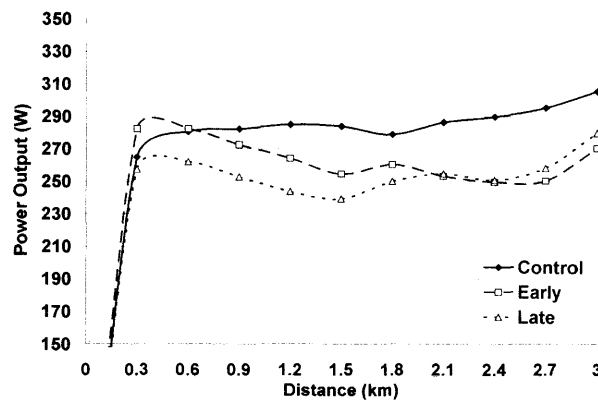


Figure 3a. Distance vs PO

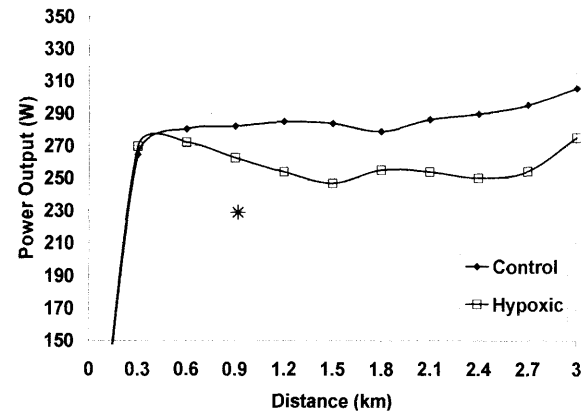


Figure 3b. Control vs Hypoxic PO

The pattern of PO between the early and late trials appeared to be similar (Figure 3a). Given that there were no apparent visual differences between the two hypoxic trials, we applied a statistical test to the early and late desaturation trials. No significant difference was seen between the trials; therefore, we pooled the early and late trials into a mean hypoxic trial. There were significant differences between the control and pooled hypoxic trials beginning at a distance of 0.9 km and continuing throughout the ride. The control versus combined hypoxic trials are presented in Figure 3b.

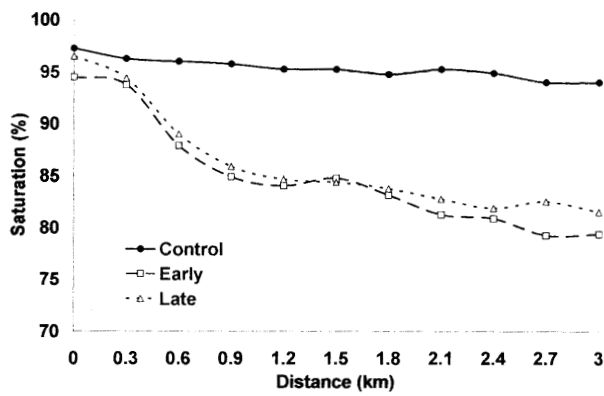


Figure 4a. Distance vs S_aO_2

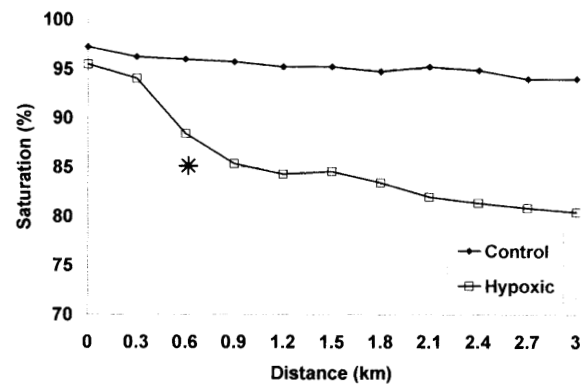


Figure 4b. Control vs Hypoxic S_aO_2

Except for the S_aO_2 at the beginning of exercise, the pattern of S_aO_2 between the early and late trials was similar during the early and late experimental trials (Figure 4). With the starting value eliminated, there were no differences between hypoxic trials. Therefore, the hypoxic trials were pooled and compared to the control trial. There was a significant difference in S_aO_2 beginning at 0.6km and continuing throughout the trial.

In pooled results between the hypoxic and control groups a significant difference was seen in S_aO_2 with a main effect for distance and condition x distance interaction. Post hoc tests determined the differences were apparent at 0.6km to the end of the TT.

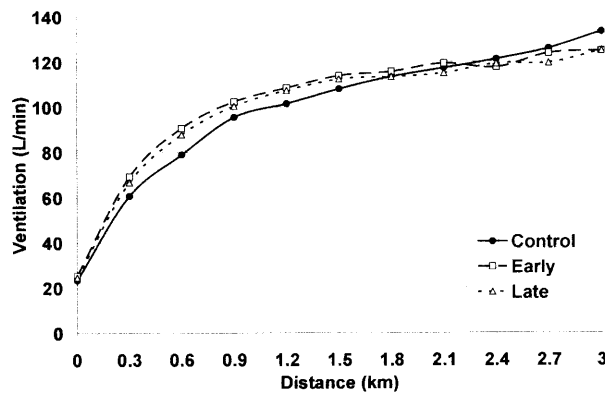


Figure 5a. Distance vs Ventilation

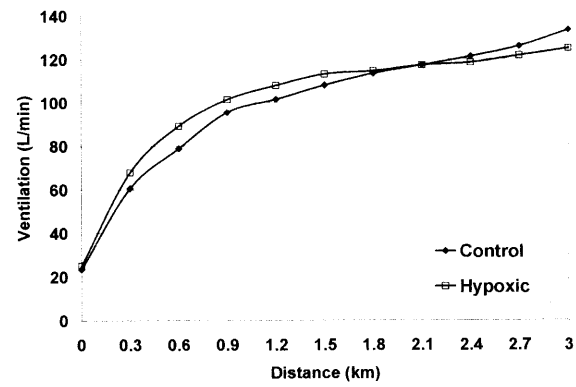


Figure 5b. Control vs Hypoxic Ventilation

Despite small significant differences in PO and S_aO_2 between all the time trials, the pattern of ventilation was similar, with no significant differences observed (Figure 5a). No significant differences were seen between the pooled hypoxic trials and the control trial for ventilation rates (Figure 5b).

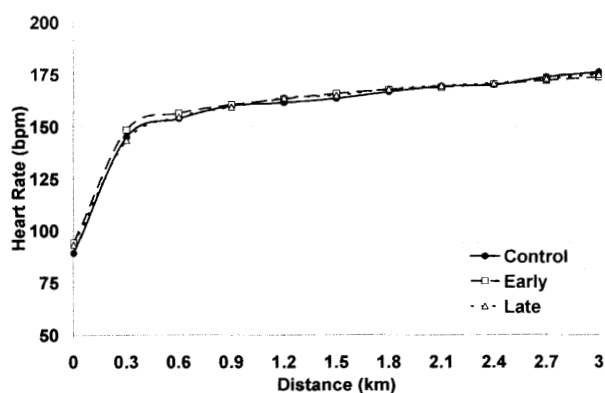


Figure 6a. Distance vs Heart Rate

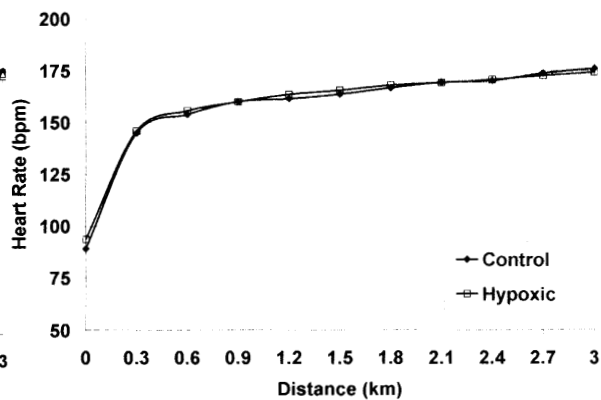


Figure 6b. Control vs Hypoxic Heart Rate

Regardless of PO and S_aO_2 changes, the pattern of heart rate was similar between the time trials, with no significant differences seen (Figure 6a). No differences were seen between the pooled hypoxic results and the control time trial in heart rate (Figure 6b).

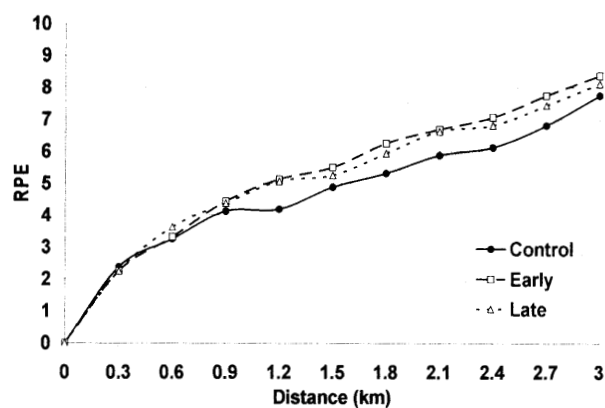


Figure 7a. Distance vs RPE

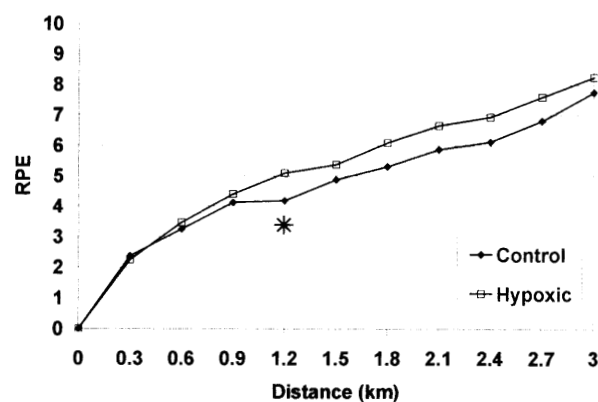


Figure 7b. Control vs Hypoxic RPE

Beginning at 1.2 km, RPE was significantly greater during the pooled hypoxic trials than during the control trial (Figure 7a). In pooled results (Figure 7b), a main effect for distance and a distance x condition interaction was observed. The difference between the pooled hypoxic results and the control trial became significant from 1.2 km and beyond.

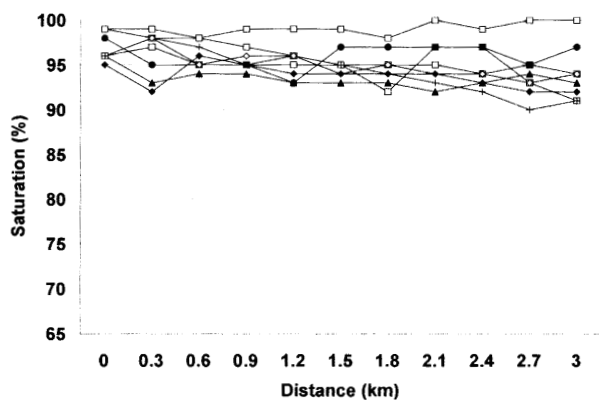


Figure 8a. Individual S_aO_2 Control.

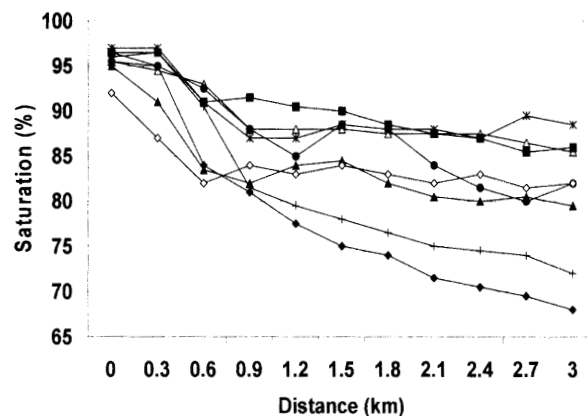


Figure 8b. Individual S_aO_2 during Hypoxic Trial.

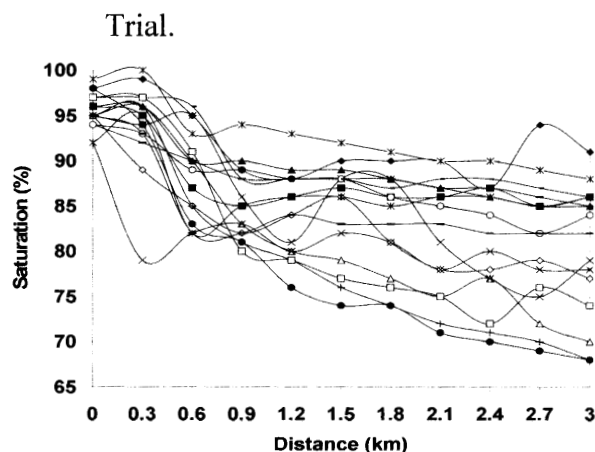


Figure 8c. Individual S_aO_2 during Early & Late Trials.

During the control trial, variance amongst individuals was uniform ranging between 94-98% at start to 91-100% S_aO_2 at the completion of the time trial (Figure 8a). More variance was seen in the pooled hypoxic trials, ranging from 92-97% at start and 68-88.5% S_aO_2 at completion of the time trial (Figure 8b).

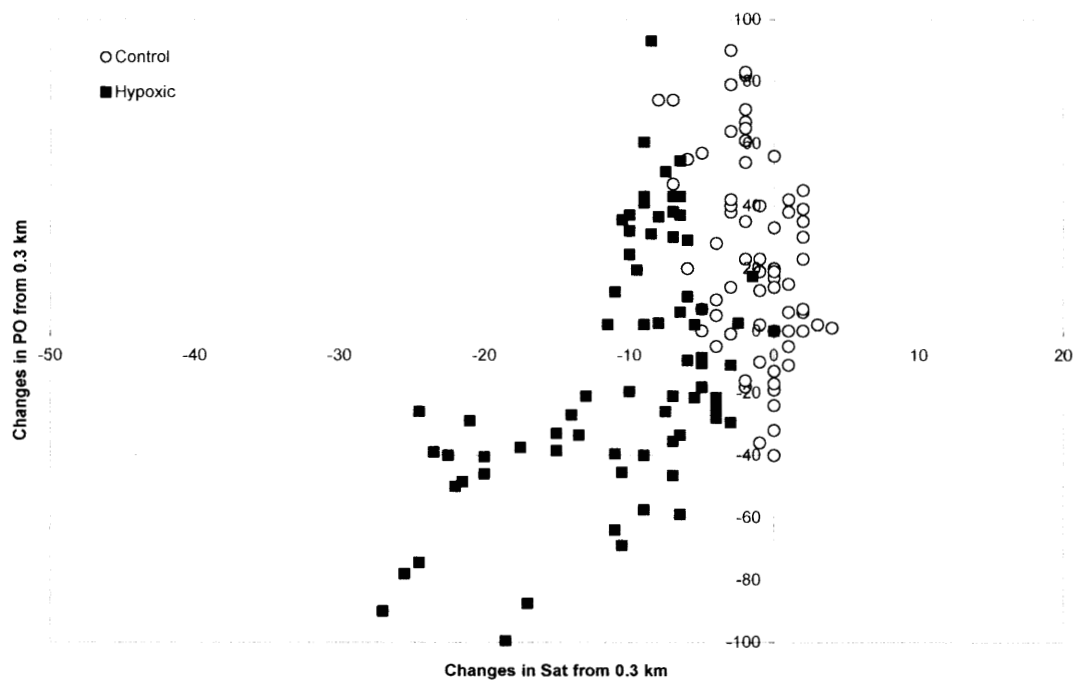


Figure 9. Control/Hypoxic S_aO_2 changes vs PO changes from 0.3km

There was a general trend toward decreases in PO after 0.3km with decreases in S_aO_2 . When the decrease in S_aO_2 was greater than 10%, there was a consistent pattern of decreases in power output (Figure 9).

DISCUSSION

We tested the hypothesis that the early PO would be the same at the start of the TT regardless of the $F_{I}O_2$ of inspired air prior to the trial, due to a strong pre-trial template. This was done to test the concept that % S_aO_2 was a direct controller of power output, as suggested by the results of Johnson [6]. By inducing an early reduction in % S_aO_2 in the early hypoxia trial, if % S_aO_2 were a direct controller of power output, the power output at the beginning of the trial should have been reduced. We induced significant changes in S_aO_2 prior to the hypoxic trials and confirmed our hypothesis that initial PO patterns would be similar between all trials. In fact, the cyclists' PO decreased significantly and then reached a plateau through the duration of the TT despite a continuing drop in S_aO_2 . In the pooled hypoxic results, the PO decline, seen at 0.9km, was preceded by a decline in S_aO_2 , seen at 0.6km.

We saw no initial effect on PO when the hypoxic condition was applied prior to the TT. Time for PO decline was approximately 40 seconds after the start of the trial. Johnson [6] saw PO decline at approximately 25 seconds after administration of hypoxic gas. The subjects in our study went from being at rest to setting their race pace, while the previous study administered the hypoxic gas in the midst of a TT. In both studies, the subjects are in different physiological states which could change the effect of decreases in % S_aO_2 as a signaling mechanism. Also, there may be differences in the training status of the subjects between studies.

The results of this study indicate that during simulated competition at altitude, a large PO reduction was not enough to alleviate the additional burden the hypoxemia causes on the cyclist, as seen by the increased RPE ratings. The increase in RPE pattern

suggests that the cyclists sense the disturbance or the change from the “normal template”. The growth of RPE during the pooled hypoxic results became different from the control trial at a distance of 1.2km, which followed both the decline of S_aO_2 and PO levels. In the results of Johnson [6], the decrease in power output during hypoxia was almost precisely adequate to maintain the normal pattern of increase in RPE during the TT. In the present data, although power output was decreased, RPE remained somewhat elevated, suggesting a dissociation between the sensed discomfort and the reduction in power output. This suggests a delay in the feedback vs anticipation element of the control of workload.

The present findings support the current literature [3,4,7] that RPE has scalar linear properties. The maximum RPE values (8.3/10) in the present study were lower than the maximum RPE value of 9.5/10 reported by Joseph [7], but similar to the maximum RPE rating of 18/20 seen by Eston [3]. Our findings may be lower due to the subjects being less familiar with the distance of 3km.

Since the hypoxic air mixture was normobaric, the PO decline cannot be due to an increase in air density. The applied condition does not simulate a true altitude environment, where there is not only a decrease in $F_I O_2$ but also a decrease in barometric pressure.

Experimental limitations include usage of a small number of sub-elite cyclists exhibiting a wide variety of training and fitness levels. Future studies may experimentally manipulate arterial oxygen saturation levels below 90% prior to the beginning of the TT to determine if blinded physiological changes can change the pre-race template by altering initial PO.

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APPENDIX A
INFORMED CONSENT

Informed Consent

Protocol Title: Rapidity of Response to Hypoxic Conditions during Exercise

Principal Investigator: Kayla Henslin, B.A.S.
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La Crosse, WI 54601
(608)785-8676

Emergency Contact: Kayla Henslin
(507)251-2031

- Purpose & Procedures
 - The purpose of my study is to determine the how long it takes the body to identify and change pacing strategy while breathing low oxygen concentrations during exercise.
 - Participation in this study will consist of 4 visits to the laboratory, including a maximum exercise test (test until complete fatigue), a practice 5km trial and three 5km time trials (simulated competition).
 - The total time requirement is 4 hours over the course of 4 weeks.
 - All testing will take place in the Mitchell Hall lab Room 225, UW-L.
 - During all tests, I will wear a snorkel-like mask to analyze my breathing or to administer my oxygen to breathe. I will also wear a heart monitor, strapped around my chest, to monitor my heart rate.
- Potential Risks
 - I may experience and substantial fatigue.
 - Individuals trained in CPR, Advanced Cardiac Life Support and First Aid will be in the laboratory. The test will be terminated if complications occur.
 - The risk of serious or life-threatening conditions is near zero in healthy individuals, like me.
- Rights & Confidentiality
 - My participation is voluntary.
 - I can withdraw from this study at any time for any reason without penalty.
 - The results from this study may be published in scientific literature or presented at professional meetings using grouped data only.
 - All information will be kept confidential through the use of number codes. My data will not be linked to any personally identifiable information.
- Possible Benefits
 - I and other athletes may benefit by gaining more understanding about how quickly the body identifies and paces itself with simulated altitude conditions.

Questions regarding study procedures may be directed to the principal investigator, or the faculty advisor, Dr. Carl Foster (608-785-8687). Questions regarding the protection of human subjects may be directed to the UW-La Crosse Institutional Review Board for the Protection of Human Subjects (608-785-8124 or irb@uwlax.edu).

Participant _____

Date _____

Researcher _____

Date _____

APPENDIX B
REVIEW OF RELATED LITERATURE

INTRODUCTION

Athletes compete in events that may take minutes or even hours to complete. During this time of competition, the body is put under stress to maintain homeostasis and to avoid metabolic disturbances. The athlete faces several challenges during trials, which change with every event including humidity, ambient temperature, competition in altitude or level of substrate availability. How an athlete paces himself in the face of these challenges seems to be a key aspect to success. Pacing strategy is regulated by a very complex system.

PACING STRATEGY

Pacing strategy is the individualized plan for completing a task in the shortest possible time. It is also described as the efficient use of limited energy sources, so that all energy stores are used prior to finishing the exercise bout, but not so early that a significant slowdown occurs [7]. Pacing strategy is determined by several factors including: individual capacities of the athlete, previous experience in training or racing, anticipation of duration, physiological feedback and central feedback. During self-paced exercise, the work rate is adjusted to prevent detrimental changes from occurring due to a disturbed homeostasis.

The current proposed model for pacing strategy is the anticipatory feedback-RPE model proposed by Tucker and Noakes [21]. This model incorporates anticipatory and feedback components. In self-paced exercise, the work rate can vary freely and many

factors influence the pace. The anticipatory component requires prior training, as well as other input such as skin temperature, muscle glycogen levels and motivation levels to set the pace. Prior to competition, the athlete develops a plan for increase in RPE based from expected duration, previous training and psychological input. Throughout the trial, conscious RPE is being compared to the RPE template. The athlete engages in self-talk to determine whether the current RPE is acceptable or not acceptable [16]. The power output is adjusted to match the template, with duration remaining in the event being the key guide. RPE quantifies fatigue, while the RPE template interprets the RPE [21].

In support of Tucker and Noakes [21], recent research in our laboratory [9,10] has found growth of fatigue to be proportional to the relative duration remaining in the event, regardless of time trial distance or hypoxic challenges. These studies support the concept that athletes continuously reevaluate their level of fatigue throughout an exercise bout.

Ansley [3] studied well-trained cyclists in three successive 4 km time trials with a 17 minute recovery between trials. The subjects were blinded to all feedback except duration completed. The major finding was the first and last time trials were completed in similar time without any form of feedback about their cycling speed, power output or elapsed time. All three trials had a peak power output at 60 seconds followed by a gradual decrease until the end of the trial. At the end of each trial (final 60 seconds), there was a significant increase in power output. This end spurt showed the subjects did not reach absolute fatigue during the trial and were able to increase intensity near the end of the trials. Hettinga [8] also studied a time trial of 4 km and found the same terminal acceleration. The study design was different than the study completed by Ansley et al. [3]; the subjects were assigned a different power output profile (even, submaximal, and

supramaximal paced) for the first half of the trial instead of being individually paced. No significant differences were found in the total time for the trials. Their results suggest that pacing involves “managing energy expenditure with its resulting peripheral fatigue so that no factor will be limiting before the end.”

In agreement with Hettinga et al. [8], Foster [7] previously found energy is expended so that the cyclist conserves anaerobic contribution throughout the duration of a competition. The findings suggest athletes monitor their energy resources and power output through a pacing strategy that preserves both aerobic and anaerobic sources to the end of the exercise bout. In support of these studies, [15] found pacing strategy may result from periphery feedback. This study investigated the role of muscle glycogen levels in pacing strategy in cyclists. In the carbohydrate-loaded trial, the subjects improved their time trial performance. Both trials began with the same power output, but after one minute in the depleted trial, PO was already approximately 14 watts lower than the non-depleted trial signaling an early change in pacing strategy. After completion of the trials, both depleted and carbohydrate-loaded had similar muscle glycogen concentrations regardless of differing starting values and time trial performances. This finding suggests a goal of pacing strategies is to regulate exercise so a certain amount of muscle glycogen concentration is available at the finish.

HYPOXIC CONDITIONS

Exercise with a differing oxygen fraction of inspired air ($F_{I}O_2$) has been a well-studied topic. It is well-supported that hyperoxia improves performance and oxygen uptake, while hypoxia impairs performance ([1,12,13,14,20]. Amann [1] studied the

effects of arterial oxygen content on peripheral muscle fatigue, which was assessed via changes in force output. Four 5km time trials were performed at four levels of arterial oxygen content. The quadriceps EMG was used to estimate central neural drive. When arterial oxygen content was changed from hypoxia to hyperoxia, central neural output increased, power output increased, and time trial elapsed time decreased. By changing the arterial oxygen content, performance time to exhaustion was negatively affected; however, no changes were seen with peripheral fatigue. This suggests exercise performance and power output are determined primarily by central motor output regulation. The decreased performance is seen to ensure the muscle fatigue does not cause detrimental changes.

In similar studies [2,19], EMG activity of the quadriceps was observed. After an exercise bout, mean force production from nerve stimulation decreased significantly and iEMG increased significantly during hypoxic, normal, and hyperoxic conditions. In the hypoxic trial, both the greatest reduction in force production and the greatest increase in iEMG activity were seen. This study indicates as the arterial oxygen content decreased, the power output continued to fall regardless of increased motor unit recruitment. This study indicates that during constant workload, peripheral locomotor muscle fatigue is highly sensitive to changes in arterial oxygen content. Taylor [19] also analyzed the iEMG of the m. vastus lateralis during two cycling trials. Each ten minute trial was with different oxygen fraction of inspired air: normoxia and hypoxia (11.6%). Post-trial, maximum voluntary isometric contractions were performed. In comparison to starting values, iEMG was significantly elevated at the end of the work trials; the iEMG recordings of the hypoxia trials were 15% higher than the normoxia trials. Hypoxia also

showed decreases in muscle fiber conduction velocity and isometric force, while increases were seen in electromechanical delay and the $iEMG_{max}/force$ ratio. This finding indicates peripheral fatigue, which is most likely caused by metabolic factors. In agreement with Amann et al. (2006b), these results show increasing motor unit recruitment to sustain power output in the hypoxia trials.

Tucker [20] tested the hypothesis that exercise performance is improved by increasing inspiratory oxygen tension partially because increases occur in muscle activation levels while the RPE does not change. Eleven subjects completed two 20km cycling time trials. Performance in the hyperoxia trial improved when compared to the normoxic trial; power output was maintained in the hyperoxic condition and decreased continuously in the normal condition. Plasma lactate concentration, heart rate, and RPE changes were similar throughout the trials; these similarities show the body regulates pacing strategy to account for metabolic differences. This study suggests the exercise performance improvement in hyperoxia might be because of the increased muscle activation due to a change in pacing strategy.

Peltonen [12,13] studied the effects of oxygen fraction in inspired air on rowing performance. Both studies used 2500m maximum exercise tests on a rowing ergometer, with three conditions of oxygen fraction of inspired air: hyperoxia (62.2%), hypoxia (15.8%) and normoxia (20.9%). The final rowing time was faster in hyperoxia and slower in hypoxia; VO_{2max} was greater in hyperoxia and lower in hypoxia. Maximal force and impulse decreased with hypoxic conditions.

Peltonen [14] studied the cardiorespiratory responses to different oxygen fraction of inspired air in cycling. The subjects cycled two progressive exercise tests: one of the

tests focused on respiratory data and the other focused on cardiac responses. Reductions in oxygen consumption, power output and cardiac output occurred in hypoxia; this was the first study to demonstrate maximal cardiac output is significantly lower in hypoxia. This decrease may be desirable because factors such as pulmonary and peripheral oxygen diffusion can limit arterial oxygen saturation.

Joseph [10] assessed the relationship between the rate of change in RPE and relative distance in time trials. The well-conditioned cyclists completed several time trials with distances varying from 2.5-10km and a time trial with a hypoxic challenge. From these trials, RPE was found to increase proportionally to relative distance completed regardless of the condition or length. The results suggest an athlete is continuously reassessing their fatigue during competition and comparing how they currently feel to how they expect to feel. The power output will either be increased or decreased to make the rate of increase of fatigue to be acceptable. In this study, power output decreased almost simultaneously with the decrease in S_aO_2 . This demonstrates that arterial oxygen saturation and power output are closely linked. Similar findings were seen by Johnson [9]. Upon administration of a blinded hypoxic challenge ($F_{IO_2} = 15\%$) during 5km time trials, the power output was significantly reduced in approximately 30 seconds. This study supports the concept of centrally mediated signaling mechanisms that quickly change power output when arterial oxygen saturation drops.

Clark [5] studied the effect of varying altitude on performance in cyclists. Four randomized simulated altitudes were used in a hypobaric chamber: 200, 1,200, 2,200 and 3,200m. As the altitude increased, the VO_{2peak} dropped and the distance covered in the 5-minute time trial decreased at a similar rate. In every trial regardless of the degree of

altitude, pacing strategy remained the same in those who had their first trial at 200 or 1,200m. While pacing strategy produced the ideal template when the cyclists had the simulated 200 or 1,200m trial first, the pacing strategy appeared to be misguided in those who had the 2,200 or 3,200 trial first. Cyclists were not as efficient at 3,200m when compared to 200 and 1,200m; higher cadences at the higher simulated altitudes caused the lower efficiency. Wehrlin and Hallen [22] had a similar study hypothesizing the $\text{VO}_{2\text{max}}$ would decrease linearly from sea-level to moderate altitude. To test this hypothesis, each subject performed a maximal exercise test on the treadmill at each simulated altitude in a hypobaric chamber: 300, 800, 1,300, 1,800, 2,300 and 2,800m. $\text{VO}_{2\text{max}}$ was shown to decrease linearly, which was linked to a 6.3% decrease every 1000m increase in altitude.

ENVIRONMENTAL CONDITIONS

Crewe [6] tested the rate of increase in RPE in different ambient conditions during five open-loop, fixed power output trials: cool (15°C) and hot (35°C). It has been shown that RPE increases as muscle glycogen decreases. Glycogen depletion takes place faster during high intensity and hot temperatures. One major finding in this study was that RPE increases linearly during exercise at a fixed intensity. The data shows the rate of increase in RPE successfully predicts the duration of exercise to exhaustion in a constant power output with different environmental conditions.

CONCLUSION

The regulation of pacing strategy still remains poorly understood, because of its complexity. Much of the research has focused on open-loop trials at certain workloads, which disregards PO variation in the typical race situation. Further research could focus on how rapidly the body changes pacing strategy and power output after administration of hypoxic air at the start of a time trial.

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