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## The effect of pacing strategies on oxygen kinetics and muscle hemoglobin status and energy system contribution in a 6 min exercise performance

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A thesis submitted in partial fulfillment of the requirements for the Master of Science degree in Kinesiology

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The effect of pacing strategies on oxygen kinetics and muscle hemoglobin status and  
energy system contribution in a 6 min exercise performance

(Thesis format: Integrated Article)

by

John David Murray

Graduate Program in Kinesiology

A thesis submitted in partial fulfillment  
of the requirements for the degree of  
Master of Science

The School of Graduate and Postdoctoral Studies  
The University of Western Ontario  
London, Ontario, Canada  
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## ABSTRACT

The purpose of this study was to compare the effects of two different pacing strategies during a 6 min exercise performance on average power output,  $\text{VO}_2$  kinetics, deoxygenation in the vastus lateralis muscle, energy system contribution, average power output and blood lactate concentration. Eight young, healthy, male subjects (age  $24 \pm 3$ ) completed two 6 min pacing strategies on a cycle ergometer; one bout was paced at a calculated fatigue threshold (FT) and the other began with a 12 s sprint and was paced 5 % below FT pace ( $5\%<FT$ ). Both strategies allowed individuals to use an incremental sprint over the last 90 s of the test to measure performance. The FT strategy yielded a significantly higher average work rate than  $5\%<FT$  ( $305 \pm 41$  W and  $281 \pm 423$  W, respectively) for the duration (0-360 s) of the pacing trials ( $p<0.05$ ). During the 90 s sprint performance, the FT strategy exhibited an increased  $\text{VO}_2$ , work rate, anaerobic energy system contribution and energy consumption ( $p<0.05$ ). No difference of post exercise blood lactate was observed between pacing strategies. In conclusion, the FT pacing strategy utilizing an even start protocol may be a more suitable strategy for a 6 min exercise performance.

### Keywords:

fatigue threshold;  $\text{VO}_2$  kinetics; pacing; near-infrared spectroscopy; rowing performance

## **CO-AUTHORSHIP STATEMENT**

This study was designed by G.R Belfry and J. D. Murray with input from the advisory committee (D. H. Paterson, P.W.R. Lemon). The majority of the data were collected and analyzed by J. D. Murray with the assistance of J.R. Leckie, and J.P. Nederveen. J.D. Murray wrote the original manuscript for the study. G.R. Belfry and D.H. Paterson provided financial support and lab support. G.R. Belfry provided editorial feedback.

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## LIST OF TERMS AND ABBREVIATIONS

5%<FT – traditional strategy, 12 s sprint to 5% below fatigue threshold  
ADP – adenosine diphosphate  
ATP – adenosine triphosphate  
CO<sub>2</sub> – carbon dioxide consumption  
CP – critical power  
FT – fatigue threshold  
GET – gas exchange threshold  
HHb – deoxyhemoglobin; measure of muscle deoxygenation  
H<sup>+</sup> – hydrogen ion  
Hz – hertz  
Hb – hemoglobin  
J – joules  
Km – kilometer  
L – liter  
La – lactate  
LDH – lactate dehydrogenase enzyme  
M – meter  
Min – minute  
Mb – myoglobin  
MHz – megahertz  
Mmol – millimol  
MRT – mean response time  
MVO<sub>2</sub> – mouth oxygen consumption  
N<sub>2</sub> – nitrogen concentration  
Nm – nanometer  
NIRS – Near-infrared spectroscopy  
O<sub>2</sub> – oxygen  
PCr – phosphocreatine  
PO<sub>2</sub> – partial pressure of oxygen  
Pi – inorganic phosphate  
Rpm – revolutions per minute  
S – second  
τ – time constant; time required to attain 63% of the steady-state response  
TD – time delay  
VCO<sub>2</sub> – carbon dioxide output  
VE – ventilation  
VO<sub>2</sub> – oxygen consumption  
VO<sub>2</sub> gain – VO<sub>2</sub> / work rate efficiency  
VO<sub>2</sub> max – maximal oxygen uptake  
W – watts  
W' – curvature constant, anaerobic energy system capacity  
WR – work rate

## Chapter1

### Review of Literature

Pacing, or pacing strategy, refers to the athlete's distribution of energy and work throughout a race. The manner an athlete paces oneself can have a tremendous impact on their performance. Differing pacing strategies adopted by athletes is dependent on event duration (1). Some have suggested that performances lasting less than three minutes will receive greater benefit from an all-out pacing strategy (7, 16, 26), whereas events greater than 3 minutes in duration may benefit most from an even paced strategy (1, 21, 22, 46). Elite rowers have adopted a parabolic shape profile strategy for 2 km races. The athlete(s) start the first quarter of the race with an all-out strategy, level off their speed for the next two quarters of the race, and for the last quarter of the race the athletes utilize a gradual sprint until the race is completed. This particular pacing strategy involves a variation of velocity through the exercise performance (45). Nevertheless, it has been suggested in research studies that any variation of velocity either above or below one's calculated average speed (fatigue threshold pace) may decrease overall performance (27). During the 2000 Summer Olympics, in Sydney, Australia, analysis of rowers performance that placed higher in the rankings yielded the least amount of boat velocity variation, regardless of: gender, boat type, or number of athletes in the boat (42).

The purpose of this investigation is to compare two different pacing strategies on a cycle ergometer that mimic a 2 km (6 min) rowing performance. The first strategy is the traditional strategy (5%<FT), fast start (12 s), and then a pace 5% below the fatigue threshold pace for  $\approx$  4 min 18 s, followed by an attempted increase in speed to the finish

at 6 min. The second strategy involves an even paced strategy with the same attempted incremental increase in speed to the finish from 4 min 30 s to 6 min. Energy system contribution, change in deoxygenation in the vastus lateralis muscle, oxygen kinetics over the first 180 s, power output, oxygen consumption and blood lactate concentrations before and after both pacing strategies will be compared.

### **1.1 Exercise Intensity Domains**

Three exercise intensity domains can characterize the metabolic and gas exchange response to exercises: moderate, heavy and severe. The moderate intensity domain refers to work rates below the lactate threshold (40). The speed of O<sub>2</sub> kinetics at moderate intensity is invariant with regards to work rate (40). However, it can be enhanced by training and slowed by chronic disease, inactivity and aging (11, 40). Heavy exercise refers to work rates above the lactate threshold and below one's critical power (the highest sustainable aerobic power output). Exercise in the heavy intensity domain results in the presence of lactate in the blood. However, with time the rate of increase in the blood is matched by its rate of removal. The severe exercise domain comprises a power output above the critical power and below one's VO<sub>2</sub> max (40). During severe exercise, VO<sub>2</sub> reaches its maximum and lactate rises inexorably until exercise is terminated due to fatigue (11).

### **1.2 Energy Systems**

There are two types of energy systems during exercise, the anaerobic and aerobic energy systems. The anaerobic system consists of two pathways, the alactic and the lactic

energy systems. The alactic system involves the splitting of stored phosphates from adenosine tri-phosphate (ATP) and phosphocreatine (PCr). This energy system is predominant in first 10 s of exercise. In the lactic energy system, lactic acid is a metabolic byproduct of the breakdown of glucose or glycogen to pyruvate in the presence of the lactate dehydrogenase (LDH) enzyme without the utilization of oxygen.  $H^+$  dissociates immediately from the lactic acid forming lactate. The lactic anaerobic system is working at its highest rate when energy from the alactic energy system is declining ( $\approx$  10 s) and the exercise duration is too short for the aerobic system to adequately supply the working musculature with ATP (40). Together the anaerobic energy systems can generate ATP at high rates but is limited by its metabolic byproducts (lactate,  $H^+$ , ADP, Pi) that are associated with fatigue (40).

The aerobic energy system utilizes the breakdown of fats and carbohydrates and relies ultimately on the consumption of  $O_2$  at the termination of the electron transport chain to complete this pathway. The aerobic energy has a great capacity to provide energy, but its rate of energy production is the slowest. The aerobic energy system has a majority contribution at work intensities below critical power. Any work performed above that intensity requires a significant contribution from anaerobic energy system (40, 50). The by-product of the aerobic energy system is carbon dioxide and water and is limited by muscle glycogen content (40).

During maximal exercise, the contribution of the aerobic and the anaerobic energy systems are dependent on the duration of the exercise bout (20, 21, 37). It has been suggested by that the 800,1500 and 3000 meter running events require 64/36%, 77/23 % and 86/14 % respectively for aerobic to anaerobic energy contribution for males (37, 38).

Duffield et al. (19, 20) also suggested that the 100, 200, and 400 meter running events require 21/79%, 28/72% and 41/59% respectively for aerobic to anaerobic energy contributes in male subjects. The majority of the running races up to 400 meters are predominantly anaerobic in nature (19, 20). The threshold between aerobic to anaerobic energy system contribution lies between 400-800 meters or 60-100 s (20, 37).

### **1.3 Exercise Efficiency O<sub>2</sub> gain**

The VO<sub>2</sub>/ work rate reflects the O<sub>2</sub> cost for a particular work rate, and has been utilized as a measure of efficiency (11). This relationship is affected by two factors: the O<sub>2</sub> uptake required for a given power output and the rate of VO<sub>2</sub> rise as a result of an increase in power output or the gain. The measurement can be taken at any point of time during exercise to measure the efficiency of aerobic metabolism. A value of 10 mlO<sub>2</sub>/min/W reflects the usual amount of VO<sub>2</sub> required at a work rate within the moderate intensity domain. An increased value reflects an increased amount of O<sub>2</sub> for a particular work rate (1).

Training may result in an increase in efficiency. Thus, a lower value for VO<sub>2</sub> gain would yield more efficient work during exercise by requiring less stress on aerobic metabolism to accomplish a given work rate (1).

### **1.4 Muscle deoxygenation: Near Infrared Spectroscopy (NIRS)**

Near Infrared Spectroscopy (NIRS) provides a measurement of oxygen bound to hemoglobin (Hb) and myoglobin (Mb) of the musculature over which the NIRS probe is placed. The concentrations of unbound to O<sub>2</sub> (deoxygenated) and bound to O<sub>2</sub>

(oxygenated) Hb and Mb can be used to monitor the balance of the O<sub>2</sub> delivery and O<sub>2</sub> utilization of the muscle under interrogation, i.e., muscle O<sub>2</sub> extraction. Differences in deoxygenation are associated with the partial pressure of oxygen (PO<sub>2</sub>). By comparing the rate of adjustment between pulmonary oxygen uptake with deoxyhemoglobin or level of muscle deoxygenation, the rate of O<sub>2</sub> delivery to O<sub>2</sub> utilization may be determined. The ratio of deoxyhemoglobin to pulmonary oxygen demonstrates how oxygen uptake at the cell is facilitated by blood flow and arterial oxygen content, thus O<sub>2</sub> delivery. If blood flow is adequate to the muscle, then the level of deoxygenation should match pulmonary oxygen uptake under normal conditions. If the ratio of deoxyhemoglobin to pulmonary oxygen uptake were to rise during a period of increased metabolic demand or increased exercise intensity, this signifies there is a decrease in perfusion of oxygen to target muscle, in which a compensatory response increases oxygen extraction. This ratio is a valuable tool in deciphering the relationship between O<sub>2</sub> delivery and utilization at the target muscle, specifically at a time point in which the aim is to accelerate the target muscle's adjustment to O<sub>2</sub> consumption (13).

### **1.5 Oxygen Kinetics**

The transition from rest to exercise results in an immediate change of energy requirements at the muscle cell (40). This immediate demand in energy by the muscle cell cannot be met entirely by oxidative metabolism. The initial requirement for ATP is met through anaerobic metabolism, and then aerobic metabolism (11, 40).

Oxygen kinetics quantifies the rate of change in pulmonary O<sub>2</sub> uptake at the muscle due to changes in metabolic demand, as a result of exercise and/or recovery from exercise



(40). A slow rate of adjustment to  $\text{VO}_2$  at the start of exercise increases the reliance of anaerobic metabolism to yield energy. The increased reliance on the anaerobic systems increase those metabolites associated with fatigue (ADP,  $\text{H}^+$ , Pi, lactate. Thus, fast kinetics may be advantageous in the delay of fatigue during a race (11, 40).

Oxygen kinetics is determined utilizing three distinct phases of the increase in  $\text{VO}_2$  at the onset of exercise. The first phase, representing the cardio dynamic phase, is associated with an sudden increase in  $\text{VO}_2$  due to the increased venous return caused by the muscle pump and increased right ventricular output, in which elevates pulmonary blood flow (11, 32, 40). A short period (~10-20 s) exists in which pulmonary oxygen uptake does not reflect actual muscle oxygen uptake. This delay leads to greater offloading of  $\text{O}_2$  in the muscle from Hb and the arrival of the same blood in the pulmonary vasculature (32). The second phase demonstrates a rapid increase in  $\text{O}_2$  consumption. The  $\text{O}_2$  kinetics during phase II largely reflects the events occurring at the muscle and reflected at the lung. This phase is the most examined phase of oxygen kinetics, as Phase II reflects the rate of adjustment of  $\text{O}_2$  at the working muscle and the degree of mitochondrial oxidative phosphorylation. Oxygen uptake kinetics of this phase is measured as a time constant ( $\tau$ ), which represents the time required to reach 63% of the Phase II  $\text{VO}_2$  response. The eventual steady state condition is reached at the time equivalent of  $4 \tau$ . The greater the  $\tau$  value, the greater the duration required to adjust to the steady state condition. The smaller the  $\tau$  value, the less amount of time required adjusting to steady state conditions and vice versa. (6, 12, 17, 40, 47, 53)

The third phase and following phase may have differing profiles. The  $\text{VO}_2$  profile of this stage is dependent on the intensity of exercise. If exercise intensity falls in the

moderate realm (below lactate threshold), then phase III corresponds to the point in which a steady state of  $O_2$  consumption is reached. The lactate threshold is the intensity in which a rise of blood lactate occurs. If work intensity continues to rise, an imbalance between pyruvate produced by glycolysis and its oxidation in the mitochondria of muscle cells is present. (40). Above the lactate threshold the  $VO_2$  slow component will eventually be observed. The  $VO_2$  slow component has been suggested to be the result of recruitment of the less efficient type II muscle fibers along with already active type I muscle fibers (28, 40). During the slow component,  $VO_2$  rises above levels that would have been predicted by relationship of  $VO_2$  to work rate, below the lactate threshold. The use of type II muscle fibers requires a greater amount of ATP for force production and a greater amount  $O_2$  cost for oxidative phosphorylation (40).

## **1.6 Fatigue**

Fatigue is the inability to maintain the expected or necessary power output for a given exercise performance (4, 23). The primary site of fatigue lies in the muscle cell. The etiology of fatigue during exercise may depend on the intensity of exercise and environmental conditions (4, 22). Short term, high intensity exercise involves the recruitment of type I and type II muscle fibers, a high degree of anaerobic energy metabolism, and high muscle contraction frequency (24). This increased anaerobic energy system involvement leads to an increased level of intra-cellular hydrogen ions, and inorganic phosphates (Pi) (18, 24), which results in an increase in hydrogen ion accumulation and a decrease in cellular pH that has been associated with inhibition of peak force output (18, 24). A high muscle contraction frequency may also lead to

disturbances in excitation contraction coupling. Specifically a block of the action potentials to the muscle cell, or an inhibition of calcium release and reuptake from the sarcoplasmic reticulum (24). It has also been observed that a high concentration of inorganic phosphate may decrease the maximal force of muscle fibers, specifically, the type II muscle fibers (22). Furthermore, high inorganic phosphates levels contribute to fatigue by inhibiting thin actin filament activation and by slowing the aforementioned reuptake of calcium by the sarcoplasmic reticulum (22, 28).

During prolonged sub maximal exercise, the causes of fatigue are much different (22, 35). Type I muscle fibers are primarily recruited in prolonged endurance exercise. The energy required for these fibers during sub maximal exercise is primarily from aerobic metabolism. The circulating levels of intracellular lactate, hydrogen ions, and inorganic phosphate levels remain stable (39, 41). The causes of fatigue are related to muscle glycogen depletion and low blood glucose (4, 14). It has also been suggested that depletion in muscle glycogen may trigger functional changes in the sarcoplasmic reticulum affecting  $\text{Ca}^+$  uptake and release. (4, 14, 22). Type II muscle fibers may be recruited during prolonged sub maximal exercise if the intensity reaches the severe domain (11, 40). During the severe exercise a  $\text{VO}_2$  slow component is observed, in which may result in the recruitment of less efficient type II muscle fibers. The slow component in the severe exercise domain fails to stabilize and rises until  $\text{VO}_2$  max is reached. The attainment of  $\text{VO}_2$  max may result in the termination of exercise (40, 53).

## 1.7 Critical Power

Critical power (CP) is the maximal power output in which a steady state can be maintained in pulmonary gas exchange, blood lactate, and blood acid-base balance (58). Therefore, CP is the highest theoretical work rate that can be maintained without a continuous and progressive contribution from anaerobic energy systems (40, 50). In actuality, it has been reported that CP cannot be maintained beyond approximately 30 min (10). Critical power also represents the boundary between the 'heavy' and 'severe' exercise intensities. The severe domain is between one's CP and  $\text{VO}_2$  max (40, 58).

A mathematical model utilizes critical power and the curvature constant ( $W'$ ) to predict the time to exhaustion while working at a particular power output, and/or, can predict the specific power output that will elicit fatigue over a specific duration of a work rate that is above CP (44, 58).  $W'$  is a measure of the finite amount of work that can be done above one's CP (58).

## 1.8 How is Critical Power and $W'$ measured?

Both CP and  $W'$  can be determined by a single, 3-min, all out test on cycle ergometer, in which power output can be measured for the duration of the test. The advantage to using a single bout critical power test is that it requires only one visit to the laboratory and the power profiles do not require linear or nonlinear regression analysis to determine this critical power output. Critical power is derived from the eventual plateau or leveling out of the power output. The last 30 s of the 3 min test is used to determine CP ( $W$ ) (57).  $W'$  refers to the sum of the work that was done above critical power and

over the duration of the entire three min maximal test (58). These parameters are utilized to predict one's Fatigue Threshold (FT), using equations 1, and 2 (58):

$$\text{Equation 1: } \text{Time} = W' / (P - CP)$$

$$\text{Equation 2: } P = W'/\text{Time} + CP$$

Where, "T" is time to exhaustion and "W" is the total work that can be done above one's critical power (Joules) J. "P" represents the calculated fatigue threshold pace, measured in watts (W).

### **1.9 Pacing Strategies**

The term pacing strategy refers to an athlete's work, speed and pattern of energy distribution during an exercise bout (1). Variations in pacing strategies can have a tremendous impact on exercise performance (25). Short duration events lasting up to 2 min may benefit most for an all-out pacing strategy (56). Whereas events greater than 2 min in duration may benefit from a pacing strategy where energy is distributed more evenly through the exercise bout (16).

Differing pacing strategies have been used. Negative pacing refers to a strategy that utilizes a negative split, or a strategy in which the athlete increases their velocity throughout the race and the latter part of the race is faster than the initial portion (1). This strategy may reduce the accumulation of fatigue-related metabolites, (hydrogen ions, inorganic phosphate and blood lactate) early on in the performance (2). This type of strategy has been observed to be successful in middle distance events that are >2 min (25). The lower initial power output may yield successful performance outcomes in these events due to the increased utilization of the anaerobic energy systems available towards

the end of the exercise bout (25). In other words, if one can manipulate another athlete away from their optimal pace, an individual with a greater anaerobic capacity may out sprint a superior aerobically conditioned opponent in the latter stages of the race.

Positive pacing refers to a strategy whereby an athlete gradually decreases velocity throughout the duration of the event (fast start) (1). Positive pacing may result reaching  $VO_2$  max sooner, and a faster accumulation of fatigue metabolites and an increased rate of perceived exertion. In essence, this is opposite to a negative pacing strategy (55). There are two main objectives for utilizing a positive pacing strategy 1) Athletes gain a lead on their competitors so one can adjust to any advances from their opposition (2, 11), 2) and faster  $VO_2$  kinetics of the working musculature. The advantages of faster oxygen kinetics are outlined later in this review.

The even pacing strategy involves utilizing a constant pace throughout the exercise bout (1). Thompson et al. suggest (55), for events greater than 2 min, (rowing, swimming, running) an even paced protocol is the best (55). Fukuba and Whipp support this model by utilizing CP to determine a participants fatigue threshold pace (27). Both CP and Fatigue Threshold are outlined later in this review.

Parabolic shaped pacing strategy exhibits a parabolic shaped profile in which participants begin the performance at a fast initial velocity, then decrease velocity in the mid portion of the race and then conclude the race at a faster velocity (30).

The most common pacing strategy for a two-kilometer rowing performance follows this parabolic shaped profile (52). Garland (30) studied the velocity of 2000 m rowing performances in elite rowers in the 2000 Olympic games, the 2000 and 2001 World Championships, and the 2000 and 2001 British Indoor Championships. He found

that rowers completed the first 500 m on average, 5.1 s faster than subsequent sections. The next 1000 m or the middle portion of the race was found to be the slowest portion. The last 500 m up to conclusion of the race was the second fastest component. These results were consistent among different crews (individual vs. team), positioning or rank, and gender. The fast initial start was also consistent in off the water rowing ergometer competitions (30, 52).

### **1.10 The Optimal Pacing strategy for 2 km (6 min) Rowing Performance**

The even paced pacing method may provide a better strategy for pacing in the elite rowers for their 2 km rowing performance. Fukuba and Whipp (27) examined the ability to make up lost time in endurance activity in a 5 km running performance. They theorized that high intensity running is reliant on performing at one's "fatigue threshold" (FT). They defined the FT as the tolerable power output for a specific time frame. They suggest that participants who ran below the FT pace at any time during the performance could not make up for the lost time. Participants who ran a faster pace than the FT pace could not maintain the velocity and would experience greater deceleration and lose time. They suggest that endurance exercise of >120 s may benefit the most by maintaining this constant pace at the fatigue threshold (27). Furthermore, fluid resistance is greater in water sports, such as, swimming and rowing, than on land sports (1), and as such, leads to an increased energy cost that results from these fluctuations in velocity (27). Thus, any decelerations or accelerations in boat speed may hinder exercise performances (54). An even paced strategy utilizing the fatigue threshold concept may be more suitable for an elite rower performing a 2 km race performance.

## **Chapter 2: The effect of pacing strategies on oxygen kinetics and muscle hemoglobin status and energy system contribution in a 6 min exercise performance**

### **2.1 Introduction**

Pacing may play a crucial role in achieving an optimal exercise performance. Rowing is a sport that involves a closed loop design. A closed loop design is an exercise performance in which the objective of exercise is to finish the race in the shortest possible time (1). The rowing shell with the shortest duration over a 2000 m distance wins the race.

The most common pacing strategy for a two-kilometer rowing performance follows parabolic shaped profile. Garland (30) studied the velocity of 2000 m rowing performances in elite rowers in the 2000 Olympic games, the 2000 and 2001 World Championships, and the 2000 and 2001 British Indoor ergometer Championships. They found that rowers completed the first 500 m, on average, 5.1 s faster than subsequent sections. The next 1000 m of the race was found to be the slowest portion. The last 500 m, up to conclusion of the race, was the second fastest phase. These results were consistent among different crews (individual vs. team), positioning or rank, gender or using an off the water rowing simulator (e.g. Concept 2 Rowing ergometer) (30).

The first 500 m may be completed at the fastest rate due to two factors. The first is to allow participants to gain the lead position, which will allow the participants to react to and adjust to any advances made by opponents. This may not be as advantageous for rowing performances completed on a rowing ergometer where opponents may be competing at another location. The second purpose for a faster initial work rate is to speed oxygen kinetics (30). The advantage of faster oxygen kinetics is that there is a



smaller O<sub>2</sub> deficit. A smaller O<sub>2</sub> deficit indicates a decreased degree of intracellular perturbation (lactic acid formation, decreased PCr). Conversely slower oxygen kinetics may predispose one to a reduced exercise tolerance later in performance (40).

It has been suggested (7) that in race performances up to 180 s the fast initial start may be the most beneficial. It has also been suggested that the fast initial start elicits faster VO<sub>2</sub> kinetics. This may spare the W' (anaerobic capacity) reserves and allow for additional non-oxidative energy to be used for a sprint phase at the end of the race. It has been suggested that this would allow for greater total work output for race performances up to 180 s (7).

Races longer than 180 s may benefit from an even paced strategy compared to a faster initial start strategy (56). Bailey et al. (7) examined the effect of a fast initial start on oxygen kinetics in both three and six minute cycle ergometer performances. They observed that short exercise performances of 180 s benefited from the faster initial start.

Fukuba and Whipp (27) theorized that high intensity running races would be more successful utilizing a constant power output at FT. They defined FT as the maximum tolerable work rate over a specific duration. They examined pacing strategies above and below one's FT in a 5 km running performances. The participants who ran below the FT pace could not make up for the lost time. The participants that ran at an initial faster pace than the FT pace could not maintain the velocity and dropped to a pace below the FT pace and lost considerable time (27).

The purpose of this investigation is to compare the effects two different pacing strategies, 6 min in duration, on a cycle ergometer. The first strategy mimics the traditional 2 km pacing strategy utilized by top coaches in Canada. Participants begin the

test with a 12 s all out sprint. The next phase (4 min 22 s) requires a reduced work rate that is 5% below each participant's fatigue threshold (2 s below their average 500 m split). The final 90 s prescribes, if possible, an incremental increase in power output with the last 30 s of the 6 min being an all-out sprint. The second pacing strategy is a constant power, evenly paced strategy. Participants begin this exercise bout at their specific fatigue threshold work rate. This is continued to 4 min 30 s. The prescription for the final 90s is identical to the traditional pacing strategy.

The current study will compare the effects of these two pacing strategies on  $\text{VO}_2$  kinetics, deoxygenation in the vastus lateralis, energy system contribution, average power output and blood lactate concentration. The hypothesis being tested is: 1) The fatigue threshold pacing strategy will yield a higher average power output compared to the sprint start performance and 2) The "traditional" pacing strategy will have a faster  $\text{VO}_2$  and HHb kinetic response.

## **2.2 Methods**

### *Participants*

Healthy male subjects ( $n = 8$ ; age,  $24 \pm 3$  yrs)  $\text{VO}_2$  max ( $4.31 \pm 0.96$  L/min) volunteered to participate in this study. All subjects participated in exercise at a club and/or intercollegiate level. All subjects were non-smokers with no known history of cardiovascular, respiratory, metabolic or musculoskeletal disease. All subjects were not on any medication that could have affected the physiological variables that were investigated. All subjects were informed of the procedures, risks, and potential benefits of the study prior to giving written consent to participate. Subjects were asked to arrive at

the laboratory in a rested state and to avoid strenuous exercise in the 24 hours preceding each exercise session. The Western University Health Sciences Research Ethics Board approved this study.

### *Experimental Overview*

The participants reported to the laboratory on four occasions over a two-week period with at least 48 hours rest between each visit. Upon completion of the incremental ramp test (visit 1) and the 3 min all out test (visit 2), subjects completed two randomly ordered pacing strategies in which pulmonary  $\text{VO}_2$ , blood lactate, vastus lateralis muscle deoxygenation (HHb), and work rate (W) were recorded.

### *Ramp $\text{VO}_2$ max Testing*

Prior to testing each subject was measured for appropriate seat, mouth piece and handle bar height. These adjustments were used for all four testing protocols. On the first laboratory visit, all subjects completed a ramp  $\text{VO}_2$  max test (25 W/min) to volitional fatigue on an electrically braked cycle ergometer (H-300-R Lode; Lode BV). This test was administered to determine the absolute  $\text{VO}_2$  max; gas exchange threshold (GET). GET was determined by gas exchange utilizing standard ventilatory indices. GET was defined as the  $\text{VO}_2$  consumption in which 1) pulmonary  $\text{CO}_2$  increased out of proportion to the increase in  $\text{VO}_2$ , 2) a systematic rise occurred in the ventilatory equivalent ( $\text{VE}/\text{VO}_2$ ), and 3) a systematic rise occurred in end-tidal  $\text{PO}_2$  with no simultaneous rise  $\text{VE}/\text{VCO}_2$  (33).  $\text{VO}_2$  define as gain was used to determine efficiency at max. The subjects initially performed three minutes of minimal load cycling (20 W) after which the

work rate increased to 25 W/min to volitional fatigue. The subjects cycled at a rate of 60-90 rpm. VO<sub>2</sub> was measured breath-by-breath and VO<sub>2</sub> max was determined by the average VO<sub>2</sub> over the last 15 s of the test. Fatigue was determined to occur when the subject was unable to maintain 60 rpm.

### *Critical Power Test*

On the second laboratory visit, all subjects completed a 3-min all-out critical power test (57). This test was used to determine the power duration parameters, W' and CP. Before the start of the test, subjects completed 3 min of unloaded cycling. During the last 5 s of the unloaded load cycling the subjects were asked to increase their rpm to over 100 rpm. At 180 s the subjects began a 3-min maximal performance test. The cycle ergometer was set using the linear mode (linear factor = power/ cadence squared) at 4 W/pedal revolution. Verbal encouragement was provided throughout the test. Subjects were not informed of the elapsed time to prevent pacing. The CP was determined to be the mean power output of the last 30 s of the test (57). W' was calculated as the total work completed above the calculated CP (58). W' is measured in joules. Watts can be converted in Joules by the following equation:

Equation 3:  $J = W \times s$

In the above equation, “J” relates to Joules, “W” refers to watts and “s” refers to time in seconds. The work rate for both 6 min pacing strategies was calculated using Equation 2 [ $P = (W'/T_{ime}) + CP$ ].

A work rate that would yield a time to exhaustion of 360 s (6 min) for a subject with a W' of 18 000 J and a CP of 220 W would be:  $(18000/360) + 220 = 270$  W.

*Total Work Completed:* The total amount of work performed was measured over the entire duration of the test. The work performed was calculated as the sum of the power (W) at each second that was above their critical power pace. These W were then converted to joules (see Equation 3).

*Anaerobic Contribution:* The  $\text{VO}_2$  gain was recorded over the last 30 s of each incremental ramp test to fatigue. The  $\text{VO}_2$  gain was multiplied by the work rate (W) during each pacing protocol for every 5 s point from 0-360 s. This value would represent the highest possible  $\text{VO}_2$  for a particular work rate. The predicted maximal  $\text{VO}_2$  was then divided by the actual  $\text{VO}_2$  at that particular time and multiplied by 100 % to yield a percentage of aerobic contribution. Anaerobic was calculated by subtracting the aerobic contribution from 100 %.

### *Pacing Strategies*

The two 6 min pacing strategies tests were administered in random order. Both trials were preceded by 3 min of 20 W cycling. During the last 5 s of the 20 W cycling subjects were asked to increase their rpm to over 100 rpm, similar to the critical power test.

1) Pacing at 5 %<FT: Following the 3-minute 20 W exercise, subjects performed a 12 s all out sprint against the fixed linear mode resistance (4 watts/pedal revolution). This was followed by 4:18 s in which the power output (W) was set at 5 % below the subject's calculated 6-minute FT pace. During the last 90 s of both pacing strategies the subjects pedaled against the same linear resistance mode (4 W/Rev) as the resistance during the 3 min all out test. Participants were instructed to complete a progressive sprint over the last 90 s to the finish of each trial. The specific instructions were “with 90 s to 60 s remaining

go at a pace that was “faster” than their previous pace. Over the next 30-s interval, go faster than the previous 30 s. The last 30 s interval is to be an all-out sprint to the finish. The 5%<FT strategy was based on a Canadian national rowing coaches’ preferred strategy for a 2 km performance.

2) Pacing at Fatigue Threshold: The performed their predicted 6-minute fatigue threshold work rate up to 4 min 30 s. After 4:30 s subjects performed the final 90 s with the identical instructions given before the 5% below FT trial.

### *Data Collection*

Gas exchange was measured measurements were similar to those previously described (5). The inspired and expired flow rates were analyzed by a low dead space (90mL) bidirectional turbine. (Alpha Technologies VMM 110). The turbine was calibrated before each test using a 3 L syringe. Inspired and expired gases were monitored at the mouth and analyzed for concentrations of O<sub>2</sub>, CO<sub>2</sub> and N<sub>2</sub> by a mass spectrometer (Innovision, Amis 2000, Lindvedvej, Denmark). Each subject wore a nose clip to cut off nasal airflow. The delay between volume and gas concentration was accounted for by measuring by the time delay for a square wave bolus of gas to travel from the turbine transducers through a capillary line to analysis by the mass spectrometer. Flow volume data and gas concentration data were then transmitted to the lab computer. The lab computer built a profile of each breath by aligning the gas concentration information with the inspiratory and expiratory volume recordings. Breath by breath gas exchange was determined from the algorithms developed by Beaver et al. (8).

Muscle deoxygenation of the vastus lateralis muscle was monitored continuously, during all trials, using frequency-domain multi-distance NIRS system (Oxiplex TS, Model 95205, ISS, Champaign, IL, USA). The NIRS machine was calibrated before each trial commenced; the probe was turned on 20 min period prior to each test to allow the system to stabilize. The NIRS probe was placed on the flexed vastus lateralis muscle with the left knee completely extended and with the heel resting on a level surface. The probe was placed between the lateral epicondyle and the greater trochanter of the left leg. An elastic strap was tightened completely around the left thigh in order to prevent movement and ensure the probe was securely attached. A black vinyl sheet was securely placed on top of the probe to prevent loss of Near-Infrared light and any intrusion of external light.

The NIRS system was made up of a single channel with eight laser diodes that operated at two wavelengths ( $\lambda = 690$  and  $828$  nm, four at each wavelength) which were pulsed at 110 MHz and utilized a photomultiplier tube. The NIRS probe was connected to the photomultiplier tube and the laser diodes. The NIRS probe contained one detector fiber bundle and two parallel rows of light emitting fibers. For both wavelengths, the source-detector separations of the NIRS probe was 2.0, 2.5, and 3.5 cm. Data were stored and collected at an output frequency of 25 Hz, however, were reduced to 1 s. bins for analysis within this study.

Power Output data was inputted into the Lode workload programmer box and recorded using the breath M program. Power output data was measured following each breath.

Blood lactate data was measured prior to testing and exactly 2 min after every trial. Blood was drawn using ACCU-CHEK Safe-T-Pro Plus sterile, single use lancing

device and was measured by SensLab GmbH Lactate SCOUT blood lactate analyzer. Prior to the use of the lancet, a rubbing alcohol swab was used to sterilize the left index finger.

### *Data Analysis*

Gas exchange data were edited by the removal of deviant data points that lied outside of 4 standard deviation points of the mean. The  $\text{VO}_2$  data for each trial was interpolated to 1 s intervals. The  $\text{VO}_2$  data were then time aligned so that time zero represented the completion of the 3-min load less exercise and the start of each trial. The  $\text{VO}_2$  data was also averaged to 5 s intervals for the critical power and two pacing strategies in order to provide a single average time response for each subject. The rate of oxygen uptake was measured in 30 s increments up to 180 s, as opposed to calculating the  $\tau$  value.

The HHb data (obtained from the NIRS probe) was time aligned and averaged to 5 s for each pacing protocol. The Baseline HHb and  $\text{VO}_2$  values for all trials were determined by the mean value 60 s before the start of test during load less cycling.

### *HHb/ $\text{VO}_2$*

The HHb and  $\text{VO}_2$  data was recorded and normalized for each subject (0 % represented the 20 W baseline value and 100 % represented the maximum HHb and  $\text{VO}_2$  data point recorded for each test) (46, 48). The data was averaged to 5 s bins to assess the rate of adjustment for HHb and  $\text{VO}_2$  (53).

### *Work Rate*



Work rate data was recorded over the entire duration of the pacing trials, incremental ramp test, and critical power test. Work rates for all tests were recorded at each breath taken by the participant. The work rate of the pacing trials and critical power test were then interpolated to 1 s intervals. The work rate over the final 30 s for the 3 min all out test was averaged to determine each participant's critical power work rate. The pacing trial work rate data was averaged to 5 s for each subject and each pacing trial.

### *Statistical Analysis*

Statistical analysis was performed using Sigma Plot 11. Differences between both pacing strategies from the beginning to the conclusion of trial were compared on the basis of NIRS (HHb), work rate,  $\text{VO}_2$  consumption, work output (J) and percentage anaerobic contribution. The data were analyzed using two way repeated measures ANOVA. The pacing strategies were compared between each other for every 30 s interval (NIRS, work rate,  $\text{MVO}_2$ ) and 5 s interval. (% Anaerobic contribution, energy consumption) The Holman-Sidak method was used for pairwise multiple comparisons for each interval. Glantz (31) describes the Holman Sidak as the recommended multiple comparison test to use after an ANOVA. Data collected for post-exercise blood lactates were compared between each pacing strategy and analyzed using a paired t-test.

## **2.3 Results**

Subject's physical characteristics and aerobic parameters (derived from the incremental ramp test to fatigue) are presented in Table 1. Power output data derived

from the 3 min all-out-test including: critical power,  $W'$ , and the calculated power outputs for both pacing strategies are also included in Table 1.

### *Work Rate*

Work rate was recorded throughout both pacing strategies (see Figure 1). Work rate was then calculated similar to both  $VO_2$ , and HHb in 5 and 30 s increments from 0-360s, following baseline exercise. The work rate over the first 30 s of 5%<FT was higher than the same duration of the FT strategy ( $396 \pm 200$  W compared to  $295 \pm 5$  W respectively ( $p<0.05$ )). There was no statistically significant difference from 30s to 300s between pacing strategies. The work rate of the FT strategy was higher than the 5%<FT from 300 – 330 s and 330-60 s ( $284 \pm 22$  W compared to  $272$  W  $\pm 12$  and  $339 \pm 29$  compared to  $272 \pm 10$  watts ( $p<0.05$ )). Finally, mean work rate over the total 360 s exercise duration was higher in FT compared to 5%<FT ( $305 \pm 41$  W compared to  $282 \pm 43$  W).

### *$VO_2$*

$VO_2$  data are presented in Figure 2 as group means for every 5 s increments (0-360 s) for both pacing strategies. There was no statistically significant difference observed in the baseline  $VO_2$  for both pacing trials. Mean  $VO_2$  over the first two 30 s intervals was greater ( $p<0.05$ ) during the 5% below pacing strategy compared to the fatigue threshold ( $2.40 \pm 0.62$  L/min compared to  $2.06 \pm 0.32$  L/min for the first 30 s and  $3.30 \pm 0.08$  L/min, compared to  $2.70 \pm 0.20$  L/min from 30 – 60 s). Over the last 90 s of  $VO_2$  was higher in the FT strategy ( $P<0.05$ ); (FT pacing strategy;  $4.22 \pm 0.10$  L/min,  $4.55 \pm 0.17$ ,  $4.70 \pm 0.14$  for 270-300 s, 300-330 s, 330-360 s; 5%<FT was;  $4.07 \pm 0.07$  L/min,

4.33 ± 0.06, 4.28 ± 0.56 for 270-300 s, 300-330 s, 330-360 s. There was no significant difference in VO<sub>2</sub> between 60 s - 270 s between pacing strategies. The rate of oxygen uptake was measured in Table 2 as group means for every 30 s increment up to 180 s for both pacing strategies. The 5%<FT strategy was significantly higher at 30 s and 60 s compared to the FT strategy (14.5 and 17.80 L/min, compared to 11.06 and 14.50 L/min).

### *HHb*

HHb data (Figure 3) was measured as a percentage of the baseline value to the maximum HHb recorded value (averaged over 5s intervals) throughout both pacing strategies. The values were also analyzed, similar to the VO<sub>2</sub> data, in 30s increments (0-360s.). HHb was higher over the first 30 s of 5%<FT (5%<FT 65.61 ± 25.3%, FT 48.9 ± 23.4 %; p<0.05).

### *HHb/VO<sub>2</sub>*

VO<sub>2</sub> was expressed in the same fashion as HHb (outlined above) and recorded in 5 s increments. The HHb/VO<sub>2</sub> ratio can be observed in Figure 4, the ratio was significantly higher for the 5%<FT strategy from 0-20 s of exercise. The ratio compared at 5 s, 10 s, 15 s and 20 s of 5%<FT to FT strategy was: 3.7 % ± 1 % compared to 1.5 % ± 0.5 %, 3.5 % ± 0.8 compared to 2.2 % ± 0.6, 3.3 % ± 0.5 % compared to 2.2% ± 0.3 %, 3.0 % ± 0.4 % compared to 2.3 % ± 0.2 %, respectively. Thus, the level of muscle deoxygenation to accomplish a given VO<sub>2</sub> during this period of time was greater than later in exercise. The ratio of 1.0 indicates a steady state HHb/VO<sub>2</sub> ratio has been reached (46).

### *Energy (W') Consumption*

The mean W' available for both pacing conditions was 15700 J  $\pm$  5239 J. The quantity of the total W' utilized over the first 30 s for the 5%<FT and FT was 5239 J  $\pm$  1119 J. and 1256 J  $\pm$  202 J ( $p < 0.05$ ) respectively. The quantity of the total W' utilized from 30 to 300 s for the 5%<FT and FT was 7342.3 J  $\pm$  3845.5 J 11700.5 J  $\pm$  3204.9 J respectively. The quantity of the total W' utilized from 300-360 s for 5 %<FT and FT was 2296 J  $\pm$  3597 J and 5705 J  $\pm$  4642 J ( $p < 0.05$ ). Expressed as percentages of the total W' the first 30 s of 5 %<FT was 33%  $\pm$  6%, compared to 8%  $\pm$  2% for FT. From 30 to 300 s the percentages were 46%  $\pm$  21% for 5 %<FT compared to 74%  $\pm$  16% for FT and for the final 60 s, the 5 %<FT strategy was utilized approximately 14.6%  $\pm$  20%, compared to 36%  $\pm$  24% for the FT strategy ( $P < 0.05$ ).

### *Blood Lactate*

Blood lactate was measured prior to testing and two minutes post-test. There was no significant difference observed between pre-exercise and post exercise blood lactate levels between the pacing strategies ( $p = 0.225$ ) (see Table 1).

### *Anaerobic Energy System Contribution*

The anaerobic energy system contribution (see Fig. 5.) was significantly higher in the 5%<FT strategy during the first 10 s of exercise, 100 %  $\pm$  5.5, compared to 72%  $\pm$  13% the FT strategy ( $p < 0.05$ ). The FT strategy yielded a higher anaerobic contribution from 25 s – 45 s, (50 %  $\pm$  8.5) compared to 26 %  $\pm$  16 % ( $p < 0.05$ ) for the 5%<FT. The FT strategy was also significantly higher than the 5%<FT traditional strategy at 80, 95,

260 s:  $30 \pm 6.2\%$ ,  $28 \pm 9\%$ ,  $5.9 \pm 11\%$  compared to,  $9\% \pm 23.5\%$ ,  $9\% \pm 21\%$ ,  $0\%$  respectively. During the final 90s incremental sprint, the FT strategy was significantly higher than the traditional strategy at 305, 315, 355, 360 s.:  $6\% \pm 19\%$ ,  $-13\% \pm 33$ ,  $3\% \pm 24$ ,  $15.5\% \pm 14\%$ ,  $17\% \pm 24$ , compared to  $0\%$  for 305, 315, 355, 360 s for the 5%<FT strategy (see Fig 6).

Subject	Height cm	Weight Kg.	VO <sub>2</sub> Max (L•min <sup>-1</sup> )	GET (L•min <sup>1</sup> )	CP (Watts)	W' (Joules)	FT Pace (Watts)	5%<FT (Watts)
1	190	96	5.07	2.43	207	18200	257	244
2	177	86	4.88	2.31	272	19123	325	309
3	185	104	4.43	2.34	264	19907	319	304
4	178	69	3.52	2.01	180	13976	219	209
5	183	101	3.49	1.93	217	15736	261	248
6	180	95	4.42	2.51	282	11022	312	296
7	190	98	5.35	2.50	290	15306	333	316
8	188	77	3.43	2.52	216	12334	250	238
Mean	184	90	4.32	2.31	241	15700	284	270
SD	$\pm 5$	$\pm 12$	$\pm 0.76$	$\pm 0.2$	$\pm 41$	$\pm 3207$	$\pm 43$	$\pm 40$

Table 1. Physical characteristics, response to incremental ramp test and critical power test. Values are expressed individually and as a mean  $\pm$  SD for: Height, Weight, VO<sub>2</sub> Max, Gas Exchange Threshold, Critical Power, W', FT Pace, 5%<FT Pace.

Table 2 – Mean VO<sub>2</sub> response from 0-180 s for both pacing protocols, measured in 30 s increments. Values are represented as group means for each pacing protocol and the difference between pacing strategies (p<0.05).

<b>Time</b>	<b>FT(VO<sub>2</sub> l/min)</b>	<b>5%&lt; (VO<sub>2</sub> l/min)</b>	<b>% Difference</b>
0-30 s	11.06	14.5	24 *
31- 60 s	14.50	17.80	19 *
61- 90 s	17.16	19.76	13
91- 120 s	18.50	20.69	11
121 – 150 s	18.51	21.2	13
151 – 180 s	20.7	22.3	7

\*

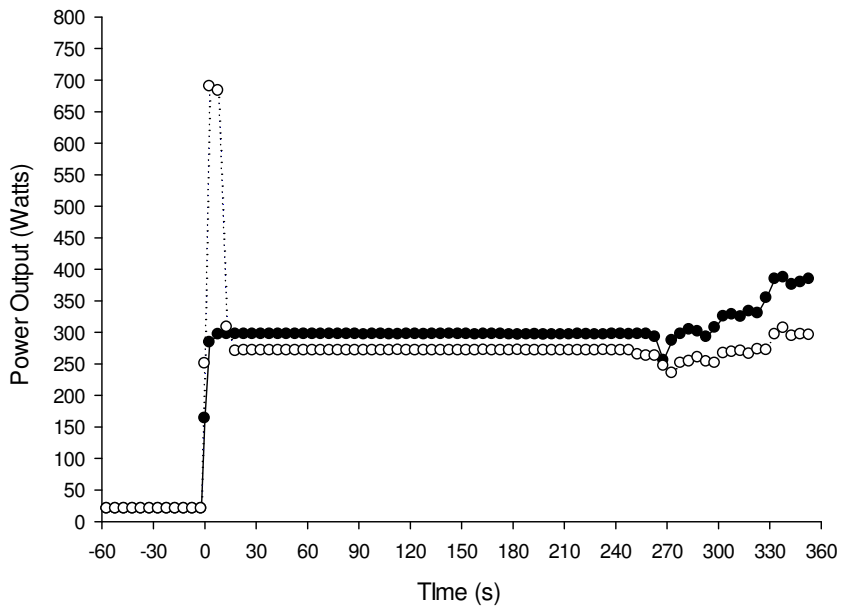


Figure 1: Mean power outputs of the two pacing (FT and 5%<FT) strategies, in 30 s increments. The mean power output over the duration of each test was significantly higher in the FT strategy compared to the 5%<FT strategy ( $305 \pm 41$  W vs  $282 \pm 43$  W) ( $p < 0.05$ ).

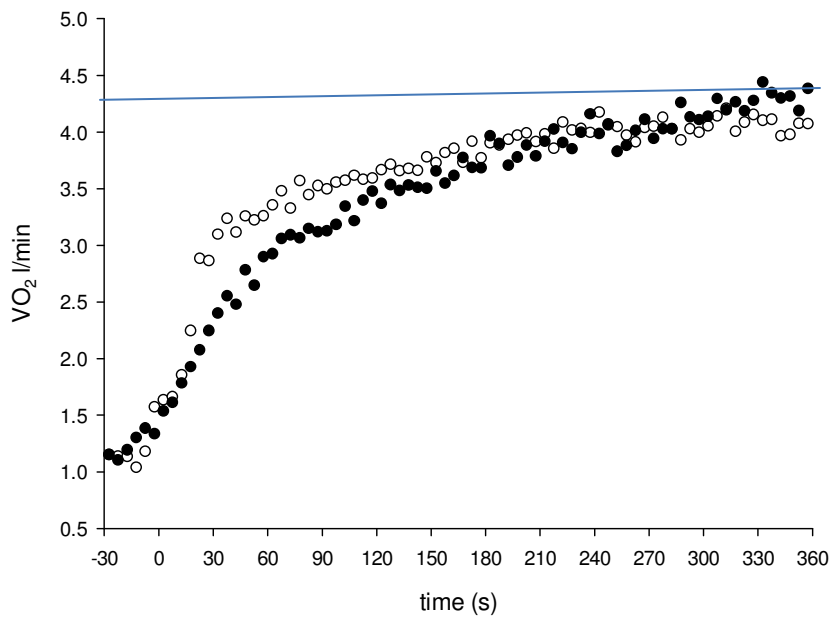


Figure 2: Mean VO<sub>2</sub> response from baseline cycling to end of exercise. Open circles represent FT strategy, closed circles represent 5%<FT strategy. Solid line represents the mean VO<sub>2</sub> max for all participants ( $4.32 \pm 0.76 \text{ L}\cdot\text{min}^{-1}$ )



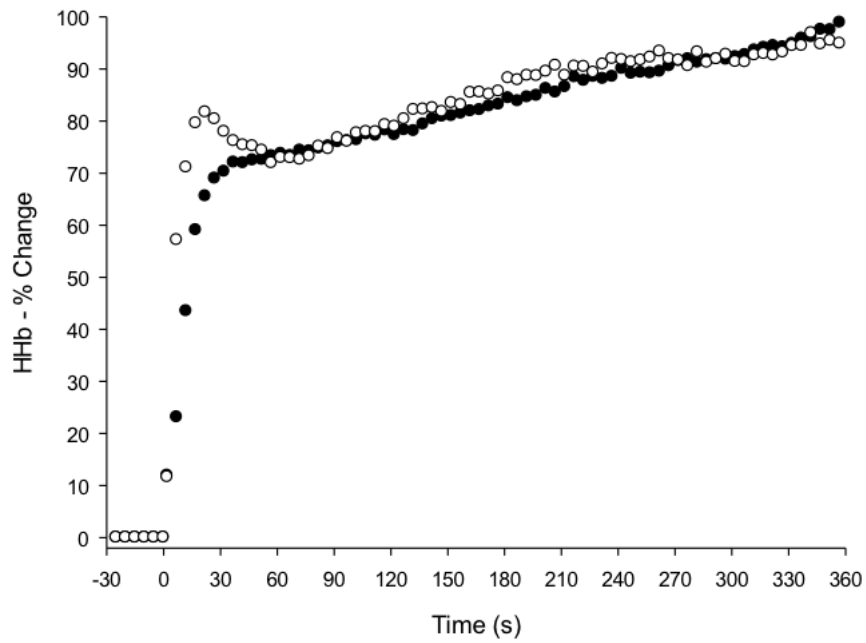


Figure 3: Mean HHb response from baseline cycling to end of exercise, measured in 5 s increments. Open circles represent 5%<FT strategy, closed circles represent FT strategy. 5%<FT strategy was significantly higher during first 30 s and from 120-230 s ( $p < 0.05$ ).

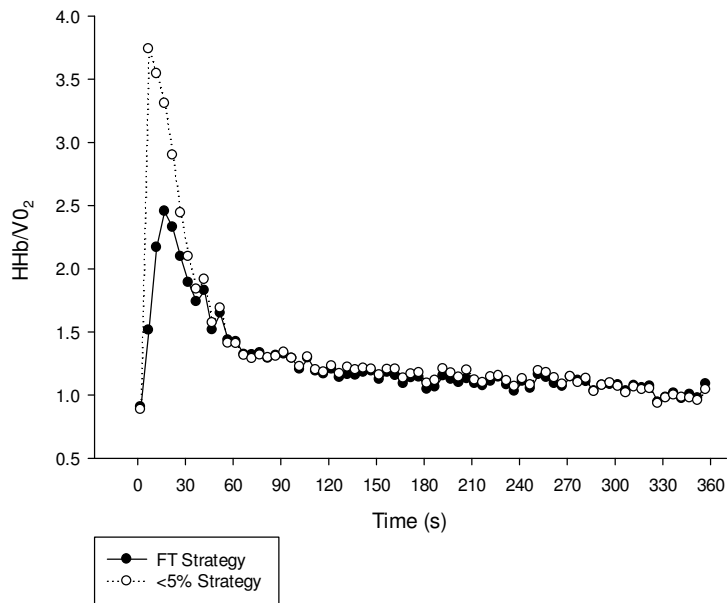


Figure 4: Mean HHb/VO<sub>2</sub> response from exercise onset to 360 s, measured in 5 s increments. Open circles represent 5%<FT strategy, closed circles represent FT strategy. HHb/VO<sub>2</sub> was significantly higher during the first 30 s of exercise for 5%<FT ( $p < 0.05$ ).

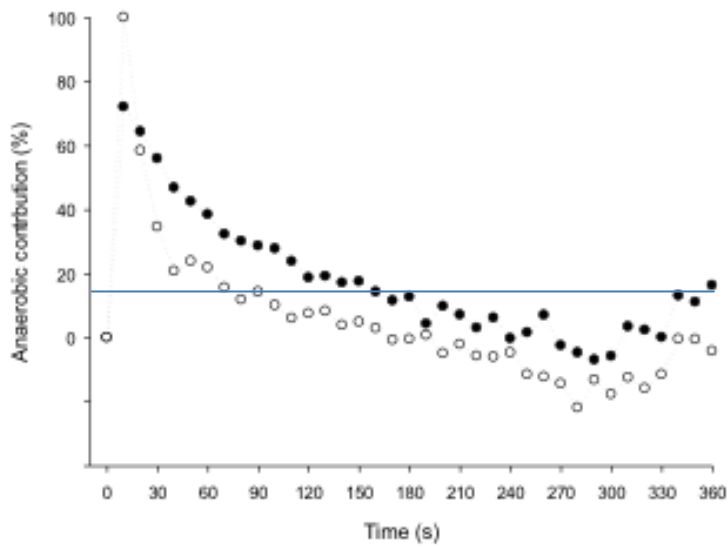


Figure 5: Mean anaerobic contribution from exercise onset to 360 s, measured in 5 s increments. Open circles represent 5%<FT strategy, closed circles represent FT strategy. Anaerobic contribution was significantly higher during first 30 s for 5%<FT but was significantly higher at: 25-45 s, 80, 95, 260, and 305-360 s ( $p < 0.05$ ). Solid line represents boundary between anaerobic and aerobic energy contribution.

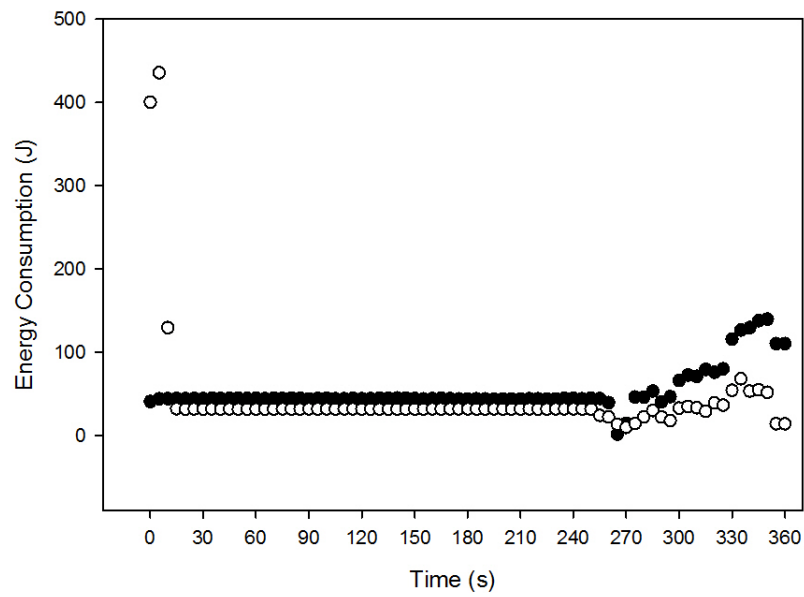


Figure 6: Mean energy consumption ( $W'$ ) from exercise onset to 360 s, measured in 5 s increments. Open circles represent 5%<FT strategy, closed circles represent FT strategy. Anaerobic contribution was significantly higher during first 15 s for 5%<FT but was significantly higher significantly in the FT strategy from 270-360 s ( $p < 0.05$ ).

### 2.3 Discussion

The purpose of the investigation was to compare two different pacing strategies on a six-minute cycle performance. Comparisons of  $\text{VO}_2$ , power output, muscle deoxygenation, and energy system contribution were made. The 5%<FT mimicked a parabolic shaped pacing strategy that is adopted by elite rowers (30). The FT utilized an even paced protocol at one's calculated fatigue threshold power output. The major findings of the present study are: 1) The mean work rate throughout the entire pacing strategy (0-360 s) was significantly higher in FT compared to 5%<FT and 2)  $\text{VO}_2$  was higher during the first 180 s for 5%<FT, whereas  $\text{VO}_2$  was higher in FT during the last 90 s of the 6 minute test. 3

#### *Performance*

The mean work rate for the entire duration of the pacing strategies was higher in the FT strategy (FT;  $305.1\text{W} \pm 41.1\text{W}$ , compared to the 5%<FT  $281.89\text{W} \pm 42.8\text{W}$  [ $P < .05$ ]). The Concept 2 watts-to-pace calculator was utilized to determine the average 500-meter pace (W) for both strategies. The average pace for the FT strategy was 1:44.7s, compared to 1:47.5s for the 5%<FT strategy. The average time to complete a 2 km rowing ergometer performance with these 500 m splits would be 6:58.8s (418.8 s) for the FT strategy compared to 7:10s (430 s) for the 5%<FT strategy. This means the FT group would finish the race 11.2 s faster than the 5%<FT group. The mean velocity of the FT strategy was 4.77 m/s versus the 5%<FT 4.65m/s and as such, the FT pacing strategy “boat” would finish some 52 m ahead of the 5%<FT strategy boat.

These findings are consistent with Fukuba and Whipp (27) who compared pacing strategies for a 5000m running performance. They split the race duration into two equal

distances. They concluded that running at a pace above one's FT for the first phase would eventually consume all of one's  $W'$ . This would cause the athlete to regress to a power output well below one's FT pace, and despite the early advantage, would be unable to sustain that advantage over the remainder of the race. They also concluded that an initial pace below one's FT pace would also be detrimental to performance, as the athlete would be unable to make up for the lost time. The athlete would run at a velocity that would deplete the  $W'$  at such a high rate, that the precursors of fatigue would be accelerated to a point where they would be unable to sustain that pace for the remaining distance (27). Our results concur with Fukuba and Whipp (27) as it seems that the initial 12s sprint, which introduces variability of velocity away from the predicted FT pace, has hindered exercise performance in the  $5% < FT$  strategy (27).

Bailey et al (7) observed that in a 6 min performance there was no significant difference between a fast start strategy and an even paced strategy although they did see improvements when utilizing a fast start strategy during a 3 min exercise bout (7). The current study differs from the study conducted by Bailey et al., as the fast start procedure in the current study is a 12 s all out sprint, compared to a start that was 10% above one's FT pace then leveled off over the 180 s to a pace that is 10% below one's FT pace. The next 120 s was at the subjects calculated FT pace, followed by a 60 s sprint to the finish. The Fast start examined by Bailey et al. (7) mimics more of a positive pacing strategy (1) as opposed to an initial sprint start strategy in the current study. Thus, the fast start pacing strategy proposed by Bailey et al. may not have resulted in the reduction of  $W'$  of the  $5% < FT$  in this present study. Additionally, the amount of  $W'$  utilized during the last 60s of exercise by the protocol suggest by Bailey et al. should theoretically be significantly

different during the fast start protocol. The current study observed a significantly higher amount of energy consumed in the FT compared to the 5%<FT ( $19352.7 \text{ J} \pm 4438.7 \text{ J}$  compared to  $16552.7 \text{ J} \pm 4172.7 \text{ J}$ ). Whereas, the total joules generated in the study by Bailey et al yielded  $20100 \text{ J} \pm 4900 \text{ J}$  for the fast start strategy and  $20100 \text{ J} \pm 4600 \text{ J}$  for the even start strategy, there was no difference between pacing protocols. However, in the current study there was a significant difference in total energy (J) consumed between pacing protocols. Thus, an increased amount of energy (J) remained for the fast start strategy in the study conducted by Bailey et al.

The decline in energy available in the later stages of the 5%<FT strategy versus the FT may be due to the increased Phosphocreatine (PCr) degradation as a result of the 12s all out sprint. The concentration of the intramuscular stores of PCr in a rested state is approximately 75-85 mmol/kg. The turnover rate of PCr during maximal exercise is approximately 7-9 mmol/kg/sec. Maximal sprinting results in a severe reduction in PCr stores (15, 29). After a 6s all out sprint, PCr stores can drop to 35-55% of their resting values (29). After 30s, PCr may decline to approximately 5-10% of its resting values (24). Previous work (9) from this lab utilizing a similar intensity exercise as the middle portion of the 5%<FT observed an  $\approx 45\%$  drop in PCr after 120 s with a continued reduction in PCr over the following 3 min. This, coupled with the already dramatically reduced PCr elicited by the 10 s sprint, suggests that despite the work rate being set at 5% below FT start there was no recovery of PCr observed over the final minutes of the 5%<FT performance. Therefore, there was little PCr available for use in the final 90 s of the 6 min performance. It has also been reported by Dawson et al. that the greater the PCr degradation, the greater the time that is required for the resynthesis of PCr (15, 40).

Furthermore, the resynthesis of PCr is dependent on the availability of energy oxidative phosphorylation over and above that required for the work performed (34). This decrease in PCr also results in a stoichiometric increase in Pi. It has been suggested that this increase in Pi causes decreased force generation in the exercising musculature (22) which may, in part, explain the inability of the 5%<FT group to accelerate in the final 90 s.

It would seem that the 5%<FT power output was too high to enable any significant recovery to occur from the initial sprint of 5%<FT. Whether a further reduction over the middle portion of the race would elicit a different result requires further study, but appears unlikely.

#### *Anaerobic Energy System Contribution*

From exercise onset to 10 s of work, the 5%<FT yielded a higher anaerobic energy system contribution than FT ( $p < 0.05$ ). For the remainder of the 6 min performance, the opposite was true (see Figure 5). Gaitanos et al. (29) has reported that during a 6 s sprint exercise the contribution of anaerobic glycolytic and PCr to total ATP production was 44% and 50 % respectively (29). This glycolytic phosphorylation contribution was reflected by the 28 mmol muscle lactate, in that 6 s (29). The simultaneous increase in hydrogen ion accumulation, which decreases cellular pH, may inhibit peak force output (18, 24). Peak force output is inhibited as a decreased cellular pH reduces the troponins affinity for  $Ca^{+}$ (23). Over this 12 s sprint in the 5%<FT strategy one would expect a similar or perhaps greater accumulation of these fatigue metabolites, thus inducing a further reduction of force generation in the latter stages of the 6 min 5%<FT performance.



### *Blood Lactate*

There was no significant difference between the pacing strategies in regard to pre and post-exercise blood lactate levels. This is consistent with previous studies examining pacing strategies (3, 26, 36). We speculate that the similarities in post-blood lactate concentration may be attributed to the initial accumulation due to the sprint of the 5%<FT versus the final increased power output and anaerobic contribution over the last 60 s of FT.

### *The Depletion of W'*

Almost one third of the total W' was utilized in the first 30 s of exercise of the 5%<FT, 33% compared to 8% for the FT strategy. The percentage of W' utilized during middle portion (30-300 s) for the 5 %<FT was  $46\% \pm 21\%$ , compared to the FT, which was  $74\% \pm 16\%$ . Thus, during the pacing period, the FT strategy used up  $\frac{3}{4}$  of its anaerobic energy store, compared to the 5 %<FT, which used almost half of its anaerobic energy stores. During the final 60 s of the FT performance, 36% of W' was utilized whereas only 14% was utilized over this same period during the 5%<FT strategy. Burnley and Jones (11) suggest that severe or sprint exercise may expend one's anaerobic capacity so that benefits of faster O<sub>2</sub> kinetics may not be realized as the anaerobic capacity is expended before the end of an exercise bout (11). In the present study, this may have been the case in the 5%<FT strategy.

### *VO<sub>2</sub> Consumption*

It is reasonable to suggest that the initial speeding in VO<sub>2</sub> kinetics for the 5%<FT pacing strategy was a function of the initial 12 s all out sprint, as seen in Table 2. It has been suggested (11, 40, 51) that faster VO<sub>2</sub> kinetics are beneficial to performance as the oxygen deficit is decreased, and thus, the anaerobic contribution from exercise onset is reduced. Furthermore, it has been suggested that faster VO<sub>2</sub> kinetics reduces the rate of depletion of high-energy phosphate (phosphocreatine), while simultaneously reducing the flux through anaerobic glycolysis reducing the accumulation of fatigue metabolites, ADP, Pi and H<sup>+</sup> ions (40). Presumably, this is not the case in the 12 s sprint of the 5%<FT where, as previously stated, the sprint phase would accelerate the accumulation of these fatigue metabolites.

The VO<sub>2</sub> slow component was evident in both pacing protocols. The VO<sub>2</sub> slow component represents a continuous rise in VO<sub>2</sub> following phase II of oxygen kinetics, which is only evident in heavy and severe exercise intensity. The current study utilized power outputs that were in the severe intensity domain. The tolerable duration of exercise in this domain is dependent on the interaction between the anaerobic contribution, the amplitude of the VO<sub>2</sub> slow component and one's VO<sub>2</sub> max (40). While exercising in the severe domain, the VO<sub>2</sub> slow component fails to stabilize and continues to increase until VO<sub>2</sub> max is achieved. The attainment of VO<sub>2</sub> max may signal the termination of exercise. The FT protocol seems to delay the onset of the slow component and extend the time before one's VO<sub>2</sub> max is reached.

The slow component is a precursor to fatigue as the simultaneous decline in pH may impair the contractile function of the type I fibers (49). The decline in pH is due the

type II muscle fibers that are selectively recruited to further assist in force generation. Recruitment of these less efficient type II fibers may have a limiting capacity as they generate ATP from primarily anaerobic pathways which generate high volumes of fatigue metabolites that can further impair the contractile abilities of the muscle (28). Rossiter et al observed that PCr concentrations fall in synchrony to a rising  $\text{VO}_2$  slow component (51). It is suggested that the earlier onset of the slow component observed in 5%<FT may further deplete  $W'$  (11).

It also has been suggested that the increased work rate during the last 90 s of exercise of the FT strategy can attribute to a combination of the higher  $\text{VO}_2$  observed and the increased contribution from  $W'$  compared to the 5%<FT. The FT strategy was able to work at a higher efficiency than 5%<FT over the 90 s incremental sprint. The advantage of a high mechanical efficiency is a decreased energy cost at a given work rate and increased the power output at which  $\text{VO}_2$  max is reached (11). For example, Athlete A and Athlete B may have a  $\text{VO}_2$  of 4.8 L/min. Athlete A may reach  $\text{VO}_2$  max at 320 watts, whereas Athlete B may reach  $\text{VO}_2$  max at 350 watts. Since Athlete B reached  $\text{VO}_2$  max at a higher power output, they have a higher  $\text{VO}_2$  / work rate or metabolic efficiency.

### *HHb*

In the current study, there was greater deoxygenation over the first 30 s of the 5%<FT strategy. This increase in deoxygenation may be attributed to the 12 s sprint, in which caused a much higher ATP demand at the vastus lateralis muscle. Thus there was a greater initial deoxygenation observed. It is suggested that the 12 s sprint was associated with a greater impedance to local blood flow as a result of the increased pressure from

forceful contractions (9, 43). Thus, muscle blood flow, O<sub>2</sub> delivery, and blood flow distribution may have been impaired in the 5%<FT strategy. There was also an increase in HHb, compared to the 5%<FT from 120-220 s (see Figure 3). The concurrent lower power output and greater HHb during this middle period of the 5%<FT versus FT suggests that muscle blood flow distribution was blunted during this middle portion of the 6 min performance due to possible O<sub>2</sub> delivery limitations.

### *HHb/VO<sub>2</sub>*

This ratio provides a sense of how the balance between O<sub>2</sub> delivery and changes in muscle blood flow distribution. The ratio of 1.0 indicates a steady state HHb/VO<sub>2</sub> ratio has been reached. A ratio above 1.0 suggests that an under-perfusion of the active muscle is being compensated for by increased fractional O<sub>2</sub> extraction (46).

The 5%<FT strategy yielded a significantly higher HHb/VO<sub>2</sub> ratio during the first 30s of exercise (P=<0.05). Additionally, the 5%<FT was higher than the FT strategy from 120-230 s of the 6 min performance. The increase observed in the 5%<FT during the sprint and middle portion of the pacing phase suggests a poorer matching of O<sub>2</sub> delivery to O<sub>2</sub> utilization, therefore a higher reliance on O<sub>2</sub> extraction as a result of an reduced O<sub>2</sub> delivery (43).

### *Conclusion*

The purpose of the current study was to compare two different pacing strategies on a cycle ergometer that mimicked a 2 km rowing performance. The traditional strategy (5%<FT) encompasses a fast all out 12 s start, accompanied by pacing at 5% below one's fatigue threshold. The even paced (FT) encompasses pacing at one's fatigue threshold up until the 90 s incremental sprint to the finish.

Despite faster  $\text{VO}_2$  and HHb kinetics, it is suggested that there were several detrimental effects as a result of the 12 s all out sprint, which include: a reduction in overall power output, a decreased utilization of anaerobic energy store (lower anaerobic energy system contribution), and a reduction in  $\text{O}_2$  delivery (blood flow distribution) to the exercising musculature. In conclusion, the FT pacing strategy utilizing a even start protocol may be a more suitable strategy for a 2km (6 min) rowing performance.

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# Appendix A



## Use of Human Participants - Ethics Approval Notice

**Principal Investigator:** Glen Belfry  
**File Number:**101904  
**Review Level:**Full Board  
**Approved Local Adult Participants:**8  
**Approved Local Minor Participants:**0  
**Protocol Title:**The effect of a differing 2k pace strategies on VO<sub>2</sub>, muscle haemoglobin status, energy system contribution and performance in rowing. (REB # 18489)  
**Department & Institution:**Health Sciences\Kinesiology,Western University  
**Sponsor:**  
**Ethics Approval Date:**May 22, 2012  
**Ethics Expiry Date:**December 31, 2012

### Documents Reviewed & Approved & Documents Received for Information:

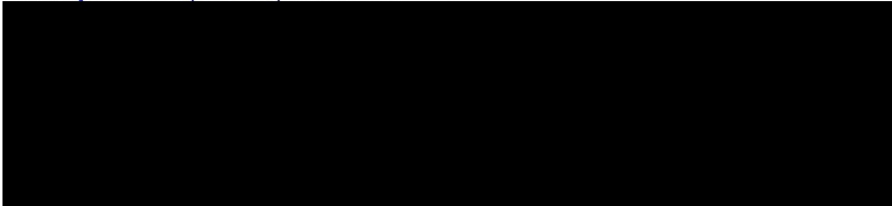
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Western University Protocol		
Letter of Information & Consent		
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This is to notify you that the University of Western Ontario Health Sciences Research Ethics Board (HSREB) which is organized and operates according to the Tri-Council Policy Statement: Ethical Conduct of Research Involving Humans and the Health Canada/ICH Good Clinical Practice Practices: Consolidated Guidelines; and the applicable laws and regulations of Ontario has reviewed and granted approval to the above referenced study on the approval date noted above. The membership of this HSREB also complies with the membership requirements for REB's as defined in Division 5 of the Food and Drug Regulations.

The ethics approval for this study shall remain valid until the expiry date noted above assuming timely and acceptable responses to the HSREB's periodic requests for surveillance and monitoring information. If you require an updated approval notice prior to that time you must request it using the University of Western Ontario Updated Approval Request form.

Member of the HSREB that are named as investigators in research studies, or declare a conflict of interest, do not participate in discussions related to, nor vote on, such studies when they are presented to the HSREB.

The Chair of the HSREB is Dr. Joseph Gilbert. The HSREB is registered with the U.S. Department of Health & Human Services under the IRB registration number IRB 00000940.



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*The University of Western Ontario*



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