CHANGES IN THE CHEMOREFLEX RESPONSE AND PERFORMANCE MEASURES WITH TRAINING IN COMPETITIVE SWIMMERS

Gregory D. Wells

A thesis submitted in conformity with the requirements for the
Degree of Master of Science
Graduate Program in Exercise Science,
University of Toronto

© Copyright by Gregory D. Wells, 1999
The author has granted a non-exclusive licence allowing the National Library of Canada to reproduce, loan, distribute or sell copies of this thesis in microform, paper or electronic formats.

The author retains ownership of the copyright in this thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without the author’s permission.

L’auteur a accordé une licence non exclusive permettant à la Bibliothèque nationale du Canada de reproduire, prêter, distribuer ou vendre des copies de cette thèse sous la forme de microfiche/film, de reproduction sur papier ou sur format électronique.

L’auteur conserve la propriété du droit d’auteur qui protège cette thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou autrement reproduits sans son autorisation.

0-612-45923-3
Changes in the Chemoreflex Response and Performance Measures with Training in Competitive Swimmers
1999
Gregory D. Wells
Graduate Department of Exercise Science,
University of Toronto

Abstract

Breathing during swimming performance is entrained to the stroke rate, suggesting that there may be an interaction between training, performance, and the control of breathing. The purpose of this study, therefore, was to evaluate the effect of training on the chemoreflex response to carbon dioxide, and to evaluate these changes in light of alterations in swimming performance measures.

Subjects were 21 competitive swimmers (mean age 17 ± 2 years) and 14 non-athletes (mean age 18 ± 2.5 years). The hypoxic rebreathing test results showed that, after training, the chemoreflex threshold increased significantly (p=0.01) and chemoreflex sensitivity decreased (p=0.05). There was a non-significant decrease in the frequency sensitivity (p=0.06). There were no significant changes in the rebreathing test measures for the athletes in hyperoxia. The swimming tests indicated that measures of both critical velocity (p<=0.001) and peak swimming velocity (p<=0.001) were increased after training. Improvement in swimming economy variables were detected, although the results were not significant. Distance per breath on the peak swimming velocity test was significantly improved (p=0.01). There were no significant changes in the non-athlete group over the training period.

The results of this study suggest that swim training acts to attenuate the hypoxic chemoreflex response, an adaptive response that may allow the swimmers to swim at faster velocities through improvements in technical efficiency.
Acknowledgements

I would like to thank my supervisors Dr. Michael Plyley and Dr. James Duffin for their support and guidance in making this a truly incredible learning and growth experience. Also, thanks to Dr. Duffin for the use of the respiratory laboratory and for his personal assistance in nearly every aspect of the research.

Thank you to my mentor, Mark Temple, for his ideas and encouragement.

I would never have been able to complete this work without the assistance of my editor Barbara Bauer.

I would like to express my gratitude to coaches John Grootveld, Grey Fairley, Gaye Stratten, and Murray Drudge, the swimmers from NYAC, HWAC, TSC, and MMSAC, and to the non-athlete students (especially Ashley Bennion) for taking the time and energy to participate in the research and for their commitment to the project.

Thanks to my research assistant, Safraaz Mahamed for his ability to trouble shoot the system and for ensuring that the data collection went smoothly.

Finally, thank you to my family, I cannot say enough about how your love and support have helped to get me through this.
## Table of Contents

Abstract .................................................. i
Acknowledgements ........................................ ii
List of Tables ........................................... vii
List of Figures ........................................... vii
List of Appendices ........................................ viii

### CHAPTER 1: PROBLEM, REVIEW OF THE LITERATURE, AND PURPOSES

Background and Review of Literature .............. 1
   The Control of Breathing .............................. 1
   The Role of the Chemoreflexes in the Control of Breathing 2
   The Control of Breathing in Exercise ............... 4
   Contributions of the Chemoreceptors to the Control of Breathing During Exercise 6
   The Case for an Attenuated Chemoreceptor Response in Trained vs. Untrained Subjects 8
   Summary of Background and Review of the Literature 10
   Purposes of the Study ................................ 11

### CHAPTER 2: METHODS

Research Design and Research Questions .......... 13
Hypotheses ................................................. 13
Definition of Variables and Outcome Measures .... 14
Sample and Sampling Procedures ..................... 15
Experimental Protocols ................................. 17
Ethical Considerations .................................. 21
Statistics and Data Analysis .......................... 23
REFERENCES

APPENDICES

A - Human Ethics Submission and Approval
B - Raw Data Swimming Tests
C - Raw Data Rebreathing Tests
List of Tables

Table 1  Descriptive Characteristics of Athlete Group  25
Table 2  Descriptive Characteristics of Non-Athlete Group  26
Table 3  Swimming Test Results and Statistical Analysis  28
Table 4  Rebreathing Test Results and Statistical Analysis  39
Table 5  Correlation Analysis Chart  45

List of Figures

Figure 1  Rebreathing test apparatus set-up  18
Figure 2  Training volume over time  27
Figure 3  An example of critical velocity analysis for one subject  29
Figure 4  Changes in mean critical velocity over time  30
Figure 5  Changes in peak velocity on maximal performance test over time  31
Figure 6  Example of distance per stroke decay during progressive swimming test for one subject  33
Figure 7  Changes in mean distance per stroke on maximal performance test over time  34
Figure 8  Changes in breathing frequency over time  35
Figure 9  Distance per breath on maximal performance test over time  36
Figure 10  Graphical analysis model for rebreathing test results  38
Figure 11  Changes in chemoreflex threshold (hypoxia) for the athlete and non-athlete groups over time  41
Figure 12  Changes in chemoreflex sensitivity (hypoxia) for the athlete and non-athlete groups over time  43
Figure 13  Changes in frequency sensitivity (hypoxia) for the athlete and non-athlete groups over time  44
Figure 14  Summary of swimming and rebreathing discussion  57
List of Appendices

<table>
<thead>
<tr>
<th>Appendix</th>
<th>Title</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appendix A</td>
<td>Human Ethics Submission and Approval</td>
</tr>
<tr>
<td>Appendix B</td>
<td>Raw Data – Rebreathing Test Results</td>
</tr>
<tr>
<td>Appendix C</td>
<td>Raw Data – Swim Test Results</td>
</tr>
</tbody>
</table>
CHAPTER 1: PROBLEM, REVIEW OF THE LITERATURE AND PURPOSES

One of the requirements for training and performance in sports such as running, cycling, speed skating, and rowing is developing the ability to control breathing under stressful circumstances. A close relationship exists between breathing rate and exercise cycle rate, that is, breathing is often entrained to the limb movement rhythm. In addition, each athlete has an optimal cycle rate for peak muscular efficiency.

These relationships also hold true for the sport of swimming. However, the complicating factor in swimming is that the athlete is not able to breathe on demand, but must wait for that portion of the stroke cycle that allows for breathing to occur. Thus, the question arises as to the nature and extent of the interaction between the breathing rate and the cycling rate during swimming. More specifically, (a) does the drive to breathe influence the arm cycling rate and, therefore, potentially alter the technical aspects of the skill (i.e., cycle rate and stroke length); and (b) since changes in cycling rate and stroke length are known to occur during training, do concomitant changes to the drive to breathe occur during the training process?

Background and Review of Literature

The Control of Breathing

Over the last 100 years, various authors have proposed numerous mechanisms to explain the hyperpnea (increased breathing) observed with exercise (Mateika & Duffin, 1995). Currently, most researchers have acknowledged that no one mechanism totally explains the observed response, with numerous investigators focusing on analyzing the different factors that may be involved, and the contribution that each makes to the control of breathing under exercise conditions. Researchers generally subscribe to one of two
fundamentally different theories that partially explain the control of breathing, each theory based on a different view of ventilatory drive.

Dejours first presented the neuro-humoral theory of ventilatory control in 1963. Reviews by Cunningham (1974, 1987) and Mateika and Duffin (1995) have synthesized more recent and current developments. The first theory suggests that ventilation is driven by feed-forward predictive neural drives from central command. The second theory posits that ventilation is controlled by feedback regulators, such as the peripheral and central chemoreflexes, that respond to changes in the cellular chemical environments (i.e., partial pressure of carbon dioxide, the hydrogen ion concentration, or the potassium ion concentration) in an effort to maintain cellular homeostasis. Simply put, the feed-forward neural drives from central command and the exercising limbs can be described as the brain noticing and/or anticipating a need to ventilate and then sending a signal that increases breathing. The feedback regulators are sensitive to levels of various chemicals in the body and/or to the neural feedback from the exercising limbs. When the levels of these signals change, the regulators send messages that increase or decrease breathing as necessary. The contribution that each of these drives makes to the overall control of breathing at rest and during exercise remains a topic of debate.

The Role of the Chemoreflexes in the Control of Breathing

The focus in the following section will be on exploring how the various feedback regulators control breathing through the peripheral and central chemoreflexes.

Peripheral chemoreceptors. The peripheral chemoreceptors are located in the carotid bodies at the bifurcation of the carotid artery, and are sensitive to the levels of various substances in the perfusing blood (Nye, 1994). Specifically, hypoxia acts to increase the sensitivity of the peripheral chemoreceptors to hypercapnia, hydrogen ion concentration, and potassium ion concentration (Mateika & Duffin, 1995). The relationship between ventilation and the arterial partial pressure of oxygen has been described as hyperbolic. The resulting curve is relatively flat at arterial partial pressure.
of oxygen levels exceeding 100 mm Hg and becomes progressively steeper at arterial partial pressure of oxygen levels lower than 40 mm Hg (Cunningham, 1974). Peripheral chemosensitivity has been indirectly examined by the measurement of this relationship, although the exact peripheral threshold and sensitivity have not been determined. Other potential stimuli include (a) changes in the hydrogen ion concentration induced by altered partial pressure of carbon dioxide, as explained by the Henderson-Hasselbalch equation \([\text{pH}] = 24\text{PCO}_2 \, \text{mmHg} \cdot [\text{HCO}_3^-] \, \text{mmol/L}\); (b) blood acid-base status (Nye, 1994); and (c) changes in the potassium ion concentration, perhaps caused by spillover from exercising muscles (Paterson et al., 1992). Duffin (1990) stated that it is preferable to view the peripheral chemoreceptors as primarily hydrogen ion sensors whose sensitivity is controlled by the level of hypoxia. Mohan & Duffin (1997) found that at a constant partial pressure of oxygen, the ventilatory response of the peripheral chemoreceptors to hypercapnia is linear with increasing partial pressure of carbon dioxide above a threshold level (approximately 40 mmHg carbon dioxide). Their study represents a different method of assessing the peripheral chemoreflex response from that which has been traditionally used (the ventilation – hypoxia hyperbola). The slope of the linear response described by Mohan and Duffin was taken to be the sensitivity of the peripheral chemoreceptors, which was shown to increase significantly from 100 to 80, 60, and 40 mm Hg partial pressure of oxygen. The advantage of their method is that it allows for direct measurement of the chemoreflex threshold and sensitivity to carbon dioxide at varying levels of hypoxia. A review of the research has indicated that the between-subject variability of the peripheral chemoreflex sensitivity to carbon dioxide has not been extensively studied.

Central chemoreceptors. The central chemoreceptors are distributed within the brain stem and are concentrated mainly on the ventro-lateral surface of the medulla (Coates et al., 1993). They are stimulated by the hydrogen ion concentration of the surrounding environment (Bruce, 1987). Bledsoe and Hornbein (1981) hypothesized that the hydrogen ion concentration of the arterial blood and the cerebrospinal fluid surrounding the central chemoreceptors does not easily equilibrate across the blood-brain barrier, but that the partial pressure of carbon dioxide does equilibrate easily. In this
scenario, the hydrogen ion concentration sensed by the central chemoreceptors is influenced, over the short term, by changes in the arterial partial pressure of carbon dioxide rather than by changes in the arterial hydrogen ion concentration. Further, relatively small changes in arterial partial pressure of carbon dioxide (due to diffusion of carbon dioxide across the blood-brain barrier) will elicit large changes in the hydrogen ion concentration of the cerebrospinal fluid (according to the linear form of the Henderson-Hasselbalch equation).

It is possible to examine the central chemoreflex response to increased carbon dioxide by rebreathing a hyperoxic gas mixture (Read, 1967). Isolation of the central chemoreceptor response is achieved by way of the inspiration of a hyperoxic gas mixture, which nearly abolishes the ventilatory response of the peripheral chemoreceptors to partial pressure of carbon dioxide, hydrogen ion concentration, and potassium ion concentration (Cunningham et al., 1986). The central chemoreflex response is described by plotting ventilation vs. the inspired partial pressure of carbon dioxide, with the slope of the response representative of the central chemoreceptor sensitivity to carbon dioxide (Read, 1967). This relationship between ventilation and inspired partial pressure of carbon dioxide has been reported to be linear whether measured by the rebreathing or the steady state techniques. However, various authors disagree about whether the techniques produce different (Linton, 1973; Read, 1967), or similar (Berkenbosch, 1989), results. The sensitivity of the central chemoreceptors has also been found to vary between subjects (McGurk, 1995; Mohan & Duffin, 1997).

The Control of Breathing in Exercise

The ventilatory sensitivity to carbon dioxide is governed by the peripheral and central chemoreflex responses. A number of studies have reported a correlation between the magnitude of the ventilatory response to exercise (ΔVe/ΔVCO₂), and the ventilatory sensitivity to carbon dioxide (ΔVe/ΔPCO₂). Specifically, Martin et al. (1978) found that the hypoxic (low oxygen) and hypercapnic (high carbon dioxide) ventilatory responses were correlated during both light (1/3 VO₂max) and heavy (2/3 VO₂max) exercise
intensities in athletes. Martin et al. used the steady state method of determining the ventilatory response to carbon dioxide. Using the same method, McConnell et al. (1992) noted a stronger correlation between the ventilatory response to exercise and carbon dioxide sensitivity during exercise than was observed at rest. These descriptive reports suggest the need for a systematic investigation of the role of ventilatory sensitivity to carbon dioxide in determining the magnitude of exercise hyperpnea in athletes vs. non-athletes while at rest vs. during exercise, and sequentially during training.

Since the changes in ventilation that occur with exercise are correlated with the ventilatory sensitivity to carbon dioxide, it follows that ventilatory sensitivity to carbon dioxide may play a role in determining not only the magnitude of exercise hyperpnea, but also attenuation of the response. Thus, if training exerts an influence in attenuating exercise hyperpnea, it may do so through its impact upon changing an individual’s ventilatory sensitivity to carbon dioxide.

In some athletic events, such as swimming, cycling, running and rowing, it may be desirable to control the hyperpnea of exercise. Craig and Pendergast (1979), who described the relationship between swimming velocity, cycle rate, and cycle length, reported that the fastest swimmers had the longest cycle length at sub-maximal velocities, and that there was a positive correlation between maximal cycle length and maximal velocity. Most competitive swimmers have a breathing pattern where they breathe every (x) number of cycles. Therefore, cycle length and breathing pattern are closely related. It follows that an alteration in an athlete’s drive to breathe when sustaining increased velocities may influence cycle rate and thus swimming velocity (McConnell & Semple, 1996). One potential physiological explanation for improvements in cycle length, in swimming, concerns an altered chemoreflex response (McGurk et al., 1995)

During intense exercise, increased ventilation is accompanied by lactic acid accumulation, metabolic acidosis, increased metabolic production of carbon dioxide, and increased potassium ion concentrations (Mateika & Duffin, 1995). These factors partially account (~15-20%) for the increased ventilation through their actions upon the peripheral
chemoreflexes (Cunningham, 1987). This increased drive to breathe and ventilate can lead to a decrease in an athlete's ability to maintain optimal cycle length due to the resulting decrease in time between breaths. Thus each athlete, at any training state, would be expected to have an optimal combination of cycle length and cycle rate, which may involve considerations of local muscle power, muscular endurance, and an attenuated chemoreflex response, the latter of which would lead to a decreased exercise hyperpnea. A reduced drive to ventilate would be of benefit to those athletes who need to control breathing patterns and maintain an optimal cycle length. Wakayoshi et al. (1995) presented supporting evidence for the potential relationship of velocity, cycle length, and cycle rate; they found that high performance swimmers had a greater cycle length and a lower cycle rate than lower performance swimmers at a given velocity. Some authors hypothesize that in endurance events, it may be beneficial to conserve energy by restraining hyperpnea and allowing the partial pressure of carbon dioxide to rise, and that such a strategy would be unnecessary in sprint events (McGurk et al., 1995). Given these conditions, it may be expected that the sensitivity to carbon dioxide would be lower in successful endurance athletes than in sprinters or untrained subjects (McConnell & Semple, 1996). These authors do not discuss the possible physiological challenges caused by decreased ventilation during exercise which include decreased oxygen delivery (Town & Vanness, 1990), and increased lactic acidosis in the muscle during exercise (Yamamoto et al., 1988). Thus the interactions between the control of breathing in exercise and performance remain unclear.

Contributions of the Chemoreceptors to the Control of Breathing During Exercise

The current theories on the role that the central and peripheral chemoreceptors may play in the control of breathing during exercise will be reviewed in the following sections.

Peripheral chemoreceptors. At rest, the drive to breathe from the peripheral chemoreceptors accounts for approximately 20% of total ventilation (Stockley, 1977). The increased ventilation observed from the onset of exercise to a level at which the
venous partial pressure of carbon dioxide and partial pressure of oxygen begin to change is said to be mediated by neural mechanisms (Duffin, 1991; Ward, 1994). In light and moderate exercise below the anaerobic threshold (the onset of lactic acid accumulation), the partial pressure of carbon dioxide, blood lactate concentration, hydrogen ion concentration, and potassium ion concentration remain near resting values; thus, the peripheral chemoreceptors would not be expected to contribute greatly to increased ventilation at these work loads. However, the peripheral chemoreflexes may exert considerable influence (up to 20% of total ventilation) in the ventilatory response to moderate exercise at or slightly above the anaerobic threshold (Griffiths et al., 1986). In high intensity exercise above the anaerobic threshold, the peripheral chemoreceptors play a role in mediating ventilation to compensate for metabolic changes associated with high intensity exercise (Rausch et al., 1991). These changes may include the metabolic acidosis that occurs as a result of lactic acid accumulation (Wasserman et al., 1990) and the increasing potassium ion release from the exercising muscles (Paterson, 1992). The ventilatory response occurs even though the partial pressure of carbon dioxide may decrease as a result of the increased ventilation. However, direct estimates by Jeyranjan et al. (1988) showed that the only about 20% of ventilation in heavy exercise is mediated by the peripheral chemoreflex. Thus, the peripheral chemoreflex may account for up to 20% of the increased ventilation in moderate and heavy exercise.

Central chemoreceptors. The evidence to date suggests that the central chemoreceptors do not contribute to the exercise hyperpnea observed in light to moderate exercise below the anaerobic threshold. The central chemoreceptors would not be expected to be involved because of the slow and slight increase in the arterial partial pressure of carbon dioxide expected at this exercise intensity (Whipp & Ward, 1991), and because of the need for the carbon dioxide to diffuse across the blood-brain barrier before it can stimulate the central chemoreceptors (Belleville et al., 1979).

Decreases in pH and bicarbonate concentration, and increases in the volume of expired carbon dioxide, blood lactate concentration, and potassium ion concentration (Mateika & Duffin, 1995; Ward, 1994) characterize high intensity exercise. It has been
suggested that the role of the central chemoreceptors in the regulation of breathing in short-term, high intensity exercise may be to constrain ventilation, possibly as a result of the respiratory alkalosis of the cerebral spinal fluid (Bisgard et al., 1978). However, these hypotheses, which have been derived from studies on exercising ponies, have limitations, specifically that the environment of the cerebral spinal fluid may not be the same as that of the brain interstitial fluid. The ionic state of the brain interstitial fluid may be quickly altered by the diffusion of carbon dioxide across the blood-brain barrier, which would, in turn, render the central chemoreceptors more responsive to short-term changes in hydrogen ion concentration (Dempsey & Smith, 1994). In chronic, steady state exercise, the slow diffusion of carbon dioxide across the blood-brain barrier into the cerebrospinal fluid may contribute to a central chemoreceptor drive to ventilation in non-acute conditions (Eldridge et al., 1985). However, the contribution of the central chemoreceptors to exercise hyperpnea remains unclear.

The Case for an Attenuated Chemoreceptor Response in Trained vs. Untrained Subjects

Miyamura et al. (1976) presented supporting evidence for an attenuated ventilatory response in athletes. These investigators examined the differences between untrained subjects and marathon runners with regard to the ventilatory response to carbon dioxide rebreathing both at rest and during steady state, sub-maximal exercise. They used the rebreathing technique described by Read (1967) to measure the ventilatory response to carbon dioxide. The mean slope (the rate of increased ventilation in response to increased carbon dioxide) of the ventilation vs. partial pressure of carbon dioxide curve at rest was 1.86 (L·min⁻¹·mmHg⁻¹) for the controls vs. 1.12 (L·min⁻¹·mmHg⁻¹) for the athletes. During exercise, the slopes were 1.20 (L·min⁻¹·mmHg⁻¹) for the controls vs. 0.62 (L·min⁻¹·mmHg⁻¹) for the athletes. The differences between the two groups were significant under both conditions. Thus, the average slopes of the response curves were lower for the athletes than for the untrained group, both at rest and during exercise (athletes ventilated less than non-athletes at given levels of carbon dioxide). While this study demonstrated differences in response between the two groups, it unfortunately measured the central and not the peripheral chemoreceptors as the rebreathing was
conducted under hyperoxic conditions only. In addition, the study also leaves unanswered the question of whether neural factors had any influence on the differences observed between the two groups. Finally, a note of caution must be introduced to the exercise findings of this study because the rebreathing technique does not work effectively under exercise conditions due to the necessity to maintain equilibration of carbon dioxide in all compartments of the body during the rebreathing.

McGurk et al. (1995) recently attempted to establish a relationship between the ventilatory sensitivity to carbon dioxide and measures of sprint vs. endurance performance in young swimmers. The subjects were categorized into two groups: high responders (n=17) and low responders (n=17), based on the hyperoxic (high oxygen) carbon dioxide rebreathing test at rest. In this rebreathing test, a version of Read's method modified by Rebuck (1972), 7% carbon dioxide and 93% oxygen was used. McGurk et al. compared the responses to carbon dioxide rebreathing with the results of two sprint and two endurance treadmill performance tests. The results indicated that the low responders had a significantly faster 1.6-km run time than the high responders, and that the high responders recorded significantly better results on the 10 second alactic power test. Unfortunately, the rebreathing test was conducted under hyperoxic conditions, only the response of the central chemoreflex drive could be assessed as high oxygen conditions abolish the peripheral chemoreflex response. Also, the performance measures used to evaluate the swimmers were not sport specific and, therefore, may not have accurately reflected the actual abilities of the swimmers.

Godfrey et al. (1971) used Read's rebreathing technique (1967) to compare the ventilatory responses to hypercapnia (high carbon dioxide) under hyperoxic conditions (initial gas mixture in bag: 50 mmHg carbon dioxide, 150 mmHg oxygen) at rest in seven athletes and seven control (non-athlete) subjects. They found a slightly higher slope in the carbon dioxide rebreathing response curve [2.36 vs. 2.05 (L·min⁻¹·mmHg⁻¹)] in the athletes than in control subjects, respectively. This implies an increased ventilatory response to carbon dioxide in the athletes than in the controls, a contradiction of Miyamura’s results. Again, because the rebreathing was done under hyperoxic
conditions, only the central chemoreflex ventilatory response to carbon dioxide was assessed.

Mahler et al. (1982) examined the ventilatory responses to hypercapnia and its possible role in athletic performance by comparing 20 male marathon runners to 20 sedentary subjects of the same age. They used a modified version of Read's rebreathing technique (1967) with a gas mixture of 7% carbon dioxide in 93% oxygen at rest. Their results indicated a reduced ventilatory drive in response to hypercapnia in the runners and sedentary subjects of 2.23 vs. 2.61 (L·min⁻¹·mmHg⁻¹), respectively. Although the authors reported a poor correlation between chemosensitivity and endurance performance, the rebreathing technique employed in this study was useful only in assessing the chemosensitivity of the central chemoreceptors, not the peripheral chemoreceptors.

Heigenhauser et al. (1983) examined the ventilatory response to carbon dioxide during rebreathing at rest in 8 synchronized swimmers, 8 competitive swimmers, and 8 recreational swimmers. The synchronized swimmers participated regularly in training that involved prolonged periods of breath holding, and the competitive swimmers trained at high exercise intensities to increase their aerobic and glycolytic capacities. Both groups had been training for at least 3 years. The rebreathing technique was performed according to Read's method (1967). During rebreathing, the slope of the ventilatory response curve to carbon dioxide was lower for the synchronized swimmers [1.48 (L·min⁻¹·mmHg⁻¹)] than for either the competitive swimmers [2.04 (L·min⁻¹·mmHg⁻¹)] or the recreational swimmers [1.87 (L·min⁻¹·mmHg⁻¹)]. The researchers also reported that ventilation was significantly higher in the recreational swimmers than in both the synchronized and competitive swimmers during arm cranking exercise at a given level of carbon dioxide production.

Blum et al. (1979) used the hyperoxic (Read's) rebreathing technique to examine the central chemoreflex response to carbon dioxide before and after training. They found
a decrease in the ventilatory response to carbon dioxide after training in five normal subjects. Because the length of training and initial fitness levels were not stated, the implications and interpretation of these results are unclear with regard to training adaptations.

**Summary of Background and Review of the Literature**

Research investigators have demonstrated that ventilation at a given work load is attenuated after training. Convincing evidence has also been presented that the peripheral and central chemoreceptors act as feedback regulators in the control of breathing. Moreover, research findings also suggested that the peripheral chemoreceptors would be expected to contribute to exercise hyperpnea, whereas the central chemoreceptors would not be involved. Unfortunately, studies to date have examined only the role of the central chemoreceptors yet the central chemoreceptors are not expected to be affected by training. Further, research on the differences in central chemoreflex response between athletes and controls is difficult to compare given the different results obtained on control groups between papers that are supposedly using the same technique. No research has been done concerning the effect of training on the peripheral chemoreflex response to carbon dioxide.

Therefore, the questions concerning differences between athletes and non-athletes, and regarding the trainability of the peripheral and central chemoreflex responses need to be resolved. Further investigation is also needed to differentiate between the relative contributions of the peripheral and central chemoreflexes to the control of breathing during exercise and at rest, and to improve our understanding of the interaction between the control of breathing and optimal performance.

**Purpose of the Study**

Recently, research on the control of breathing has focused on the role played by the central and peripheral chemoreceptors. Few studies have evaluated the alterations in
the control of breathing during a period of physical training. The purposes of this study, therefore, were threefold. The first was to assess the difference between competitive swimmers and non-athletes in terms of their chemoreflex response to carbon dioxide. The second was to evaluate the effect of swim training on the chemoreflex response to carbon dioxide. Third to determine if there is a relationship between the changes in chemoreflex response with alterations in stroke mechanics, critical velocity, performance and anaerobic power.
CHAPTER 2: METHOD

Research Design and Research Questions

A comparative descriptive, correlational design with longitudinal and cross sectional elements (Burns & Grove, 1997) was employed in this study to answer the following research questions.

1. Does training have an effect on measures of performance (critical velocity (Treffene, 1985), peak velocity, distance per stroke, and breathing economy) in swimmers?
2. Does training have an effect on the chemoreflex response to carbon dioxide in athletes?
3. Is there a correlation between the training induced changes in the chemoreflex response to carbon dioxide and the various measures of performance (critical velocity, peak velocity, distance per stroke, and breathing measures) in swimmers?
4. Is there a difference between athletes and non-athletes in terms of their chemoreflex responses to carbon dioxide?

Hypotheses

The hypotheses of this study were:

1. Swim training will result in improved critical velocity, increased peak velocity, increased distance per stroke, and increased distance per breath in the athlete group.
2. Training will result in an increase in chemoreflex thresholds and a decrease in chemoreflex sensitivity to carbon dioxide in hypoxia. Training will not have an effect upon the chemoreflex response to carbon dioxide in hyperoxia.
3. There will be a correlation between changes in the chemoreflex sensitivity to carbon dioxide and changes in measures of swimming performance, specifically distance per stroke and distance per breath.
4. Athletes will have an attenuated chemoreflex response to carbon dioxide (increased chemoreflex thresholds, decreased chemoreflex sensitivity). There will not be a difference between the chemoreflex response to carbon dioxide between athletes and non-athletes under hyperoxic conditions.

**Definition of Variables and Outcome Measures**

Several variables and their observed characteristics defined the chemoreflex response to alterations in inspired carbon dioxide level. These variables were:

- Hyperoxic chemoreflex threshold and sensitivity (central chemoreflex).
- Hypoxic chemoreflex threshold and sensitivity (peripheral and central chemoreflexes).

Tidal volume and frequency characteristics were also observed and measured. These variables were measured via a rebreathing test (Mohan & Duffin, 1997), and were determined through plotting and graphical analysis of the ventilation vs. partial pressure of carbon dioxide curve.

The following performance measures were used to assess the physiological and technical characteristics of swimmers, including:

- Critical velocity (a measure of endurance performance)
- Peak velocity (a maximal performance test)
- Distance per stroke (a measure of technical efficiency)
- Distance per breath and breathing frequency (a measure of breathing efficiency)

Critical velocity as defined by Treffene (1982), was determined from a plot of swimming velocity vs. heart rate. Distance per stroke was determined from a plot of velocity vs. distance per stroke (Craig and Pendergast, 1980). Distance per breath was determined from a plot of velocity vs. distance per breath, and breathing frequency from a plot of velocity vs. breathing frequency.
Sample and Sampling Procedures

This study was conducted using swimmers and non-athlete participants from a large metropolitan area. In total, the researcher recruited 36 participants (22 athletes and 14 non-athletes) for in the study. Athletes included in the study had to (a) be healthy, male or female volunteers, (b) be post-pubescent swimmers between the ages of 13 and 22, (c) be members of competitive swimming teams, and (d) have achieved a swimming performance time within 5% of national qualification standards. The 14 non-athletes who were recruited for the study’s comparison group had to (a) be healthy, male or female volunteers, and (b) post-pubescent non-athletes between the ages of 13 and 22.

Potential athlete participants were recruited from 10 national level competitive swimming teams through advertisement. A notice of the study was sent to the head swim coaches of the top 10 teams in the Greater Toronto Area that are most highly ranked in Canada to inform them of the research and to solicit their assistance in recruiting potential participants, that is, National Championship calibre athletes ranging from 13 to 22 years of age. The large participant pool (200 potential athletes of National calibre on these 10 teams) allowed a diverse range of participant ages. It was necessary to use participants of this age group because the majority of high performance swimmers in the Greater Toronto Area are under the age of 18. It should be noted that it is unclear whether the potential adaptation under investigation is progressive, and what the time course of the adaptive response may be. In this case, younger swimmers with less training history would be advantageous for determining the possible differences in trainability, training status, or time response for this variable. Research has indicated that post-pubescent males and females perform similarly in the rebreathing test as long as the females are tested in the mid-follicular phase of their menstrual cycles and the participants are post-pubescent (McGurk et al., 1995). Female participants were asked to schedule their rebreathing tests as close to one-week after the end of their menstrual periods as possible.

The head coaches were asked only to announce the research to all swimmers at a team meeting and to parents at a parents’ meeting, using a standardized letter of
introduction to the study (see Appendix A, page 91). They also were asked to post a copy of the letter of introduction at the pools in a site visible to both swimmers and their parents. In order to prevent a conflict of interest from arising between these swim coaches and the competitive swimmers, the investigator alone presented the details of the study to the swimmers and parents or guardians. For those swimmers and parents who demonstrated interest in the study, the head coach requested permission to forward their names and phone numbers to the investigator, who then contacted potential participants to arrange a meeting at a time and place convenient to them in order to explain the study in further detail (see Appendix A, page 92). Written informed consent concerning their participation (see Appendix A, page 95) was obtained from the swimmers and parents who agreed to participate.

The comparison group of non-athletes had not been involved in any type of regular training program over the previous 12 months. Not being involved in a regular training program was defined as not exercising more than three times per week for no more than 40 minutes at a heart rate in the training zone; this exercise regimen has been defined as being the minimum exercise program needed to develop one’s level of fitness. The American College of Sports Medicine has developed this definition (Bryant et al., 1995).

The non-athlete participants were recruited through advertisements posted at community centres in the Greater Toronto Area (see Appendix A, page 99). The inclusion criteria were stated in the advertisement. The investigator then contacted all potential participant volunteers, who demonstrated interest in the study by leaving a message at a phone number provided in the advertisement. A meeting was then scheduled at a time and place convenient to the participants in order to explain the study in further detail (see Appendix A, page 100). If the potential participants and parents agreed to participate in the study, written informed consent was obtained concerning their participation (see Appendix A, page 103). An agreed upon time to conduct the study was established with the participants.
**Experimental Protocols**

For both groups, descriptive data were collected, including gender, date of birth, history of competitive swimming for the athletes and history of exercise for the non-athletes, and baseline fitness level (PWC$_{170}$ test). Data were also collected from both groups during the rebreathing test. The swimming tests were conducted with only the athletes.

**Rebreathing protocol.** During the laboratory data collection, each participant was asked to complete two rebreathing tests at constant end-tidal partial pressures of oxygen at 50 mmHg and 150 mmHg, respectively. A hypoxic level of 50 mmHg was chosen to allow for the assessment of both the peripheral and the central chemoreflex responses. The single test at 150 mmHg oxygen allowed for the isolation of the central chemoreflex. Measuring the response of both chemoreflexes variables allowed the researchers to investigate the relative contributions of each chemoreflex to the total chemoreflex response. Each rebreathing test session lasted approximately 120 minutes. No more than three 10-minute tests were conducted in a given session, with each assessment being separated by a rest period of at least 30 minutes. The participants were asked to refrain from drinking any caffeinated beverages for 2 hours prior to testing.

The rebreathing test used in this experiment was first proposed by Read (1967) and subsequently modified by Duffin and McAvoy (1988). The test included a prior voluntary hyperventilation to lower carbon dioxide to sub-threshold levels, along with computer control of the supply of oxygen to ensure iso-oxic conditions during the rebreathing. These modifications allowed for the determination of the thresholds and sensitivities of both the central and peripheral components of the chemoreflex response to carbon dioxide at specific levels of hypoxia.

The apparatus used for the rebreathing tests has been used previously in this laboratory (Duffin & McAvoy, 1988; Mohan & Duffin, 1997), with the addition of a computer-controlled feedback system to maintain iso-oxic conditions at 50 mmHg or 150
mmHg of oxygen throughout the experiment (see Figure 1). The entire apparatus was calibrated before each test session.

![Rebreathing Apparatus](image)

**Figure 1.** Rebreathing Test Apparatus Set-up

An oximeter probe (Bruel and Kjaer, Model 8852) was placed on the participant's index finger in order to monitor heart rate and oxygen saturation. The participant wore a nose clip throughout the experiment and breathed via a mouthpiece connected to a Y valve (Collins P-319; 80 ml dead space). This valve allowed the participant to be switched from room air to the rebreathing bag. A tube attached to the valve sampled the air breathed at the mouth which allowed for close monitoring of the end-tidal values of carbon dioxide and oxygen (Bruel and Kjaer, anaesthetic monitor). The rebreathing bag, approximately 5 litres, was enclosed in a rigid container, and was connected to a dry rolling, real seal spirometer (Morgan Spiroflow, Model 130) by a short length of wide bore (37 mm) tubing to allow for monitoring of ventilation on a breath-by-breath basis. The rebreathing bag was filled with a gas mixture in which the partial pressure of carbon dioxide was set at 42 mmHg, the partial pressure of oxygen was established at 30 mmHg (hypoxic trial) or 170 mmHg (hyperoxic trial), and the remainder of the volume was comprised of nitrogen.

The participant was asked to hyperventilate for 5 minutes with room air through the Y valve in order to lower the end-tidal partial pressure of carbon dioxide to a level
between 20 to 24 mmHg. Then the participant was switched to the rebreathing bag and asked to take three deep breaths to ensure that the end-tidal pressure of carbon dioxide and oxygen in the bag, lungs, and arterial blood equilibrated quickly with the mixed venous partial pressure, which served as an estimate of the partial pressure in the tissues. A proper plateau in the end-tidal partial pressure of carbon dioxide evidenced adequate equilibration. The participant then rebreathed, under iso-oxic conditions maintained by a computer-controlled feedback system, at 50 mmHg or 150 mmHg of oxygen.

The end-tidal partial pressure of carbon dioxide increased linearly with time, with the test being terminated at a level of 60 mmHg. At this time, the participant was switched back to room air and remained seated for several minutes during recovery.

Swimming test protocol. The test was conducted in a long course (50 metre) pool. Before the swimming test, the foot length, arm length, and height of each participant was measured and recorded (Grimston & Hay, 1986). The anthropometric data was used to characterize the participants.

Each swim test consisted of a set of 5 x 200 metre and 1 x 150 metre freestyle swims on a pace time of 5 minutes. The researcher calculated the required speed for each 200 metre swim prior to the test, and the participants were informed of these target speeds before the test began. Each target speed was based upon percentage of the participant's best time, which in turn corresponded to a percentage of maximum heart rate. The 150 metre swim was a maximal effort performance at a distance (150 m) not normally swum competitively, and requiring maximal anaerobic power. The participants performed the swimming test using the freestyle stroke. Times were recorded using manual stop watches.

The 200 metre swims were accomplished as follows: (a) the first at a speed that would result in a heart rate of 140 (± 10) beats per minute (bpm), (b) the second at a speed that would result in a heart rate of 150 (± 10) bpm, (c) the third at a speed that would result in a heart rate of 160 (± 10) bpm, (d) the fourth at a speed that would result
in a heart rate of 170 (± 10) bpm, and (e) the fifth at a speed that would result in a heart rate of >180 (± 10) bpm. The final time and post-exercise heart rate for each 200 metre swim were measured and recorded. The 50 metre splits within each 200 metre swim were also measured and recorded. The post-exercise heart rates of the participants were measured using a heart rate monitor and were recorded after each 200 metre swim. The number of strokes taken on the third length of each 200 metre swim and the number of breaths on the last 50 of each 200 metre swim were counted by an assistant and recorded. Three minutes after participants completed the final 150 metre swim, a venous blood sample was collected after a finger stick and analyzed by an automated lactate analyzer (Arkray Lactate Pro LT-1710) for the determination of whole blood lactate.

The data from each 5 x 200 test were plotted and analyzed graphically to determine the swimmer’s critical velocity (Treffene, 1982), and measures of swimming economy and breathing economy.

The swim training consisted of 25 weeks of competitive swim training that included aerobic and anaerobic components. The training also included elements of controlled frequency breathing whereby the swimmers were asked to breathe every “x” number of strokes and “x” was a varied depending on the intensity of training that was required by the respective coaches.

PWC_{170} Test Protocol. All participants completed this general fitness test. A Monark cycle ergometer was utilized for this sub-maximal exercise test. The test involved riding a cycle ergometer at a number of progressively heavier workloads with the individual’s steady state heart rate being measured during the fifth minute of exercise at each load. The ergometer is designed so that one complete turn of the pedal moves a point on the rim of the fly wheel 6 metres. A metronome was set at 100 beats per minute to establish a cadence of 50 flywheel revolutions per minute. The tension (N) applied to the flywheel of the cycle ergometer, multiplied by the distance pedaled (m), yielded the amount of work done (Nm). The rate of work (power output) was expressed in kpm per minute (Nm min^{-1}). The subjects were given 3 minutes to warm up and familiarize
themselves with the cycle ergometer and prepare them for the exercise intensity in the first stage of the test. Heart rates were taken during the final 15 seconds of the second and third minutes of each stage. The participants then performed an additional four 3 minute stages at progressive increments in work rate, all of which remained sub-maximal. The heart rate measured during the last minute of each stage was plotted against work rate. The regression line generated from the plotted points was then extended to a heart rate of 170 beats per minute to determine the power output at this common heart rate. An analysis of covariance was used to determine differences between the groups.

Ethical Considerations

Informed consent. All information was given to the participants in a manner that was understandable to them (see Appendix A, pages 92, 100). Care was taken to explain the project, the procedures involved, and the goals and potential outcome of the research. In the informed consent, it was made clear to potential participants, in lay terms, that they had complete freedom of choice to participate or not, and that they were free to withdraw from the study at any time, with no repercussions from the investigators or from their coaches (see Appendix A, pages 95, 103). Copies of the letter explaining the study and the signed consent form were given to each participant.

Risks and benefits. Young, normal, healthy individuals are fully capable of completing the proposed rebreathing protocol. The risks from the procedure include dizziness, headaches, nausea, tightness of the chest, a sense of smothering, feelings of apprehension, heart palpitations, abdominal pains, faintness, or impaired consciousness. These symptoms have been reported for the Read rebreathing test (1967), but the modified version of the rebreathing test employed in this study (Mohan & Duffin, 1997) used lower partial pressures of carbon dioxide (35-60 mmHg vs. 45-70 mmHg PCO₂). No adverse effects have been observed in over 150 tests in the respiratory laboratory since these changes have been made. However, to ensure the safety of the participants, the
following additional precautions were taken during the rebreathing tests: (a) the end-tidal volumes of oxygen and carbon dioxide were monitored at all times, (b) the test was terminated if the carbon dioxide level rose above 60 mmHg (a normal level is 40 mmHg) or the oxygen level fell below 45 mmHg (a normal level is 100 mmHg), and (c) the oxygen saturation was continuously monitored with a pulse oximeter and was not allowed to fall below 75% (a normal level is 100%).

The swimming test (five 200 metre sub-maximal swims and the maximal 150 metre swim) was a normal experience for the participants. There were no known risks from completing the swimming test. Following the maximal 150 metre swim, a 50 µL blood sample was obtained. The risks associated with blood sampling included bruising and the possibility of infection. In order to eliminate the risks associated with blood sampling, the following precautions were taken. First, any materials that potentially could come into contact with blood were used only once and then immediately discarded into a biohazard waste disposal container. Such materials included all rubber gloves, finger sticks, capillary tubes, and alcohol swabs. Second, participants’ fingers were thoroughly disinfected with alcohol swabs prior to the finger stick puncture and after the blood sample had been drawn. Participants were provided with a Band-Aid for their fingers.

The PWC$_{170}$ test is a popular assessment technique for cardiorespiratory endurance that has been approved by the American College of Sports Medicine. All participants completed a physical activity readiness questionnaire prior to performing this test (see Appendix A, page 107). The risks associated with the PWC$_{170}$ test include muscle discomfort and shortness of breath. The researcher controlled for these risks by (a) monitoring heart rates at all times and terminating the test if the participant’s heart rate reached 85% of his or her age-predicted maximum heart rate (220 bpm-age), and (b)
terminating the test if the participant experienced signs of excessive discomfort or if the participant failed to conform to the exercise protocol.

Maintaining anonymity and confidentiality. The investigators kept all test results; each participant was assigned an individualized code, and only group result data will be published. All records will be kept in a secure location at the University of Toronto, and all data will be encoded in order to protect the names of the participants. Participants were able to request an examination of their own data.

Statistics and Data Analysis

The data (velocity, heart rate, lactate, number of strokes, and number of breaths) from each 5 x 200 test were analyzed graphically to determine critical velocity, swimming economy, and breathing economy parameters. The threshold and sensitivity of the central and peripheral chemoreflexes were determined through graphical analysis of the rebreathing test data.

Statistical analyses of the data proceeded as follows: (a) the data from the swimmers were assessed by a one-way repeated measures ANOVA, (b) the data from the swimmers and the non-athletes were examined via a two-way ANOVA, and (c) the data from the non-athletes (comparison group) were evaluated by a paired t-test to indicate stability of the measures. In each case, a p-value of <.05 was used to indicate statistical significance, and a Duncan's post-hoc or a Tukey test was used to identify differences between specific subgroups following demonstration of a significant ANOVA.
CHAPTER 3: RESULTS

This chapter will provide the reader with the main results of the research. The swimming tests indicated that measures of both swimming endurance and peak swimming speed were increased. Improvement in swimming economy variables was detected, although the results were not significant. A measure of breathing economy on the swimming tests was significantly improved. The rebreathing test results showed that the chemoreflex threshold changed with training, as did the chemoreflex ventilation sensitivity (non-significant), and the chemoreflex frequency sensitivity.

Sample Characteristics

The athlete group consisted of 21 highly trained competitive swimmers from 6 swim clubs in the Greater Toronto Area. These athletes ranged in age from 15 to 22, with a mean of 17.7 ± 2.0 years. All swimmers had achieved a performance within 5% of Canadian National Championship qualification times prior to the beginning of the study. Table 1 summarizes the descriptive information for the athlete group only. Table 1 also includes number of years swimming and the swimmers' best events.

The non-athlete group consisted of 14 high school and university students who had not participated in a regular training program for at least one year preceding this research project. Table 2 includes descriptive information about the non-athlete comparison group. Both tables summarize age, sex, height, arm length, foot length, and PWC\textsubscript{170} test results.
Table 1. Descriptive Characteristics of the Athlete Group

<table>
<thead>
<tr>
<th>Subject</th>
<th>Sex</th>
<th>Age (y)</th>
<th>PWC170 (Nm·min⁻¹)</th>
<th>Height (m)</th>
<th>Arm length (m)</th>
<th>Foot length (m)</th>
<th>Mass (kg)</th>
<th>Experience (y)</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1</td>
<td>F</td>
<td>16</td>
<td>4.95</td>
<td>1.70</td>
<td>0.79</td>
<td>0.26</td>
<td>58.0</td>
<td>7</td>
<td>200 Bk</td>
</tr>
<tr>
<td>A2</td>
<td>M</td>
<td>16</td>
<td>5.17</td>
<td>1.82</td>
<td>0.85</td>
<td>0.28</td>
<td>63.6</td>
<td>7</td>
<td>1500 Fr</td>
</tr>
<tr>
<td>A3</td>
<td>M</td>
<td>15</td>
<td>3.54</td>
<td>1.77</td>
<td>0.84</td>
<td>0.28</td>
<td>68.1</td>
<td>4</td>
<td>1500 Fr</td>
</tr>
<tr>
<td>A4</td>
<td>F</td>
<td>17</td>
<td>4.95</td>
<td>1.70</td>
<td>0.76</td>
<td>0.24</td>
<td>58.1</td>
<td>7</td>
<td>100 Fl</td>
</tr>
<tr>
<td>A5</td>
<td>M</td>
<td>17</td>
<td>4.50</td>
<td>1.80</td>
<td>0.85</td>
<td>0.27</td>
<td>65.8</td>
<td>8</td>
<td>1500 Fr</td>
</tr>
<tr>
<td>A6</td>
<td>F</td>
<td>15</td>
<td>4.50</td>
<td>1.60</td>
<td>0.72</td>
<td>0.22</td>
<td>49.9</td>
<td>5</td>
<td>400 Fr</td>
</tr>
<tr>
<td>A7</td>
<td>F</td>
<td>16</td>
<td>8.65</td>
<td>1.61</td>
<td>0.78</td>
<td>0.24</td>
<td>56.3</td>
<td>6</td>
<td>800 Fr</td>
</tr>
<tr>
<td>A8</td>
<td>F</td>
<td>16</td>
<td>7.17</td>
<td>1.77</td>
<td>0.81</td>
<td>0.24</td>
<td>57.6</td>
<td>7</td>
<td>800 Fr</td>
</tr>
<tr>
<td>A9</td>
<td>F</td>
<td>19</td>
<td>3.54</td>
<td>1.68</td>
<td>0.75</td>
<td>0.24</td>
<td>49.7</td>
<td>10</td>
<td>50 Fr</td>
</tr>
<tr>
<td>A10</td>
<td>M</td>
<td>18</td>
<td>5.80</td>
<td>1.72</td>
<td>0.78</td>
<td>0.25</td>
<td>69.5</td>
<td>8</td>
<td>200 Fl</td>
</tr>
<tr>
<td>A11</td>
<td>F</td>
<td>18</td>
<td>6.89</td>
<td>1.77</td>
<td>0.77</td>
<td>0.25</td>
<td>57.7</td>
<td>6</td>
<td>200 Fl</td>
</tr>
<tr>
<td>A12</td>
<td>M</td>
<td>18</td>
<td>6.00</td>
<td>1.72</td>
<td>0.83</td>
<td>0.27</td>
<td>72.6</td>
<td>9</td>
<td>1500 Fr</td>
</tr>
<tr>
<td>A13</td>
<td>M</td>
<td>18</td>
<td>4.08</td>
<td>1.90</td>
<td>0.90</td>
<td>0.28</td>
<td>81.7</td>
<td>9</td>
<td>1/200 Br</td>
</tr>
<tr>
<td>A14</td>
<td>F</td>
<td>17</td>
<td>7.17</td>
<td>1.68</td>
<td>0.74</td>
<td>0.23</td>
<td>52.2</td>
<td>3</td>
<td>200 Br</td>
</tr>
<tr>
<td>A15</td>
<td>M</td>
<td>18</td>
<td>6.75</td>
<td>1.80</td>
<td>0.82</td>
<td>0.26</td>
<td>72.2</td>
<td>9</td>
<td>1/200 Br</td>
</tr>
<tr>
<td>A16</td>
<td>F</td>
<td>18</td>
<td>3.54</td>
<td>1.66</td>
<td>0.77</td>
<td>0.24</td>
<td>56.8</td>
<td>7</td>
<td>100 Fl</td>
</tr>
<tr>
<td>A17</td>
<td>F</td>
<td>22</td>
<td>5.43</td>
<td>1.85</td>
<td>0.83</td>
<td>0.26</td>
<td>72.7</td>
<td>13</td>
<td>200 Fr</td>
</tr>
<tr>
<td>A18</td>
<td>F</td>
<td>16</td>
<td>6.89</td>
<td>1.73</td>
<td>0.82</td>
<td>0.26</td>
<td>60.3</td>
<td>6</td>
<td>100 Bk</td>
</tr>
<tr>
<td>A19</td>
<td>F</td>
<td>21</td>
<td>5.80</td>
<td>1.72</td>
<td>0.77</td>
<td>0.25</td>
<td>57.7</td>
<td>11</td>
<td>200 Fr</td>
</tr>
<tr>
<td>A20</td>
<td>M</td>
<td>18</td>
<td>6.75</td>
<td>2.06</td>
<td>0.99</td>
<td>0.32</td>
<td>83.5</td>
<td>7</td>
<td>100 Fr</td>
</tr>
<tr>
<td>A21</td>
<td>M</td>
<td>22</td>
<td>5.17</td>
<td>1.87</td>
<td>0.88</td>
<td>0.27</td>
<td>63.6</td>
<td>13</td>
<td>1500 Fr</td>
</tr>
</tbody>
</table>

Mean: Age 17.7, PWC170 5.58, Height 1.76, Arm length 0.81, Foot length 0.26, Mass 63.2, Experience 7.7

SD: Age 2.01, PWC170 1.40, Height 0.11, Arm length 0.06, Foot length 0.02, Mass 9.50, Experience 2.57
Table 2. Descriptive Characteristics of the Non-Athlete Comparison Group

<table>
<thead>
<tr>
<th>Subject</th>
<th>Sex</th>
<th>Age (y)</th>
<th>PWC170 (Nm min(^{-1}))</th>
<th>Height (m)</th>
<th>Arm length (m)</th>
<th>Foot length (m)</th>
<th>Mass (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NA1</td>
<td>F</td>
<td>18</td>
<td>4.11</td>
<td>1.61</td>
<td>0.77</td>
<td>0.23</td>
<td>52.2</td>
</tr>
<tr>
<td>NA2</td>
<td>F</td>
<td>27</td>
<td>4.02</td>
<td>1.64</td>
<td>0.72</td>
<td>0.24</td>
<td>54.5</td>
</tr>
<tr>
<td>NA3</td>
<td>M</td>
<td>18</td>
<td>4.52</td>
<td>1.77</td>
<td>0.83</td>
<td>0.27</td>
<td>61.3</td>
</tr>
<tr>
<td>NA4</td>
<td>F</td>
<td>18</td>
<td>4.62</td>
<td>1.63</td>
<td>0.77</td>
<td>0.24</td>
<td>49.9</td>
</tr>
<tr>
<td>NA5</td>
<td>F</td>
<td>18</td>
<td>3.18</td>
<td>1.65</td>
<td>0.66</td>
<td>0.24</td>
<td>61.3</td>
</tr>
<tr>
<td>NA6</td>
<td>F</td>
<td>17</td>
<td>2.65</td>
<td>1.58</td>
<td>0.74</td>
<td>0.25</td>
<td>60.0</td>
</tr>
<tr>
<td>NA7</td>
<td>F</td>
<td>18</td>
<td>3.89</td>
<td>1.63</td>
<td>0.76</td>
<td>0.23</td>
<td>52.2</td>
</tr>
<tr>
<td>NA8</td>
<td>F</td>
<td>18</td>
<td>5.56</td>
<td>1.56</td>
<td>0.71</td>
<td>0.21</td>
<td>45.4</td>
</tr>
<tr>
<td>NA9</td>
<td>F</td>
<td>18</td>
<td>1.27</td>
<td>1.63</td>
<td>0.75</td>
<td>0.25</td>
<td>53.2</td>
</tr>
<tr>
<td>NA10</td>
<td>F</td>
<td>18</td>
<td>4.23</td>
<td>1.60</td>
<td>0.73</td>
<td>0.25</td>
<td>54.5</td>
</tr>
<tr>
<td>NA11</td>
<td>M</td>
<td>18</td>
<td>2.82</td>
<td>1.69</td>
<td>0.80</td>
<td>0.25</td>
<td>66.7</td>
</tr>
<tr>
<td>NA12</td>
<td>F</td>
<td>19</td>
<td>3.18</td>
<td>1.71</td>
<td>0.76</td>
<td>0.24</td>
<td>51.2</td>
</tr>
<tr>
<td>NA13</td>
<td>M</td>
<td>18</td>
<td>3.76</td>
<td>1.92</td>
<td>0.93</td>
<td>0.31</td>
<td>85.4</td>
</tr>
<tr>
<td>NA14</td>
<td>F</td>
<td>22</td>
<td>3.89</td>
<td>1.60</td>
<td>0.76</td>
<td>0.22</td>
<td>54.7</td>
</tr>
</tbody>
</table>

| Mean    | 18.9 | 3.69  | 1.66 | 0.76 | 0.24 | 57.3 |
| SD      | 2.59 | 1.04  | 0.09 | 0.06 | 0.02 | 9.76 |

The findings of a statistical analysis (t-test) indicated statistically significant differences between the athlete and non-athlete groups only in the PWC\(_{170}\) test results. The mean PWC\(_{170}\) score for the athletes (5.58 Nm min\(^{-1}\) ± 1.40 SD) was significantly higher (\(p=<0.001\)) than that of the non-athlete group (3.69 Nm min\(^{-1}\) ± 1.04 SD).
The three test sessions coincided with the key phases of the competitive swimming season. The first test (test 1) was conducted after the preparation phase at week 3. The second test (test 2) coincided with the end of a high volume training camp and occurred during week 17. The last test (test 3) took place during the taper phase (one week before the major competition of the season) at week 25. Mean training volume (in m \cdot wk^{-1}) performed by the athletes, the sequencing and timing of the tests, and information about the swimming season are presented in Figure 2.

**Figure 2.** Training volume over time for the 25-week season.
Swimming Test Results

The data resulting from the swimming tests are presented in Appendix B. The results of analyzing the data are summarized in Table 3.

Table 3. Swimming Test Results and Statistical Analysis

<table>
<thead>
<tr>
<th>Variable (units)</th>
<th>Test</th>
<th>Trend</th>
<th>Significant?</th>
<th>ANOVA Level</th>
<th>t-test Level</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T₁</td>
<td>T₃</td>
<td>T₄</td>
<td>test 1, 2, 3</td>
<td>test 1 vs. 3</td>
</tr>
<tr>
<td>$V_{crl} (m \cdot s^{-1})$</td>
<td>1.40</td>
<td>1.49</td>
<td>1.49</td>
<td>increase</td>
<td>Yes</td>
</tr>
<tr>
<td>± SD</td>
<td>0.09</td>
<td>0.13</td>
<td>0.12</td>
<td></td>
<td>p&lt;=0.001</td>
</tr>
<tr>
<td>$DPS_{decay} (str \cdot sec^{-1})$</td>
<td>-1.07</td>
<td>-0.89</td>
<td>-0.89</td>
<td>decrease</td>
<td>No</td>
</tr>
<tr>
<td>± SD</td>
<td>0.51</td>
<td>0.46</td>
<td>0.39</td>
<td></td>
<td>p=0.18</td>
</tr>
<tr>
<td>$B_{slope} (BF/m.s^{-1})$</td>
<td>46.45</td>
<td>40.81</td>
<td>38.91</td>
<td>decrease</td>
<td>No</td>
</tr>
<tr>
<td>± SD</td>
<td>13.78</td>
<td>11.41</td>
<td>17.45</td>
<td></td>
<td>p=0.206</td>
</tr>
<tr>
<td>$V_{peak} (m.s^{-1})$</td>
<td>1.45</td>
<td>1.49</td>
<td>1.50</td>
<td>increase</td>
<td>Yes</td>
</tr>
<tr>
<td>± SD</td>
<td>0.12</td>
<td>0.13</td>
<td>0.11</td>
<td></td>
<td>p&lt;=0.001</td>
</tr>
<tr>
<td>$DPS_{peak} (m)$</td>
<td>1.10</td>
<td>1.11</td>
<td>1.14</td>
<td>increase</td>
<td>No</td>
</tr>
<tr>
<td>± SD</td>
<td>0.07</td>
<td>0.10</td>
<td>0.10</td>
<td></td>
<td>p=0.167</td>
</tr>
<tr>
<td>$DPB_{peak} (m)$</td>
<td>2.72</td>
<td>3.03</td>
<td>3.09</td>
<td>increase</td>
<td>Yes</td>
</tr>
<tr>
<td>± SD</td>
<td>0.41</td>
<td>0.38</td>
<td>0.51</td>
<td></td>
<td>p=0.013</td>
</tr>
</tbody>
</table>

Abbreviations: $V_{crl}$, critical velocity; $DPS_{decay}$, distance per stroke decay; $B_{slope}$, breathing frequency slope; $V_{peak}$, velocity on maximal 150 metre performance test; $DPS_{peak}$, distance per stroke on maximal 150 metre performance test; $DPB_{peak}$, distance per breath on maximal 150 metre performance test.

Critical Velocity

An example of the results of one critical velocity test is presented in Figure 3. The changes in mean critical velocity over time are illustrated in Figure 4. The mean critical velocity (Treffene, 1983) for the 15 swimmers who completed all the swimming tests showed statistically significant ($p<=0.001$) improvement from 1.40 m·s⁻¹ (±0.09) at test 1 to 1.49 m·s⁻¹ (±0.13) at test 2 after a high volume training camp (see Figure 2). Mean
Critical velocity remained relatively unchanged from the end of the training camp at test 2 to test 3, where mean critical velocity was also 1.49 m·s⁻¹ (±0.12). Critical velocity at test 3 was also significantly increased from test 1 (p<=0.001). A pairwise multiple comparison procedure (Tukey Test) confirmed the results of the one-way ANOVA, and indicated a statistically significant difference between test 1 and test 2 (p<=0.05) and between test 1 and test 3 (p<=0.05).

![Critical Velocity Analysis](image)

**Figure 3.** An example of the critical velocity analysis for one subject

In this example, critical velocity is the point on the x-axis that corresponds with the intersection of regression line of the velocity vs. heart rate plot from the 5 x 200 metre swim test and the individual athletes' maximum heart rate.
Figure 4. Changes in mean critical velocity over time.

This figure illustrates the changes in mean critical velocity at the three test points during the swimming season. The data points indicate the mean critical velocity and the error bars are ± standard error. There is a significant difference between test 1 and test 2, and between test 1 and test 3.

Peak Velocity on Maximal 150 Metre Performance Test

The changes in peak velocity are represented graphically in Figure 5. The mean swimming velocity on the maximal 150 metre performance test ($V_{\text{peak}}$) increased from test 1 to test 2 after the training camp, and again from test 2 to test 3 during the taper phase of the season (see Figure 2 and Table 3). The mean $V_{\text{an}}$ at test 1 was 1.45 m·s$^{-1}$ ($±$ 0.12), 1.49 m·s$^{-1}$ ($±$ 0.13) at test 2, and 1.50 m·s$^{-1}$ ($±$ 0.11) at test 3. There was a statistically significant increase (one-way repeated measures ANOVA, with the differences between the results at test 1, test 2, and test 3 ($p<0.05$) being isolated using a
Tukey test) in $V_{\text{peak}}$ from test 1 to test 2 ($p<0.001$), and from test 2 to test 3 ($p<0.001$), but no difference was detected between test 2 and test 3. The increases in velocity were achieved with no increase in mean blood lactate concentration (9.7 mmol/l at test 1, 9.8 mmol/l at test 2 and 9.6 mmol/l at test 3).

![Graph showing changes in peak velocity vs. time.](image)

**Figure 5.** Changes in peak velocity on maximal 150 metre performance test vs. time. This figure illustrates the mean peak swimming velocity on a maximal 150 metre performance test at three points during the swimming season. The error bars are ± standard error. There is a significant difference between test 1 and test 2, and between test 1 and test 3.

**Swimming Economy**

The technical characteristics of the swimmers were assessed in terms of (a) the decay in their distance per stroke during the progressive 5 x 200 and 1 x 150 metre swimming test and (b) by their distance per stroke during the maximal 150 metre performance test.
**Distance per stroke decay.** Distance per stroke decay (DPS_{decay}) was reported as the slope of the line describing the decrease in distance per stroke as swimming velocity increased (see Figure 6). The mean DPS_{decay} changed from -1.07 (±0.52) at test 1 to -0.89 (±0.46) at test 2 and -0.89 (±0.39) at test 3. Although the distance per stroke decreased at a lesser rate between test 2 and test 3 when compared to test 1, the differences between the mean DPS_{decay} were not significant (p=0.18). The greatest improvement in DPS_{decay} came after the high-volume training camp that preceded test 2.

**Distance per stroke on maximal 150 metre performance test.** The changes in DPS_{peak} are presented graphically in Figure 7. The mean distance per stroke of the swimmers on the maximal 150 metre performance tests (DPS_{peak}) increased slightly from 1.10 m'stroke^{-1} (±0.07) at test 1 to 1.11 m'stroke^{-1} (±0.09) at test 2. Mean DPS_{peak} at test 3 increased to 1.14 m'stroke^{-1} (±0.10) during the taper phase of the swimming season. Although distance per stroke was increased over the test period, a one-way repeated measures ANOVA did not detect any significant differences between the results at the three test dates (p=0.17).
Figure 6. An example of distance per stroke decay during the progressive swimming test for one subject.

This figure is an illustration of the graphical analysis of the progressive swimming test results. This graph shows the plot of velocity vs. distance per stroke and the regression line through these data points.
Figure 7. Changes in mean distance per stroke on maximal 150 metre performance test vs. time.

This figure illustrates the changes in the mean distance per stroke of the swimmers on the maximal 150 metre performance test at each of the three test points during the swimming season. The error bars are ± standard error. Changes in this variable were not significant.
Breathing Economy

The breathing patterns of the swimmers were assessed to determine the rate of increase in the swimmers' breathing frequency (breaths min\(^{-1}\)) as velocity increased during the progressive 5 x 200 and 1 x 150 metre swimming tests. The swimmers were also measured on their distance per breath (metres breath\(^{-1}\)) on the peak velocity 150 metre swim.

Breathing frequency. The mean slope of the plot of breathing frequency vs. velocity decreased from 46.5 (±13.78) at test 1 to 40.9 (±11.41) at test 2, and to 38.9 (±17.45) at test 3. The change in slope at each of the test dates is presented in Figure 8.

![Graph showing changes in breathing frequency over time. Error bars are ± standard error. No significant changes were detected.](image-url)
Statistical analysis (one-way RM ANOVA) did not detect a statistically significant difference between the groups of results ($p=0.21$). A t-test comparison of breathing frequency at test 1 and test 3 did not reveal a significant difference ($p=0.10$).

**Distance per breath on maximal 150 metre performance test.** The DPB_{peak} results are presented in Figure 9. The mean distance per breath (m) on the 150 metre swims increased at each of the three test times. The mean DPB_{peak} was 2.71 ($\pm 0.41$) at test 1, 3.03 ($\pm 0.38$) at test 2, and 3.09 ($\pm 0.51$) at test 3. The greatest increase in distance per breath (0.32 m from test 1 to test 2) occurred after the high volume training camp. The trend continued during the taper phase of the swim season with another 0.06 m increase from test 2 to test 3. A one-way repeated measures ANOVA detected a significant difference ($p=0.013$) between the distance per breath results at the three test points. A Tukey Test determined that there was a significant difference between the DPB_{peak} at test 1 and test 2 ($p<0.05$), and between test 1 and test 3 ($p<0.05$). No significant difference between the DPB_{peak} at test 2 and test 3 was found.

![Graph](image)

**Figure 9.** Distance per breath on maximal 150 metre performance test vs. time.
This figure presents the mean distance per breath of the swimmers on a 150-metre sprint at each of the three test points during the swimming season. The error bars are ± standard error. Significant differences were detected between test 1 and test 2 and between test 1 and test 3.

Rebreathing Test Results

The results of the individual rebreathing tests were recorded and then analyzed graphically. The raw results of the rebreathing tests are presented in Appendix C. The rebreathing tests were conducted at both a hypoxic level (i.e., PO₂ 50 mmHg) and a hyperoxic level (i.e., PO₂ 150 mmHg). From the individual graphs, the investigators were able to determine basal ventilation (Ve₀), the first chemoreflex ventilation threshold (Ve₁), the first chemoreflex ventilation sensitivity to carbon dioxide (VeS₁), the first tidal volume sensitivity (VTS₁), and the first frequency sensitivity (FS₁) for each of the hypoxic and hyperoxic tests. During the rebreathing tests, very few subjects were allowed to attain a PCO₂ that would have resulted in the subject reaching the second ventilation chemoreflex threshold (Ve₂) or chemoreflex ventilation sensitivity (VeS₂) because of safety protocols; thus results for Ve₂ and VeS₂ are not available. An example of the graphical analysis (with all variables) of the rebreathing results at a PO₂ of 50 mmHg for one participant is presented in Figure 10.
Figure 10. Graphical analysis model for rebreathing test results

For all three plots, the individual data points represent the breath by breath information vs. PCO$_2$ at a PO$_2$ of 50 mmHg. The first figure describes the plot of ventilation vs. predicted PCO$_2$, the second tidal volume vs. PCO$_2$, and the third frequency vs. PCO$_2$. This is an additive model with the tidal volume and frequency variables contributing to the observed total ventilation. Abbreviations: Veb, basal ventilation; Ve$_T1$, first chemoreflex ventilation threshold; Ve$_S1$, first chemoreflex ventilation sensitivity; Ve$_T2$, second chemoreflex ventilation threshold; Ve$_S2$, second chemoreflex ventilation sensitivity; VT$_T1$, first tidal volume threshold, VT$_S1$, first tidal volume sensitivity; VT$_T2$, second tidal volume threshold, VT$_S2$, second tidal volume sensitivity; FT$_1$, first frequency threshold; FS$_1$, first frequency sensitivity; FT$_2$, second frequency threshold; FS$_2$, second frequency threshold.
Table 4 presents the results from analyzing the rebreathe tests, including the ventilation variables, oxygen levels, and the results of the statistical analyses.

Table 4. Rebreathing Test Results and Statistical Analysis

<table>
<thead>
<tr>
<th>Ventilation variable</th>
<th>PO2</th>
<th>Group</th>
<th>ANOVA</th>
<th>Group</th>
<th>t-test</th>
<th>level</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Athlete</td>
<td>experimental</td>
<td>test 1</td>
<td>test 2</td>
<td>test 3</td>
</tr>
<tr>
<td>Basal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Veb</td>
<td>50</td>
<td>11.76</td>
<td>10.29</td>
<td>9.50</td>
<td>p=0.22</td>
<td>9.00</td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>7.27</td>
<td>7.73</td>
<td>7.69</td>
<td>p=0.96</td>
<td>7.53</td>
</tr>
<tr>
<td>VTb</td>
<td>50</td>
<td>835.72</td>
<td>764.64</td>
<td>693.11</td>
<td>p=0.81</td>
<td>573.04</td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>338.42</td>
<td>697.12</td>
<td>601.13</td>
<td>p=0.81</td>
<td>574.25</td>
</tr>
<tr>
<td>Fb</td>
<td>50</td>
<td>15.96</td>
<td>12.39</td>
<td>14.92</td>
<td>p=0.06</td>
<td>11.60</td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>16.79</td>
<td>12.68</td>
<td>14.36</td>
<td>p=0.39</td>
<td>10.89</td>
</tr>
<tr>
<td>Thresholds</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VeT1</td>
<td>50</td>
<td>40.87</td>
<td>42.88</td>
<td>42.56</td>
<td>p=0.01</td>
<td>43.16</td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>44.48</td>
<td>45.59</td>
<td>44.82</td>
<td>p=0.59</td>
<td>46.06</td>
</tr>
<tr>
<td>VTT1</td>
<td>50</td>
<td>39.94</td>
<td>42.33</td>
<td>41.99</td>
<td>p=0.04</td>
<td>41.71</td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>41.18</td>
<td>45.33</td>
<td>44.37</td>
<td>p=0.22</td>
<td>45.09</td>
</tr>
<tr>
<td>FT1</td>
<td>50</td>
<td>40.94</td>
<td>44.03</td>
<td>44.28</td>
<td>p=0.01</td>
<td>44.16</td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>xx</td>
<td>46.77</td>
<td>47.12</td>
<td>p=0.87</td>
<td>46.35</td>
</tr>
<tr>
<td>T1 (average of VeT1, VTT1, FT1)</td>
<td>50</td>
<td>40.58</td>
<td>43.08</td>
<td>42.94</td>
<td>p=0.01</td>
<td>43.01</td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>42.83</td>
<td>45.90</td>
<td>45.37</td>
<td>p=0.32</td>
<td>45.83</td>
</tr>
<tr>
<td>Sensitivities</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VeS1</td>
<td>50</td>
<td>5.21</td>
<td>3.83</td>
<td>3.54</td>
<td>p=0.05</td>
<td>3.60</td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>2.98</td>
<td>2.52</td>
<td>2.46</td>
<td>p=0.45</td>
<td>2.28</td>
</tr>
<tr>
<td>VTS1</td>
<td>50</td>
<td>191.42</td>
<td>179.27</td>
<td>189.63</td>
<td>p=0.88</td>
<td>146.48</td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>123.72</td>
<td>133.47</td>
<td>138.51</td>
<td>p=0.73</td>
<td>97.24</td>
</tr>
<tr>
<td>FS1</td>
<td>50</td>
<td>0.91</td>
<td>0.71</td>
<td>0.34</td>
<td>p=0.06</td>
<td>1.19</td>
</tr>
<tr>
<td></td>
<td>150</td>
<td>0.14</td>
<td>0.38</td>
<td>0.32</td>
<td>p=0.96</td>
<td>0.98</td>
</tr>
</tbody>
</table>

Abbreviations: Veb, basal ventilation; VTb, basal tidal volume; Fb, basal frequency; VeT1, first chemoreflex ventilation threshold; VTT1, first tidal volume threshold; FT1, first frequency threshold; T1, the average of VeT1, VTT1 and FS1; VeS1, first chemoreflex ventilation sensitivity; VTS1, first tidal volume sensitivity; FS1, first frequency sensitivity. Units: Veb (l min⁻¹); VTb (ml min⁻¹), Fb (breaths min⁻¹); VeT1 (mmHg CO₂); VTT1 (mmHg CO₂); FT1 (mmHg CO₂); T1 (mmHg CO₂); VeS1 (L min⁻¹(mmHg CO₂)⁻¹); VTS1 (L min⁻¹(mmHg CO₂)⁻¹); FS1 (L min⁻¹(mmHg CO₂)⁻¹).

The athletes were tested on all rebreathing measures within one week of having completed their swimming tests. Therefore, test 1 in the rebreathing tests corresponds to the same point in the swimming season as test 1 in the swimming tests. The non-athletes were tested at the outset of the research, as were the athletes; thus test 1 corresponds to
the same time in the season, and testing date, for both the athlete and non-athlete groups. The non-athletes completed their second set of rebreathing tests at the end of the swimming season during the same period that the athletes were completing their third set of tests. Thus, test 2 for the non-athletes corresponds to the same testing period as test 3 for the athletes.

**Basal Ventilation**

Basal ventilation did not change significantly for either the athlete (p=0.22 in hypoxia, p=0.96 in hyperoxia, one-way RM ANOVA) or non-athlete groups (p=0.41 in hypoxia, p=0.38 in hyperoxia, paired t-test) over the testing period. There were no significant differences between the athletes and non-athletes in basal ventilation at either the hypoxic (p=0.41 on unpaired t-test) or hyperoxic levels (p=0.76 on unpaired t-test). No significant changes in basal tidal volume or basal frequency in either the athlete (one-way ANOVA) or non-athlete (paired t-test) groups were detected.

**Chemoreflex Threshold**

The chemoreflex threshold (T1) results for tests done in hypoxia are presented in Figure 11. The chemoreflex threshold represents the mean partial pressure of carbon dioxide (PCO2) level where ventilation (VeT1), tidal volume (VTT1), and frequency (FT1) all begin to increase in response to an increase in PCO2 during the rebreathing test.

The chemoreflex threshold for the athlete group in hypoxia significantly increased from 40.58 mmHg CO2 (±2.95) at test 1 to 43.08 mmHg CO2 (±2.93) at test 2, but decreased slightly to 42.94 mmHg CO2 (±2.27) at test 3. The increase in T1 was significant at the p=0.01 level (RM ANOVA). There were no statistically significant differences (p=0.76) for the non-athletes between the T1 levels (43.01 mmHg CO2 at test 1 and at test 2 42.75 mmHg CO2). The mean T1 levels for the athlete and non-athlete groups were compared at the initial and final test dates for both groups. Although the T1 of the athletes was lower than that of the non-athletes at test 1 (40.58 ± 2.95 vs. 43.01 ±
2.85), the difference was not statistically significant (p=0.08). There were also no statistically significant differences between the athletes and the non-athletes at the final test period (p=0.96).

In hyperoxia, there were no statistically significant changes in mean T1 between test 1, test 2, and test 3 for the athlete group (p=0.59), or between test 1 and test 2 for the non-athlete group (p=0.98).

![Figure 11](image.png)

**Figure 11.** Changes in chemoreflex threshold (hypoxia) for the athlete and non-athlete groups over time.

This figure illustrates the changes in chemoreflex threshold (the average of VeT1, VTT1, and FT1) in the athlete and non-athlete groups over time. The error bars are ± standard error.
Chemoreflex Ventilation Sensitivity

The chemoreflex ventilation sensitivity (VeSI) was determined as the slope of the line that represents the linear increase in ventilation that occurs in response to an increase in the inspired partial pressure of carbon dioxide (see Figure 10). VeSI was measured under both hypoxic and hyperoxic conditions.

The athlete group exhibited a statistically significant decrease in ventilatory sensitivity to carbon dioxide in hypoxia over the three tests. The mean VeSI for the athletes was 5.21 (±3.47) at test 1, 3.83 (±2.61) at test 2, and 3.58 (±2.53) at test 3. The difference between the results for the athletes at test 1 and test 2 was statistically significant (p<0.05), as was the difference between test 1 and test 3 (p<0.05) according to the results of the RM ANOVA test. No statistically significant difference was found between the results for the athletes at test 2 vs. test 3. The mean results for the non-athlete comparison group were 3.80 (±2.06) at test 1 and 4.05 (±2.53) at test 2. A t-test indicated that the difference was not statistically significant (p=0.81). No significant differences were detected between athletes and non-athletes. These results are presented in Figure 12.
Figure 12. Changes in chemoreflex ventilation sensitivity (hypoxia) over time for the athlete and non-athlete groups. Error bars are ± standard error.

The results of the statistical analysis of the rebreathing tests conducted under hyperoxic conditions did not indicate any significant differences in the chemoreflex ventilation sensitivity to carbon dioxide over time in either the athlete (p=0.45), or non-athlete (p=0.41) groups.

**Tidal Volume and Frequency Sensitivity.**

The increase in ventilation that accompanied the increase in the partial pressure of carbon dioxide (PCO₂) during the rebreathing test was analyzed graphically to determine the tidal volume and breathing frequency responses. Tidal volume and breathing frequency sensitivity to the increases in PCO₂ were derived from the slope of the increase the respective variable. An example of the graphical analysis of tidal volume is presented as the second plot in Figure 10 and an analysis of breathing frequency is the third graph in Figure 10.
Tidal volume sensitivity was not statistically different over time in either hypoxia (p=0.88) or hyperoxia (p=0.73) in the athlete group. Tidal volume exhibited a non-significant increase from test 1 to test 2 in the non-athlete group in both hypoxia (p=0.14) and in hyperoxia (p=0.27). These results are summarized in Table 4.

The mean ventilation frequency sensitivity results from the hypoxic rebreathing tests are presented in Figure 13. The first ventilation frequency sensitivity during the hypoxic rebreathing tests decreased from 0.91 (±1.09) at test 1, to 0.71 (±0.82) at test 2, to 0.34 (±0.48) at test 3 in the athlete group. The decrease over time was not statistically significant on an ANOVA analysis (p=0.06). No differences in the ventilation frequency sensitivity between tests were found in the athlete group under hyperoxic conditions (p=0.96), the non-athletes in hypoxia (p=0.93), or in the non-athlete group in hyperoxia (p=0.18).

![Figure 14. Ventilation frequency sensitivity to carbon dioxide (hypoxia) over time for the athlete and non-athlete groups. Error bars are ± standard error.](image-url)
Correlation Analysis

The mean results for each variable were analyzed to determine if there were any significant relationships between the variables. The results of the correlation analysis are presented in Table 5. Significant relationships are highlighted in bold font.

Table 5. Correlation Analysis Chart

<table>
<thead>
<tr>
<th></th>
<th>Vr</th>
<th>DPSdcoav</th>
<th>BISlope</th>
<th>Vpeak</th>
<th>DPSpeak</th>
<th>DPFpeak</th>
<th>TI</th>
<th>VeSI</th>
<th>FS1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vr</td>
<td>–</td>
<td>0.17 (p=0.03)</td>
<td>0.03 (p=0.93)</td>
<td>0.33 (p=0.27)</td>
<td>0.23 (p=0.44)</td>
<td>0.15 (p=0.65)</td>
<td>0.37 (p=0.21)</td>
<td>0.04 (p=0.91)</td>
<td>0.59 (p=0.03)</td>
</tr>
<tr>
<td>DPSdcoav</td>
<td>–</td>
<td>0.19 (p=0.52)</td>
<td>0.04 (p=0.18)</td>
<td>0.27 (p=0.37)</td>
<td>0.01 (p=0.97)</td>
<td>0.12 (p=0.71)</td>
<td>0.29 (p=0.34)</td>
<td>0.05 (p=0.87)</td>
<td></td>
</tr>
<tr>
<td>BISlope</td>
<td>–</td>
<td>–</td>
<td>0.47 (p=0.10)</td>
<td>0.40 (p=0.18)</td>
<td>0.52 (p=0.04)</td>
<td>0.08 (p=0.88)</td>
<td>0.04 (p=0.91)</td>
<td>0.41 (p=0.16)</td>
<td></td>
</tr>
<tr>
<td>Vpeak</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>0.16 (p=0.59)</td>
<td>0.47 (p=0.10)</td>
<td>0.07 (p=0.80)</td>
<td>0.02 (p=0.75)</td>
<td>0.40 (p=0.17)</td>
<td></td>
</tr>
<tr>
<td>DPSpeak</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>0.77 (p=0.002)</td>
<td>0.13 (p=0.68)</td>
<td>0.01 (p=0.98)</td>
<td>0.31 (p=0.37)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DPFpeak</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>0.13 (p=0.66)</td>
<td>0.008 (p=0.98)</td>
<td>0.24 (p=0.43)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TI</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>0.35 (p=0.25)</td>
<td>0.21 (p=0.49)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VeSI</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>0.54 (p=0.03)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FS1</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td></td>
</tr>
</tbody>
</table>

Significant relationships were detected between the increase in critical velocity and decrease in chemoreflex frequency sensitivity (p=0.03); the decrease in breathing frequency slope and increase in distance per breath on the maximal 150 metre test; between distance per stroke and distance per breath on the maximal 150 metre performance test (p=0.002); and between the decrease in chemoreflex ventilation sensitivity and chemoreflex frequency sensitivity (p=0.05). A moderate non-significant relationship was detected between the distance per breath and peak velocity on the maximal performance test (p=0.10).
CHAPTER 4: DISCUSSION

Introduction

Analysis of the swimming test results of the indicated significant increases in critical velocity (p<=0.001) and peak velocity (p<=0.001). Measurement of the decay in distance per stroke during the progressive swim test and distance per stroke on the maximal 150 metre performance test indicated a non-significant increase in both variables following training. The slope of the plot of velocity vs. breathing frequency on the progressive swim test was decreased 16% following training, however, the result was not significant. Distance per breath on the maximal 150 metre performance test increased significantly after training (p=0.01). Consideration of these results confirmed the alternative hypothesis, i.e., that swim training results in changes in various measures of swimming performance.

After training, the athlete group demonstrated a significant increase in the chemoreflex threshold (p<0.01), a significant decrease in first chemoreflex sensitivity (p=0.05), no change in the first tidal volume sensitivity, and a nearly significant decrease in the first frequency sensitivity (p=0.06) in hypoxia. There were no significant changes in the chemoreflex variables for the athlete group in hyperoxia. Further, no significant changes in the rebreathing test variables were observed in the non-athlete comparison group in either hypoxia or hyperoxia. These results provided evidence in support of the alternative hypothesis that training results in an attenuation of the chemoreflex response to carbon dioxide in hypoxia, but not in hyperoxia, i.e., an attenuation of the peripheral chemoreflex response.

Correlation analysis indicated significant correlations between (a) changes in distance per breath on the maximal 150 metre swim and changes in the slope of the increase in breathing frequency on the progressive swimming test; (b) changes in distance per breath on the maximal 150 metre swim and changes in the distance per stroke on the maximal 150 metre swim; (c) changes in critical velocity and changes in chemoreflex...
frequency sensitivity; and (d) changes in chemoreflex ventilation sensitivity and changes in chemoreflex frequency sensitivity. It was hypothesized that there would be a correlation between changes in the chemoreflex variables and changes in measures of swimming performance. Based upon the results of the correlation analysis, this hypothesis was accepted.

In addition to the evaluation of the effect of training on the athlete group, the present research compared athletes to non-athletes in terms of their chemoreflex response to carbon dioxide. It was hypothesized that athletes would have an attenuated chemoreflex response to carbon dioxide in hypoxia when compared to non-athletes. Yet, the mean results across all tests for the chemoreflex ventilation threshold, the first chemoreflex ventilation sensitivity, and the first tidal volume sensitivity did not indicate any significant differences. The mean first frequency sensitivity was lower in the athletes vs. the non-athletes (0.69 ±0.81 vs. 1.17 ±1.23), although the difference was not significant (p=0.06). Consequently, the alternative hypothesis that athletes have an attenuated chemoreflex response to carbon dioxide in hypoxia was rejected. Since the results of the present study did not show any significant differences between athletes and non-athletes in their chemoreflex responses to carbon dioxide in hyperoxia, the null hypothesis was confirmed.

Swimming Test Results

Critical Velocity

Treffene's (1982) concept of critical velocity, which Wakayoshi et al. (1993) and Norris and Smith (1995) recently expanded, was examined over a competitive swimming season. An improvement in the critical velocity indicates an increase in the athlete's capacity for doing more work (swimming at a faster velocity) at the same or a reduced heart rate. The mean critical velocity of the athlete group increased (i.e., improved) over the course of this study. The increase in critical velocity was most pronounced after the high volume training camp which occurred just prior to the second test. This result was
indicated by a rightward shift in the heart rate - velocity relationship used to determine the individual critical velocity at each test (see Figure 3). The participant’s improved critical velocity can be attributed to two factors: an improvement in aerobic metabolism and thus increased endurance, an improved resistance to fatigue, and improved stroke efficiency, i.e., an improved force application / decreased resistance during the stroke.

Since an improved critical velocity has been reported to be indicative of an increased general endurance performance (Smith, 1995), and sensitive to changes in aerobic fitness following training (Gaesser and Wilson, 1988, Jenkins and Quigley, 1992), the observed improvement in the athlete group’s critical velocity suggests that their general endurance performance and aerobic fitness had improved. The change in critical velocity would also indicate an improvement in gross cardiorespiratory fitness and aerobic capacity (Carlile, 1979). The improvement in endurance performance can be explained by Fitts et al. (1989) who reported a correlation between the increase in oxidative capacity (specifically, the end oxidation of substrates of the citric acid cycle and the respiratory chain) of limb skeletal muscle and endurance exercise training. Fitts et al. (1989) suggested that this improvement in endurance performance indicated beneficial adaptations of muscle mitochondrial content, enzymes of aerobic metabolism and fat oxidative capacity. Holloszy et al. (1984) and Hill (1993) believed that critical velocity reflects aerobic metabolic capabilities and improved endurance. Bonner (1980) and Bonen (1977) also noted potential benefits of aerobic training. They stated that endurance training leads to the following effects on the aerobic metabolic system: an increased vascularization within the muscle, which enhances the delivery of oxygen and nutrients to the muscle, an increase in the complex energy systems, which facilitates the improved aerobic metabolism of glucose and fats, and leads to a preferential utilization of fats rather than glycogen.

Thus, in the present case, training may have resulted in extensive circulatory adaptations, altered energy metabolism and fuel utilization, as well as an improved systemic fatigue resistance. These factors may have accounted for the improvement in the critical velocity observed in the current study’s participants.
Peak Velocity

The mean peak velocity of the swimmers on the maximal 150 metre performance test (a maximal 150 m sprint) showed significant improvement over the course of the research. This improvement was most pronounced during the taper phase near the end of the season, one week before the season’s major competition. The literature that suggests that the improvement in peak velocity on the maximal 150 metre performance test can be attributed to alterations in anaerobic metabolism, the development of lactic acidosis and the effects of lactic acid production on swimming velocity is presented below.

During heavy exercise, lactic acid is produced in muscle from the anaerobic breakdown of glycogen and glucose (Astrand, 1986). Wakayoshi et al. (1993) showed that aerobic swim training of the type performed in this study decreases post-maximal effort lactate levels despite significant increases in swimming velocity. Donovan (1990) attributed the increase in swimming velocity and decrease in lactate levels to an enhanced efficiency for blood lactate elimination and utilization through the adaptations that occur in response to endurance training. The decrease in blood lactate, or the increase in swimming velocity at a given blood lactate level, have been attributed to adaptations at the cellular level, such as an increased buffer capacity, enhanced removal and oxidation rate, and a reduction in the metabolic cost of swimming due to technical improvements and reduced drag (Wakayoshi et al., 1993).

Fatigue resistance is important to achieve an increased swimming velocity. Changes in swimming velocity and have been evidenced by changes in the electromyographic fatigue threshold (deVries et al., 1982). In a review of various aspects of fatigue, Green (1987) stated that chronic exercise training elicits a reduction in anaerobic glycolysis, an increase in the utilization of pyruvate, an increase in the uptake, transport, and utilization of free fatty acids, and a reduction in the intracellular utilization of glycogen. Furthermore, he contended that this “muscle glycogen sparing effect” which occurs following training is one of the main factors that facilitate the improvements in fatigue resistance (Green, 1987).
Various authors have presented several potential explanations for this improvement in resistance to fatigue. Recent literature suggests that potassium may play a role in muscle metabolism and fatigue during intense exercise (Bangsbo et al., 1996). Alternatively, intense exercise is associated with a high production of lactate and a subsequent decrease in pH within the exercising muscle, which may cause fatigue during intense exercise (Sahlin, 1986); in this case, fatigue is attributed to a decreased flux through glycolysis due to the increase in the muscle H⁺ concentration, which inhibits the activity of phosphorylase and phosphofructokinase, key regulating enzymes of the glygogenolytic and glycolytic pathways, respectively. Bangsbo (1996) has suggested that lactate accumulation and the resulting pH changes may not be the exclusive determinants of fatigue. Bangsbo (1996) postulated that progressive accumulation of potassium in the interstitium during intense exercise is implicated in the fatigue process. Bangsbo (1996) found an elevated level of blood potassium concentration in both control and high lactate groups at exhaustion, suggestive that fatigue occurs when a given potassium concentration has been reached in the interstitium. Two possible mechanisms for this finding were postulated, namely, inhibition at the spinal level from over stimulation of sensory receptors of group III and IV nerve fibres or alternatively, inhibition of the propagation of the action potential over the sarcolemma or possibly blocking its propagation to the T-tubules.

The improved peak velocity that was observed in this study may have resulted from an improvement in the technical efficiency of swimming and / or adaptations in aerobic / anaerobic metabolic capacity. The contribution of technical efficiency in terms of distance per stroke and breathing economy measures will be explored in the following sections.

Swimming Economy

Swimming velocity ($V, \text{ m } \cdot \text{s}^{-1}$), is the product of the stroke rate ($SR, \text{ strokes } \cdot \text{s}^{-1}$), and distance per stroke ($DPS, \text{ m } \cdot \text{stroke}^{-1}$), that is, $V=(SR)(DPS)$. Changes in velocity result from changes in one, or both, of stroke rate and distance per stroke. Craig and
Pendergast (1979), who described the relationship between velocity, stroke rate, and distance per stroke, reported that the fastest swimmers had the longest distance per stroke at sub-maximal velocities, and that there was a positive correlation between maximal distance per stroke and maximal velocities. Craig and Pendergast (1979) indicated that there could be an optimal combination of distance per stroke and stroke rate for each swimmer, and that this optimal combination involved considerations of local muscle power and endurance. The results of the current research indicated that there were non-significant improvements in both the decay of distance per stroke (the slope of the decrease in distance per stroke with increasing velocity), and the measured distance per stroke on the maximal 150 metre performance test.

Although the improvements in the measures of distance per stroke were not significant, they may well have contributed to the significant improvement in critical and peak velocity. The research literature supports this hypothesis. Craig, Skehan, Pawelczyk and Boomer (1985), in their examination of swimmers at the 1984 U.S. Olympic Swimming Trials, found that in 12 events, mean velocities were improved over those of the 1976 Trials. They suggested that in 9 of the 12 events, the improvement occurred as a result of an increased distance per stroke. Further analysis of the length by length profiles of the velocities during the 200 m, 400 m, 800 m, and 1500 m events showed that as fatigue developed, the distance per stroke decreased. Fatigue was thought to be related to a decreasing ability to develop the force necessary to overcome the resistance to forward movement. The authors concluded that improvements and superiority in stroke mechanics are reflected in the stroke rate and distance per stroke, and that changes in one or both account for improvements and decrements in velocity.

A review of the research literature suggests that the improvement in various measures of distance per stroke may have occurred due to an improved swimming efficiency, and improved metabolic capability, the psychological effects of a taper, and adaptations of the nervous system. Wakayoshi et al. (1995) presented evidence describing the relationship between velocity and distance per stroke, they found that high performance swimmers had a greater distance per stroke than lower performance
swimmers at a given velocity. The hypothesized rationale for a greater distance per stroke at a given velocity is that less metabolic power is required to transfer kinetic energy to a given mass of water; thus, an increased amount of energy is left available for the swimmer's propulsion. They concluded that the technical improvements that occur with swim training are reflected by an increase in distance per stroke, as well as by a decrease in oxygen uptake at a given velocity, indicating an enhanced swimming efficiency and reduced metabolic power requirements. More recently, Wakayoshi et al. (1993) studied the effects of 6 months of aerobic swim training on velocity and distance per stroke. They found that after training, velocity for 400 m was increased, and that the increase in velocity was associated with an increased distance per stroke.

Toussaint and Beek (1992) also suggested that the increase in distance per stroke would result in a decrease in the energy cost of swimming at a given velocity. Further, Craig et al. (1985) have reported that the increases in distance per stroke were accounted for by increases in the endurance ability of local muscle groups. On this basis, Wakayoshi et al. (1996) suggested that the 6 months of aerobic training promoted an increased work per stroke through an improvement in muscle endurance. Costill (1988) and Sharp (1982) proposed that biomechanics also may contribute to the performance improvements seen following a taper period. They further suggested that the relationship of power to muscular endurance, i.e., the number of repetitions and the length of performance time as described by Hickson (1980), are significant factors in muscular endurance. Power (P) is the amount of work (the product of force (F) and distance(D)) that can be performed per unit of time (T) (i.e., P=F x D/T), or in other words, power is a product of force and velocity. Hickson (1980) listed several important cellular adaptations that would result in an increase in muscular endurance and power. These adaptations include (a) an increase in the enzyme activities of phosphorylase and phosphofructokinase, the so-called rate limiting enzymes of glycolysis, (b) an increase in the concentrations of ATP and PC in the muscle, and in the ability to resynthesize and maintain ATP, (c) an increase in glycogen storage, (d) an increase in lactic acid tolerance, and (e) an increase in the rates of calcium transport and cross bridge cycling.
Interestingly, the results of the current research show an increase in distance per stroke (to 1.14 m \cdot \text{stroke}^{-1}) during the taper period. This test was performed during a full taper period in which the total training volume and intensity were reduced prior to a major competition (see Figure 2). Several other studies have shown that a taper period is related to improvements in competition performance, muscular power and distance per stroke (Costill et al., 1988; Johns et al., 1992; Van Handel et al., 1988). Rushall et al. (1990) and Wilmore et al. (1988) have attributed these performance improvements during taper to the result from neuromuscular and psychological recovery, with little evidence of change in other aspects of physiological status. They stated that strength and power could be markedly increased, and stroke efficiency improved considerably, with a reduced volume and intensity, provided that adequate training had occurred prior to the taper period. Rushall et al. (1993) and Wilmore et al. (1988) also suggested that the adaptive responses were neuromuscular and cognitive in origin. Johns and Houmard (1992) summarized what has been studied about the physiological effects during the taper phase of training. They stated that improvements in performance occur following a taper without changes in maximal aerobic power, and that any physiological changes associated with taper are likely associated with adaptations at the muscular level, rather than resulting from changes in O$_2$ delivery. Furthermore, they reported that muscular power is probably the major factor responsible for the improvement in swimming performance.

Costill et al. (1988), in a study of 17 collegiate level swimmers, demonstrated an improvement in Biokinetic SwimBench power during taper. Power on the SwimBench has been shown to be closely correlated to freestyle sprinting velocity (Sharp, 1982). During taper, power on the SwimBench improved 17.7% and power measured in the water increased 24.6%. The performances achieved following the reduced training correlated significantly ($r = -0.68$) with the percentage improvement during the swim power test. Other physiological variables that were measured in this study, such as post-swim venous blood lactic acid measurements and changes in pH, HCO$_3$-, and recovery heart rate, showed non-significant decreases after the taper period. Unfortunately, no
attempt was made to measure the impact that psychological factors may have had on the results of the power or performance tests.

Moreover, from the work of Davis et al. (1997), it seems that adaptations in the nervous system account for a significant portion of the observed increase in distance per stroke. This, and other research on the response of the nervous system to training may explain the current results which show an improvement in distance per stroke. In a review of the mechanisms leading to central nervous system (CNS) fatigue during exercise, Davis et al. (1997) have reported that the neurotransmitters serotonin (5-HT), dopamine (DA), and acetylcholine (Ach), as well as neuromodulators, like ammonia and cytokines (substances secreted from immune cells), are associated with the development of fatigue. Davis et al. (1997) defined CNS fatigue as a failure to maintain the required or expected force or power output, and is associated with specific alterations in CNS function that cannot be reasonably explained by dysfunction within the muscle itself. This definition is broad enough to allow for psychological considerations, such as motivation and perception, to be included. The wording of the working definition is important in that psychological factors are also possible explanations for fatigue during performance. Serotonin may be a mediator of CNS fatigue, because an increase in brain serotonin has been shown to have important effects on arousal, lethargy, sleepiness and mood, and has been linked to altered perceptions of effort and muscular fatigue. According to the central fatigue hypothesis (Newsholme, 1987), an increased concentration of serotonin in the brain can impair CNS function during prolonged exercise and, thereby, lead to a deterioration in sport and exercise performance. Another CNS factor to consider is the decrease in the concentrations of DA in the brain during prolonged exercise; this decrease has been correlated with the point at which an individual experienced feelings of fatigue. DA has been linked with increased arousal, motivation, muscular co-ordination, and increased endurance performance. The release of ammonia into the blood during exercise also could alter CNS function by acting directly on the brain, or by altering the permeability of the blood brain barrier to amino acids involved in neuro-transmission. Green (1987) discussed the impact of CNS fatigue on performance and listed five aspects of central fatigue to consider: supraspinal failure, segmental afferent inhibition,
depression of motor neuron excitability, loss of excitation at branch points and presynaptic failure. All of the research discussed in this paper indicates that nervous system function and adaptations to training should be taken into consideration by coaches when interpreting changes in performance (or any measures of function or adaptation) during a swimming season.

**Breathing Economy**

As a result of the current research, a new performance variable "breathing economy" is defined as the product of breathing frequency, measured in breaths·min⁻¹, and distance per breath, measured in m·breath⁻¹. In this study, breathing frequency was analyzed in terms of the slope of the increase in breathing rate vs. time during the incremental swimming test. Distance per breath was measured as the number of breaths taken divided by the distance travelled on the maximal effort 150 m performance test. Breathing economy has not been previously reported in the literature. The results of the present study indicated that there was a non-significant decrease (improvement) in the slope of breathing frequency vs. velocity but a significant increase (improvement) in distance per breath on the maximal 150 metre performance test.

It is possible that the observed improvement in breathing frequency was a major factor accounting for the observed improvement in the critical velocity. Interestingly, there was a correlation between improvements in critical velocity and improvements in chemoreflex frequency sensitivity. Frequency sensitivity was also correlated with chemoreflex ventilation sensitivity. It is possible that the improvement in chemoreflex ventilation sensitivity may have been a factor that allowed the swimmers to improve their distance per breath. Further, the significant increase in distance per breath may have allowed for the increase in peak velocity. The ability to control breathing during performance would allow for an improved technical efficiency and a reduced drag, because turning the head to breathe in freestyle swimming could compromise optimal body position and stroke mechanics. This conclusion is supported by the results of this study which indicate that there is a strong correlation between improvements in distance
per breath and distance per stroke, and previous research by Craig & Pendergast (1979) and Craig et al. (1985) that reported correlation between increases in distance per stroke and increased swimming velocity. Harms et al. (1997) provide additional research that supports the importance of decreasing hyperpnea to improve exercise performance. They concluded that the work of breathing normally incurred during maximal exercise is associated with a vasoconstriction in the locomotor muscles which may compromise muscle perfusion, and thus, the VO₂ of the working muscles.

Another view is that the entrainment of breathing during exercise is a primary determinant of the ventilatory pattern during exercise, one that can influence exercise performance. Mahler et al. (1991) conducted an examination of the impact of entrainment of breathing on performance during rowing. They suggested that both cardiorespiratory fitness and proficiency in mechanics are requirements for success in competitive rowing, and that entrainment of breathing may be necessary to produce optimal power during the drive phase of the stroke. Further, they speculated that the coupling of respiration with the mechanics of the rowing stroke may be developed through years of training, and that limb movement frequency may be a determinant of the breathing pattern. Elite rowers tended to use a greater tidal volume and a lower respiratory frequency to augment exercise ventilation than collegiate rowers did. Breathing in swimming is also entrained to the stroke rate, and thus may influence exercise performance in a similar fashion to rowing. The research discussed suggests that both technical and physiological benefits of aligning the breathing pattern with the exercise rhythm of the exercising limbs produces improved exercise performance.

In the current study, the observed reduction in breathing frequency and improvement in distance per breath are factors that can account for the improved critical and peak velocity that occurred during the swimming season (see Figure 14). This conclusion is supported by the results of the correlation analysis which demonstrated a significant correlation between improvements in chemoreflex frequency sensitivity and critical velocity, and between improved distance per breath and increased distance per stroke. Previous research suggests that the decreased work of breathing and improved
technical efficiency that result from reduced breathing frequency and increased distance per breath would be contributing factors toward improvements in velocity. The mechanism through which the swimmers were able to restrict exercise hyperpnea remains unclear. The results of this project indicate that this mechanism is an attenuation of the peripheral frequency sensitivity control of breathing.

- Decreased chemoreflex frequency sensitivity
  - Decreased urge to breathe.
- Increased distance per breath and decreased breathing frequency on swim tests
  - Increased breathing efficiency reduced restrictions to optimal swimming technique
  - Improved technical efficiency
  - Increased swimming speed on progressive and maximal tests

Figure 14. Summary of swimming and rebreathing discussion
Rebreathing Test Results

The Chemoreflex Response to Partial Pressure of Carbon Dioxide

This study demonstrated that swim training resulted in an increase in chemoreflex threshold and decrease in chemoreflex ventilation sensitivity. The decrease in ventilation that was observed during the CO₂ rebreathing test after training resulted from a decreased frequency sensitivity and not changes in the tidal volume response. There was a significant correlation (p=0.03) between improvements in chemoreflex frequency sensitivity and chemoreflex ventilation sensitivity. The results of the rebreathing test suggest that alterations in the athlete’s chemoreflex response may have allowed them to restrict the hyperpnea of exercise, which thus improved the technical characteristics of their stroke, and enabled them to swim faster and more efficiently. It is interesting to note that in order for the swimmers to improve their distance per breath on the swimming test, they would have had to breathe less frequently; the results of the present study confirmed this. The frequency sensitivity was significantly decreased after training, and the distance per breath was significantly increased. These results were obtained under hypoxic conditions. On the other hand, the results of the rebreathing test in hyperoxia did not show any significant changes in the chemoreflex characteristics of the athletes after training, nor were there any significant differences between athletes and non-athletes. Therefore, any adaptations that occurred with training were the result of changes in at the peripheral chemoreflexes, and not in the central chemoreflexes as the earlier literature review suggested would be the case.

The rationale for exploring the association between the chemoreflex response and swimming efficiency / performance may be explained as follows: during swimming, the time available for ventilatory exchange is dictated by the stroke rate, that is, breathing is entrained to the limb movements. At faster stoke rates (usually indicating a higher intensity), it would be expected that the drive to breathe would be greatly increased, yet the time interval for the ventilatory exchange to take place is decreased. The issue then is
a simple one – how is adequate exchange ensured, i.e., what adaptive strategy is required to meet this challenge?

The physiological explanation whereby improvements in the technical characteristics of the stroke are related to changes in ventilation concerns an increased chemoreflex threshold and a decreased sensitivity to increased levels of arterial carbon dioxide (McConnell et al., 1996). These changes allow the athletes to adopt the optimal breathing pattern given the demands of the exercise at a given level of work. Mercier et al. (1991) showed that the chemoreflex response to CO₂ could be the driver of breathing pattern in terms of the response to the CO₂ stimulus. During the breath holding and reduced ventilation that are characteristic of competitive swimming, an urge to breathe is developed (Harty et al. 1996), which may arise from the increased levels of CO₂ and the increased concentration of hydrogen ions that are produced as a result of lactic acidosis (Mateika and Duffin, 1994). An increased ventilation would allow the subjects to tolerate an increased level of CO₂ and to control changes in pH (Harty et al., 1996). A reduced hyperpnea would allow for an improvement in biomechanical efficiency (Mahler et al., 1991). Thus, changes in the chemoreflex threshold and sensitivity are important determinants of an athlete’s ability to restrict hyperpnea, to achieve or maintain proper stroke technique, and ultimately, to achieve optimal performance.

Physiological factors that occur under exercise conditions may stimulate an increased ventilation. Endurance training, of the type performed by the participants in the current research, has been shown to induce a reduction in the ventilatory response to a given level of work (Taylor, 1979). Results of the present study indicated that adaptations to training may stimulate changes in the carbon dioxide threshold and sensitivity, which, in turn, allow alterations in the ventilatory characteristics of the athletes during exercise.

One potential physiological explanation for the observed changes in chemoreflex response is an alteration in the buffering capacity of the body, which over the long term produces a compensated metabolic acidosis (Duffin, 1990). This, in turn, may lead to a decrease in the chemoreflex sensitivity to CO₂, as was observed in the present study.
Research about additional issues may help illuminate the interactions between exercise performance, adaptations to training, and ventilatory control. Due to the mechanics of swimming, athletes must employ one of several breathing patterns (i.e., breathing every "x" number of strokes) to provide ventilatory exchange during exercise. Coaches alter these patterns to increase the training stress, a technique termed controlled frequency breathing (Sharp, 1991). Controlled frequency breathing has been shown to increase the [H+] and the arterial levels of PCO₂ relative to the levels normally found in exercise, while VO₂ remains unchanged (Sharp, 1991). As the PCO₂ has been demonstrated to drive the central chemoreceptors, [H+] to stimulate the peripheral chemoreflexes, and a lowered PO₂ to serve as a hypoxic stimulus to the peripheral chemoreflex drive when the metabolic utilization of O₂ remains unchanged (Duffin and McAvoy, 1988), it can be inferred that aerobic metabolism was not sufficiently altered to cause the stress required for specific overloading of the metabolic system. Controlled frequency breathing may stimulate blood and muscle buffer adaptations through a consistently increased [H+] over a training cycle (Sharp, 1991), and in turn, the altered metabolic buffering may serve to decrease the ventilatory response of the chemoreflexes (Duffin, 1990). Melissa et al. (1997) recently demonstrated that the combination of exercise training and a moderate hypoxic environment results in an enhanced physiological adaptation, as evidenced by increased levels of the enzymes citrate synthase, succinate dehydrogenase, and phosphofructokinase, as well as an increased time to fatigue compared to the results from hypoxia alone.

In summary, during hypoxia, the mean chemoreflex threshold was increased and the mean chemoreflex sensitivity was decreased for the athlete group over the swim training cycle. These changes were accompanied by a non-significant increase in distance per stroke and a significant increase in distance per breath over the same period. Since improvements in stroke technique can be made as a result of the improved distance per stroke and breathing economy, which in turn are facilitated by a decreased drive to breathe and hyperpnea, it can be concluded that the observed changes in chemoreflex threshold and sensitivity contributed to the improvement in swimming test results.
CHAPTER 5: SUMMARY, IMPLICATIONS, AND DIRECTIONS FOR FUTURE RESEARCH

Summary

Results of this study indicated that competitive swim training allows athletes to swim at faster velocities through improvements in technical efficiency. These improvements appear to result from an improved breathing economy, which is facilitated by a reduction in breathing frequency and an increased distance per breath. Furthermore, the physiological mechanism that allows for the improvement in the breathing economy variables is an attenuation of the peripheral chemoreflex response, specifically, an increase in the chemoreflex ventilation threshold and a reduction in the chemoreflex ventilation sensitivity. That the decreased sensitivity was achieved through a concurrent decrease in the frequency sensitivity, and not through changes in the tidal volume sensitivity, is further evidence that alterations in breathing economy during exercise are mediated by changes in the peripheral chemoreflex response. The primary limitation of this study was that the rebreathing test safety protocols limited our ability to measure the second chemoreflex threshold and sensitivity characteristics. Further, the athlete subjects were drawn from 6 swimming clubs, therefore the swim training intervention may have varied considerably from subject to subject. Also, cognitive and psychological adaptation that may have influenced the participant’s ability to control their breathing was not measured. This places limitations on our ability to suggest that one specific type of swim training was responsible for the changes that were seen in our results.

The main results of this study were:
1. the critical velocity was increased after training.
2. the peak velocity on the maximal 150 metre performance test was increased after training.
3. the distance per breath on the maximal 150 metre performance test was increased after training.
4. the first chemoreflex threshold in hypoxia was increased in the athlete group after training.
5. the first chemoreflex sensitivity in hypoxia was decreased in the athlete group after training.
6. the first frequency sensitivity in hypoxia was decreased in the athlete group after training.
7. there were no changes in the chemoreflex response for the athlete group in hyperoxia.
8. there were no changes in chemoreflex response for the non-athlete group in either hypoxia or hyperoxia.

Implications

The implications of this research are two-fold; they relate to the application of the research in the applied sport science field. The first major implication concerns the area of competitive swimming training. The study results suggest that coaches should place more emphasis on measures of breathing economy. Increasing distance per breath may serve to facilitate improvements in performance to a greater degree than the traditional measure, that is, distance per stroke. Thus, the concept and importance of breathing economy should be publicized and explained more fully to the coaching community.

The next implication of these results is the need to refine the model for the analysis of swimming economy variables during progressive swimming tests as was proposed earlier in this paper. A model of this type would allow for more sensitive testing of the technical progress of the athletes, and could be easily integrated with the popular critical velocity physiological model that is currently in use with the Canadian and Australian national programs.
Directions for Future Research

The results of the current study suggest several possibilities for future investigation:

1. Further research should be conducted on the changes in the peripheral chemoreflex response and the resultant effect on performance. Specifically, alterations in the chemoreflex response with controlled frequency breathing, altitude training, high intensity anaerobic training, lower intensity endurance training, and specific training of the respiratory muscles should be investigated.

2. A neural drive to breathing may become significant during exercise (Duffin, 1990). Rapid changes in ventilation occur at the beginning and termination of exercise that are not related to chemical changes. Duffin (1994) hypothesized that these changes are accounted for by a fast neural drive whose magnitude is related to the frequency of limb movement. Further, Duffin (1994) suggested that ventilation after the first threshold in heavy exercise is due to a heavy exercise neural drive that is determined by the motor commands of exercising muscle. Therefore, future research should be conducted to examine the effect of exercise training on neural control of ventilation.

3. Investigation is required to specifically determine the training factors which may have accounted for the observed changes in chemoreflex variables. Specifically, were the changes induced by alterations in the input to the chemoreceptors perhaps via an increased buffer capacity, or through a change in the chemoreceptors themselves that would result in a reduced neural output.

4. Research is required to determine the effect of training on the respiratory muscles and if training of these muscles could account for the adaptations that were observed in the current research.

5. The models of swimming economy and breathing economy during progressive intensity tests that were used in this study need to be refined and validated.

6. The specific mechanism by which the attenuation in the peripheral chemoreflex response was achieved should be identified. Possibilities include changes in
buffering capacity of the blood and/or respiratory muscles, a change in potassium metabolism with training, or some other as yet unidentified factor.
REFERENCES


Appendix A – Human Ethics Submission and Approval

CHANGES IN THE CHEMOREFLEX RESPONSE AND PERFORMANCE MEASURES WITH TRAINING IN COMPETITIVE SWIMMERS

Introduction

One of the requirements for training and performance in sports, such as running, cycling, speed skating, and rowing is the necessity of developing the ability to control breathing under stressful circumstances. A close relationship exists between breathing rate and cycle rate, that is, breathing is often entrained to limb cycle rate. In addition, each athlete has an optimal cycle rate for peak muscular efficiency.

The foregoing is also true for the sport of swimming. However, the complicating factor in swimming is that the athlete is not able to breathe on demand, but must wait for that portion of the stroke cycle that allows breathing to occur. Thus, the question arises as to the nature of the interaction of the breathing rate and the cycling rate during swimming, that is, does the drive to breathe influence the arm cycling rate, and therefore potentially alter the technical aspects of the skill (i.e., cycle rate and cycle length). On the other hand, changes in stroke length and cycling rate occur during training, which suggests that changes to the drive to breathe must also occur during the training process.

Recently, research on the control of breathing has focused on the role of the central and peripheral chemoreceptors. Few studies have evaluated the alterations in the control of breathing during a period of training. The purposes of this study, therefore, are as follows. The first is to assess the difference between athletes and non-athletes in their ventilatory response to carbon dioxide. The second is to evaluate the effect of training on the ventilatory response to carbon dioxide in light of alterations in stroke mechanics, critical velocity, and anaerobic power.
We hope that the results will contribute to our understanding of the control of breathing in exercise and improve the future performances of athletes. Further, the knowledge gained from the study may assist the coaches and athletes in the design, planning and implementation of future training programs. Also, information gained in this study may, in the future, allow those athletes with low/high ventilatory responses to carbon dioxide to be identified and then placed in the appropriate distance whereby this natural ability would enhance the athlete's potential performance. The benefits to the non-athlete group may be that, following the research, they will be provided with an exercise program based upon the results of the PWC170 test.

Review of Literature

The Case for an Attenuated Chemoreceptor Response in Trained vs. Untrained Subjects.

Miyamura et al. (1976) presented supporting evidence for an attenuated ventilatory response in athletes. These investigators examined the differences between untrained subjects and marathon runners with regard to the ventilatory response to carbon dioxide rebreathing both at rest and during steady-state, sub-maximal exercise. The rebreathing technique described by Read (1967) was used to measure the ventilatory response to carbon dioxide. The mean slope of the ventilation vs. partial pressure of carbon dioxide curve at rest was 1.86 L·(min mmHg)^{-1} for the controls vs. 1.12 L·(min mmHg)^{-1} for the athletes. During exercise, the slopes were 1.20 L·(min mmHg)^{-1} for the controls vs. 0.62 L·(min mmHg)^{-1} for the athletes. The differences were significant under both conditions. Thus, the average slopes of the response curves were lower for the athletes than for the untrained group, both at rest and during exercise. This study demonstrated differences in response between the groups, but unfortunately did not distinguish between the contribution played by the peripheral and the central chemoreceptors since the level of oxygen maintained during the rebreathing was not stated. In addition, the study also leaves unanswered the question of whether the neural
factors had any influence on the differences observed between groups. Lastly, a note of caution must be placed on the exercise findings of this study in that the rebreathing technique does not work effectively under exercise conditions.

McGurk et al. (1995) recently attempted to establish a relationship between the ventilatory sensitivity to carbon dioxide and sprint vs. endurance performance measures in young swimmers. The subjects were matched into two groups of 17 high responders and 17 low responders to the hyperoxic carbon dioxide rebreathing test at rest. The rebreathing test, a version of Read's method modified by Rebuck (1972), used 7% carbon dioxide and 93% oxygen. They compared the responses to carbon dioxide rebreathing and the results of two sprint and two endurance performance tests. The results indicated that the low responders had a significantly faster 1.6-km run time than the high responders, and that the high responders recorded significantly better results on the 10-alactic power test. Unfortunately, as the rebreathing test was conducted under hyperoxic conditions, only the response of the central chemoreflex drive could be assessed. Also, the performance measures used to evaluate the swimmers were not sport specific, and therefore, may not accurately reflect the actual abilities of the swimmers.

Godfrey et al. (1971) used Read's rebreathing technique (1967) to compare 7 athlete's and 7 control subject's ventilatory responses to hypercapnia under hyperoxic conditions (initial gas mixture in bag: 50 mmHg carbon dioxide, 150 mmHg oxygen) at rest. They found a slightly higher slope in the carbon dioxide rebreathing response curve [2.36 vs. 2.05 L-(min mmHg)^{-1}] in the athletes and control subjects. Again, because the rebreathing was done under hyperoxic conditions, only the central chemoreflex ventilatory response to carbon dioxide was assessed.

Mahler et al. (1982) examined the ventilatory responses to hypercapnia and its possible role in athletic performance by comparing 20 male marathon runners to 20 sedentary subjects of the same age. They used a modified version of Read's rebreathing technique (1967) with a gas mixture of 7% carbon dioxide in 93% oxygen at rest. Their
results indicated a reduced ventilatory drive in response to hypercapnia in the runners [2.23 vs. 2.61 L·(min mmHg)^{-1}] at rest. Although the authors reported a poor correlation between chemosensitivity and endurance performance, the rebreathing technique employed in this study assessed only the chemosensitivity of the central chemoreceptors, not that of the peripheral chemoreceptors.

Heigenhauser et al. (1983) examined the ventilatory response to carbon dioxide during rebreathing in 8 synchronized swimmers, 8 competitive swimmers, and 8 recreational swimmers at rest. The synchronized swimmers participated regularly in training that involved prolonged periods of breath holding, and the competitive swimmers trained at high exercise intensities to increase their aerobic and glycolytic capacities. Both groups had been training for at least 3 years. The rebreathing technique was performed according to Read's method (1967), with a gas concentration of 7% carbon dioxide and 93% oxygen. During rebreathing, the slope of the ventilatory response to carbon dioxide curve was lower for synchronized swimmers [1.48 L·(min mmHg)^{-1}] than for either the competitive swimmers [2.04 L·(min mmHg)^{-1}], or the recreational swimmers [1.87 L·(min mmHg)^{-1}]. An interesting component of the research was that during arm cranking exercise, ventilation at a volume of 1.0 L·min^{-1} carbon dioxide was significantly higher in the recreational swimmers than the synchronized and competitive swimmers.

Blum et al. (1979) used the hyperoxic (Read) rebreathing technique to examine the central chemoreflex response to carbon dioxide before and after training. After training, they found a decrease in the ventilatory response to carbon dioxide in five normal subjects. The length of training and initial fitness levels were not stated, therefore, the implication and interpretation of the results lack some clarity.
The Interaction Between the Control of Breathing and Optimal Performance

In some athletic activities, such as swimming, cycling, running, and rowing, it may be desirable to control the hyperpnea of exercise. A number of studies have reported a correlation between the magnitude of the ventilatory response to exercise, the increased breathing during exercise, and the ventilatory sensitivity to carbon dioxide. Specifically, Martin et al. (1978) found that the hypoxic and hypercapnic ventilatory responses during light and heavy exercise were correlated in athletes. McConnell et al. (1992) noted a stronger correlation between the ventilatory response to exercise and carbon dioxide sensitivity in exercise than in rest. This evidence may support investigations of the role of ventilatory sensitivity to carbon dioxide in determining the magnitude of exercise hyperpnea.

Some authors hypothesize that in endurance events, it may be beneficial to conserve energy by restraining hyperpnea and allowing the partial pressure of carbon dioxide to rise, and that such a strategy would be unnecessary in sprint events. If this is indeed the case then it may be expected that the sensitivity to carbon dioxide would be lower in successful endurance athletes than in sprinters or untrained subjects (McConnell & Semple, 1996).

One potential physiological explanation for improvements in cycle length in a sport such as swimming concerns an altered chemoreflex response (McConnell et al., 1996). Craig and Pendergast (1979) examined the relationship between swimming velocity, stroke cycle rate, and stroke cycle length; they reported that the fastest swimmers had the longest cycle length at sub-maximal velocities, and that there was a positive correlation between maximal cycle length and maximal velocity.

When exercising intensely, athletes encounter increased ventilation accompanied by lactic acid accumulation, metabolic acidosis, increased metabolic production of carbon dioxide, and increased potassium concentrations (Mateika & Duffin, 1995). These factors may partially account (~15-20%) for the increased ventilation through their actions upon
the peripheral chemoreflexes (Cunningham, 1987). This increased drive to breathe decreases an athlete's ability to maintain an optimal cycle length due to the extended time between breaths. Thus, each athlete could have an optimal combination of cycle length and cycle rate, which may involve considerations of local muscle power, endurance, and an attenuated chemoreflex response leading to decreased exercise hyperpnea. The reduced exercise ventilation would be of benefit to athletes who need to control breathing patterns and maintain an optimal cycle length. Wakayoshi et al. (1995) presented more supporting evidence for the potential relationship of velocity, cycle length, and cycle rate; they found that high performance swimmers had a greater cycle length and a lower cycle rate than lower performance swimmers at a given velocity.

**Summary – Literature Review and Objectives of Study**

Research has demonstrated that ventilation at a given work load is attenuated after training. The literature has also presented convincing evidence that the peripheral and central chemoreceptors act as feedback regulators in the control breathing. The research would also suggest that the peripheral chemoreceptors would be expected to contribute to exercise hyperpnea, while the central chemoreceptors would not be involved. Unfortunately, studies to date have only examined the central chemoreceptors. As discussed earlier in this review, the central chemoreceptors are not expected to be affected by training. To date, there has not been any research concerning the effect of training on the peripheral chemoreflex response to carbon dioxide.

Therefore, the questions concerning differences between athletes and non-athletes, and regarding the trainability of the peripheral and central chemoreflex responses need to be resolved. Further investigation is also needed to differentiate between the relative contributions of the peripheral and central chemoreflexes to the control of breathing in exercise and at rest, and to improve our understanding of the interaction between the control of breathing and optimal performance.

The objectives of this study are:
1. to determine the differences between athletes and non-athletes in terms of their ventilatory responses to carbon dioxide (peripheral and central chemoreflex).

2. to determine the effect of training athletes on their ventilatory response to carbon dioxide (peripheral and central chemoreflex) in light of alterations in stroke mechanics, critical velocity, and anaerobic power.

Methods

Design

A comparative descriptive, correlational design (Burns and Grove, 1997) will be used to answer the following research questions:

1. Is there a difference between athletes and non-athletes in terms of their ventilatory responses to carbon dioxide?

2. Does training have an effect on the ventilatory response to carbon dioxide in athletes vs. non-athletes who do not train?

3. Does training have an effect on critical velocity (Treffene, 1985), anaerobic power, and distance per stroke of swimmers?

4. Is there a relationship between changes in ventilatory response to carbon dioxide and performance measures (critical velocity, anaerobic power, and distance per stroke) of swimmers?

Definition of Variables and Outcome Measures

Ventilatory response to carbon dioxide is defined in terms of two variables. These variables are:

1. Peripheral chemoreflex threshold and sensitivity.

2. Central chemoreflex threshold and sensitivity.

These variables are measured in a rebreathing test (Mohan & Duffin, 1997) and are plotted graphically as ventilation vs. partial pressure of carbon dioxide.

There are three performance variables that are used to assess physiological and technical characteristics of swimmers. These variables are:
1. Critical velocity.
2. Stroke length.
3. Anaerobic power.

Critical velocity as defined by Treffene (1982) is plotted as swimming velocity vs. heart rate. Stroke length is defined as velocity vs. distance per stroke (Craig and Pendergast, 1980). Anaerobic threshold is described by plotting velocity vs. blood lactate concentration (Olbrecht et al., 1985).

**Sample and Sampling Procedures**

This study will be conducted in the Greater Toronto Area. Both athlete and non-athlete participants will be recruited. Potential athlete participants will be recruited from 10 national level competitive swimming teams. The researcher will recruit 24 athletes to participate in the study (see Appendix A for statistical power data concerning sample size). The criteria for inclusion of athletes into the study will be:

a. healthy male and female volunteers,
b. post-pubescent swimmers between the ages of 13-22 years of age,
c. members of competitive swimming teams,
d. have achieved a time within 5% of national qualification standards or better,

A notice of the study will be sent to the head swim coaches of the top 10 teams in the Greater Toronto Area that are most highly ranked in Canada to inform them of the research and to solicit their assistance in the recruitment of potential participants. Potential participants that are National Championship calibre athletes in these swim clubs range in age from 13 to 22 years of age. The large participant pool (200 athletes in the National groups of these teams) will allow for the ages of the participants to be spread across the range of ages. It is necessary to use participants of this age group as the majority of high performance swimmers in the Toronto are under the age of 18. It is unclear whether the adaptation is progressive and what the time course of adaptation might be. Therefore, younger swimmers with less training history are needed to
determine the possible differences in trainability and/or training status of this variable. The researcher will use equal numbers of male and female participants. Research has indicated that post-pubescent male and females should perform similarly in the rebreathing test as long as the females are tested in the mid-follicular phase of their menstrual cycles and participants are post-pubescent (McGurk et al., 1995). The head coaches will be asked only to announce the research to swimmers at a team meeting and to parents at a parents’ meeting using a standardized letter of introduction to the study (see Appendix B). The head coaches will be also be asked to post a copy of the letter of introduction at the pools in a site visible to both swimmers and their parents. In order to prevent a conflict of interest from arising between these swim coaches and the competitive swimmers, any detailed explanation of the study to swimmer and parents / guardians will occur between the researcher and swimmers, and parents / guardians. For those swimmers and parents who demonstrate interest in the study, the head coach will request permission to forward their names and phone numbers to the investigator, Mr. Greg Wells. Mr. Wells will then contact potential participants to arrange a meeting at a time and place convenient to the participants in order to explain the study in further detail (see Appendix C). If the swimmers and parents agree to participate in the study, written informed consent will be obtained concerning their participation (see Appendix D). An agreed upon time will be established with the participants to conduct the study.

Also recruited will be 24 non-athletes for the comparison group. The inclusion criteria for these participants will be:

a. healthy male and female volunteers,

b. post-pubescent non-athletes between the ages of 13-22 years of age.

c. These participants will not have been involved in regular training programs over the previous 12 months. Regular training programs have been defined as not exercising more than 3 times per week for no more than 40 minutes at a heart rate in the training zone (exercise at the person’s maximum heart rate minus 60 beats per minute or
above). Exercising 3 times per week for 30-40 minutes at a heart rate in the training zone (exercise at the person’s maximum heart rate minus 60 beats per minute or above) is the minimum exercise program needed to develop fitness. This definition has been developed by the American College of Sports Medicine, and it has been supported by the Canadian Society for Exercise Physiology.

The non-athlete participants will be recruited through advertisements at community centres in the Greater Toronto Area (see Appendix E). The inclusion criteria will be stated in the advertisement. Mr. Wells will then contact potential participants, who demonstrate interest in the study by contacting Mr. Wells through the phone number provided in the advertisement. A meeting at a time and place convenient to the participants in order to explain the study in further detail (see Appendix F). If the potential participants and parents agree to participate in the study, written informed consent will be obtained concerning their participation (see Appendix G). An agreed upon time will be established with the participants to conduct the study.

Athlete and non-athlete participants will be matched for age and gender.

Data Collection Procedures

For both groups, descriptive data will be collected concerning date of birth and history of competitive swimming for the athletes and history of exercise for the non-athletes, gender, and baseline fitness level (PWC170 test). Also, data will be collected from both groups regarding the rebreathing test. The swimming tests will be conducted for athletes only.

Rebreathing protocol. Prior to beginning the data collection, each participant will be asked to attend an habituation session to familiarize the participant with the technique and apparatus. During the laboratory data collection, each participant will complete 2 rebreathing tests at constant end-tidal partial pressures of oxygen at 50 mmHg and one at 150 mmHg. A hypoxic level of 50 mmHg was chosen to allow for the measurement of
the peripheral chemoreflex response and the central chemoreflex response. The single test at 150 mmHg oxygen will allow for the isolation of the central chemoreflex. The review of the literature has indicated that a major gap in the knowledge in this area is the lack of research investigating the differences between athletes and non-athletes in their peripheral chemoreflex response and the trainability of the peripheral chemoreflex response. Measuring both these variables will allow the researchers to investigate the relative contributions of both variables to the ventilatory response to carbon dioxide. Each rebreathing test session will last approximately 120 minutes. No more than three 10-minute tests will be conducted in a given session, with each assessment being separated by a rest period of at least 30 minutes. The participants will be asked to refrain from drinking any caffeinated beverages for 2 hours prior to testing.

The rebreathing test used in this experiment was first proposed by Read (1967) and subsequently modified (Duffin and McAvoy, 1988). The test includes a prior voluntary hyperventilation to lower carbon dioxide to sub-threshold levels, along with computer control of the supply of oxygen to ensure iso-oxic conditions during the rebreathing. These modifications allow the determination of the thresholds and sensitivities of the central and peripheral components of the ventilatory response to carbon dioxide at specific levels of hypoxia.

The apparatus used for the rebreathing tests has been previously used in this laboratory (Duffin and McAvoy, 1988), with the addition of a computer controlled feedback system to maintain iso-oxic conditions at 50 mmHg or 150 mmHg of oxygen throughout the experiment. The entire apparatus is calibrated before each session.

An oximeter probe (Bruel and Kjaer, model 8852) is placed on the participant's index finger in order to monitor heart rate and oxygen saturation. The participant will wear a nose clip throughout the experiment, and will breathe via a mouthpiece connected to a Y value (Collins P-319; 80 ml dead space). This valve allows the participant to switch themselves from room air to the rebreathing bag. A tube attached to the valve will
sample the air breathed at the mouth and the end-tidal values of carbon dioxide and oxygen will be closely monitored (Bruel and Kjaer, anaesthetic monitor 8852). The rebreathing bag, approximately 5 litres, is enclosed in a rigid container and is connected to a dry rolling real seal spirometer (Morgan Spiroflow, model 130) by a short length of wide bore (37 mm) tubing to allow monitoring of ventilation on a breath by breath basis. The bag is filled with a gas mixture in which the partial pressure of carbon dioxide is set at 40 mmHg, the partial pressure of oxygen is established at 50 mmHg or 150 mmHg and the remainder of the volume is nitrogen.

The participant will be asked to hyperventilate for 5 minutes with room through the Y valve in order to lower the end-tidal partial pressure of carbon dioxide to a level between 20-24 mmHg. The participant is switched to the rebreathing bag, and asked to take 3 deep breaths to ensure that the end-tidal pressure of carbon dioxide and oxygen in the bag, lungs, and arterial blood quickly equilibrate with the mixed venous partial pressure, which serves as an estimate of the partial pressure in the large tissues. A proper plateau in the end-tidal partial pressure of carbon dioxide will evidence adequate equilibration. The participant will then rebreathe under iso-oxic conditions maintained by a computer controlled feedback system at 50 mmHg or 150 mmHg of oxygen.

The end-tidal partial pressure of carbon dioxide increases linearly with time, with the test being terminated at a level of 60 mmHg. At this time, the participant is switched back to room air, and remains seated during recovery for several minutes.

Swimming test protocol. The test will be conducted in a long course (50 metre) pool. Prior to each 5 x 200 test, the foot length, arm length and height of each participant is measured and recorded. (Grimston & Hay, 1986) The anthropometric data will be used to interpret the changes in distance per stroke of the participants.

Each test will consist of a set of 5 x 200 metre swims on a pace time of 6 minutes. The researcher will calculate the required speed for each 200 metre swim prior to the test and the participants will be informed of these target speeds before the test begins. Each
target speed will be based upon percentage of the participant's best time, which in turn, corresponds to a percentage of maximum heart rate. The participants will swim the 5 x 200 set using their best competitive stroke.

The first 200 metre swim is to be swum at a speed that will result in a target heart rate of 140 (± 10) beats per minute (bpm). The second 200 will be swum at a speed that will result in a target heart rate of 150 (± 10) bpm. The third 200 will be swum at a speed that will result in a target heart rate of 160 (± 10) bpm. The fourth 200 will be swum at a speed that will result in a heart rate of target 170 (± 10) bpm. The fifth 200 will be swum at a speed that will result in a heart rate of >180 (± 10) bpm. The final time and heart rate for each 200 metre swim will be measured and recorded. The 50 metre splits within each 200 metre swim will also be measured and recorded. The heart rates of the participants will be measured using a heart rate monitor and will be recorded after each 200 metre swim. The number of strokes taken on the third length of each 200 metre swim will be counted and recorded. Three minutes after completing each 200 metre swim, a venous blood sample will be collected in capillary tubes after a finger stick and pipetted into an automated lactate analyzer (Yellow Springs Instrument model 23A) for the determination of whole blood lactate.

The data from each 5 x 200 test will be analyzed graphically to determine critical velocity, anaerobic threshold, and distance per stroke.

**PWC\textsubscript{170} test protocol.** The sub-maximal exercise test will be conducted at the University of Toronto. A Monark Bicycle Ergometer will be utilized for this test. The test involves riding a bicycle ergometer at progressively heavier workloads. The system will be set so that one complete turn of the pedal moves a point on the rim of the wheel 6 metres (Astrand and Rodahl, 1977). A metronome will be set for 50 revolutions per minute. The weight put in the basket of the cycle ergometer, multiplied by the distance pedaled (m) will give the amount of in kilogram-meters (kpm). The distance will be expressed per minute, and the rate of work will be expressed in kpm per minute.
The subjects will be given 3 minutes to warm-up and familiarize themselves with the cycle ergometer and prepare them for the exercise intensity in the first stage of the test. Heart rates will be taken during the final 15 seconds of the second and third minutes of each stage. The participants will then perform 3 to 4, 3 minute stages at progressive increments in work rate, all of which will remain sub-maximal. The heart rate measured during the last minute of each stage is plotted against work rate. The regression line generated from the plotted points is then extended to a heart rate at 170 beats per minute. Analysis of covariance will be used to determine differences between the groups.

Ethical Considerations

Informed consent. All information will be given to the participants in a manner that is understandable to them (see Appendices C and F). Care will be taken to explain the project, the procedures involved, and the goals and potentials outcome of the research. In the informed consent, it will be made clear to each potential participant, in lay terms, that they have complete freedom of choice to participate or not, and that they are free to withdraw from the study at any time, with no repercussions from the investigators or from their coaches (see Appendix D and G). A copy of the letter of explanation of the study and the signed consent form will be given to the participants.

Risks and benefits. Young, normal, healthy individuals can complete the proposed rebreathing protocol. The risks from the procedure include: dizziness, headaches, nausea, tightness of the chest, a sense of smothering, feelings of apprehension, heart palpitations, abdominal pains, faintness, or impaired consciousness. These symptoms have been reported for the Read rebreathing test (1967), but the modified version of the rebreathing test used in this method (Mohan & Duffin, 1997) uses lower partial pressures of carbon dioxide (35-60 mmHg vs. 45-70 mmHg PCO₂) and no adverse effects have been observed in over 150 tests in the respiratory laboratory under the supervision of Dr. Duffin at the University of Toronto. However, to ensure the safety of the participants, the
following precautions will be taken during the rebreathing tests: (a) the end-tidal volumes of oxygen and carbon dioxide will be monitored at all times; (b) the test is terminated if the carbon dioxide level rises above 60 mmHg (a normal level is 40 mmHg) or the oxygen level falls below 45 mmHg (a normal level is 100 mmHg); and, (c) the oxygen saturation is continuously monitored with a pulse oximeter, and is not allowed to fall below 75% (a normal level is 100%).

The swimming test (five 200 metre swims) can be completed by young, normal, healthy, competitive swimmers and is a normal experience for the participants. There are no known risks from completing the swimming test. Following each 200 metre swim, a 50 μL blood sample must be obtained. The risks associated with blood sampling include bruising and the possibility of infection. In order to eliminate the risks associated with blood sampling, the following precautions will be taken. First, any materials that could potentially come into contact with blood will be used only once and then immediately discarded into a biohazard waste disposal container, which will include all rubber gloves, finger sticks, capillary tubes, alcohol swabs and any other materials. Second, participants fingers will be thoroughly disinfected with alcohol swabs prior to the finger stick puncture and after the blood sample has been drawn. Participants will be provided with a Band-Aid for their fingers.

The PWC\textsubscript{170} test is a popular assessment technique for cardiorespiratory endurance that has been approved by the American College of Sports Medicine. All participants will complete the PAR-Q questionnaire prior to performing this test (see Appendix H). The risks associated with this test are muscle discomfort, and shortness of breath. The researcher will control for these risks by (a) monitoring heart rates at all times and terminating the test if the participant's heart rate reaches 85% of their age-predicted maximum heart rate (220 bpm-age) and (b) terminating the test if the participant experiences signs of excessive discomfort, or if the subject fails to conform to the exercise protocol.
Maintaining anonymity and confidentiality. The investigators will keep all records. Each participant will be recorded only via an individualized code. Only group result data will be published. All records will be kept in a secure location at the University of Toronto, and all data will be encoded so as to protect the names of the participants. Participants may request to view their own data, and this information will be provided for them.

Data Analysis

The data (heart rate, lactate and number of strokes) from each 5 x 200 test will be analyzed graphically to determine critical velocity, lactate threshold, and distance per stroke, respectively. The threshold and sensitivity of the central and peripheral chemoreflexes are determined through graphical analysis of the rebreathing test data.

Statistical analyses of the data include (a) the data from the swimmers will be assessed by a two-way repeated measures ANOVA (b) the data from the swimmers and the non-athletes will be examined via a two-way ANOVA and (c) the data from the non-athletes (comparison group) will be evaluated by a paired t-test to indicate stability of the measures. In each case, a p-value of <.05 will be used to indicate significance, and a Duncan's post-hoc test will used to identify differences between specific subgroups following the ANOVA.
STATISTICAL ANALYSIS

Project Title: Changes in the Chemoreflex Response with Training in Competitive Swimmers and Non-Athletes

Investigators: Greg Wells, Master of Science Candidate, Graduate Department of Exercise Science
Dr. Michael Plyley, Faculty of Physical Education and Health, University of Toronto
Dr. James Duffin, Department of Physiology, University of Toronto

Based upon the results of a pilot study that are presented in the table below, statistical analysis indicated that a sample size of 17 subjects will be required to achieve statistical power. We have chosen to examine 24 subjects due to possibilities of subject withdrawal or other unforeseen problems.

DPS indicates stroke length, Tp+c indicates peripheral chemoreflex threshold, Sp+c indicates peripheral and central chemoreflex sensitivity.
Dear Swimmers and Parents / Guardians,

Greg Wells, a Master of Science student in the Graduate Program in Exercise Science at the University of Toronto is conducting a study about the effect of the intense exercise involved with competitive swimming training on the urge to breathe in swimmers as part of his thesis requirements. Each swimmer will be involved in one swim session lasting approximately 2 hours in order to evaluate the swimmer's fitness and technique using various measures, such as their heart rate, stroke length, etc. Each swimmer will have their fitness assessed during a short 10-minute test on a stationary exercise cycle. As well, 3 - 10 minute breathing tests will be conducted with a minimum of 30 minutes between each test to assess the swimmer's breathing response. The total time for the breathing tests and rest periods will be approximately 2 hours. Both the swimming and breathing tests will be conducted at the University of Toronto within 7 days of each other.

Dr. Michael Plyley, University of Toronto Faculty of Physical Education and Health and Dr. James Duffin, University of Toronto Department of Physiology are supervising Greg. Dr. Plyley can be reached at 978-8563, Dr. Duffin can be reached at 978-6379.

Permission to conduct the study has been granted from the Office of Research Services at the University of Toronto (protocol number 3670). If you are interested in hearing more about the study, could you please provide your name and phone number to the Head Coach of your Swim Club. With your permission, the Head Coach will provide your name and phone number to Greg Wells who will then contact you to describe the research in detail.

Thank you for your consideration.

Swimmer's name: ________________________________
Parent (or guardian's) name: ________________________________
Telephone number: ________________________________
Best time to contact you: ________________________________
DETAILED LETTER OF EXPLANATION OF THE STUDY –  
COMPETITIVE SWIMMERS AND THEIR PARENTS / GUARDIANS

Date: __________________________

Dear __________________________ (swimmer; parents / guardians),

I am a Master of Science student in the Graduate Program in Exercise Science at the University of Toronto. As part of my Master’s thesis I am conducting a study about the effect of the intense exercise involved with competitive swimming training on the urge to breathe in swimmers. The study involves comparing the breathing responses of swimmers and non-athletes. Dr. Michael Plyley, University of Toronto School of Physical Education and Health and Dr. James Duffin, University of Toronto Department of Physiology are supervising the study. Dr. Plyley can be reached at 978-8563, and Dr. Duffin can be reached at 978-6379. Permission to conduct the study has been granted from the Office of Research Services at the University of Toronto (protocol number 3670).

The study will be conducted over a total period of six months (one of short course or long course seasons). At the beginning of the study, the swimmers will be asked to complete a physical activity readiness questionnaire (PAR-Q). As well, we will be asking them for information about their date of birth, height, arm length, foot length, and competitive swimming history. Before the first swimming test, the participants will be asked to complete one 10-minute fitness test on a stationary exercise cycle.

On four separate occasions during the six months, we will ask the participants to come to the University of Toronto for testing. There are two aspects involved in the testing protocol; a swimming test and a breathing test. Both will be conducted within 7 days of each other at the University of Toronto. The swimming test will consist of one usual practice lasting approximately 2 hours during which the swimmer’s fitness and technique will be assessed. The assessment will include the determination of blood lactate levels by finger prick tests, measurement of heart rates using a heart rate monitor, and measurement of stroke counts and breathing patterns. The second aspect of the research involves assessing the swimmer’s breathing response. To this end, the swimmers will be asked to attend a habituation session to familiarize the swimmers to the breathing test and apparatus. On a separate occasion, the swimmers will complete 3 – 10 minute breathing
tests separated by a minimum of 30 minutes between each test to assess the swimmer's breathing response. The breathing test consists of approximately 5 minutes of deep breathing of room air (hyperventilation) followed by 4 minutes of breathing a mixture of low (but constant) level of oxygen and a gradually higher level of carbon dioxide by asking the swimmer to breathe into and out of a bag (rebreathing). The total time for the breathing test session will be approximately two hours.

We hope that the results will contribute to our understanding of the control of breathing in exercise and improve the future performances of athletes. Further, the knowledge gained from the study may assist the coaches and athletes in the design, planning and implementation of future training programs. Also, information gained in this study would, in the future, allow those athletes with low/high ventilatory responses to carbon dioxide to be identified and then placed in the appropriate distance whereby this natural ability would enhance the athlete's potential performance.

There may be certain risks associated with participation in the study. The risks are:
1. Bruising and infection of the fingers may occur as a result of the finger prick during the lactate test.
2. During the breathing test, the swimmer could experience dizziness, headaches, nausea, tightness of the chest, a sense of smothering, feelings of apprehension, heart palpitations, abdominal pains, faintness, or impaired consciousness.

The researcher, Greg Wells, will control the above risks. The measures are:
1. Finger bruising and infection will be prevented by adhering to proper technique (disinfecting and cleansing of the fingers before and after the finger prick) for finger pricking.
2. The risks of the breathing test will be prevented from arising during the hyperventilation phase of the breathing test by monitoring the expired level of carbon dioxide to prevent it from going too low (below 20 mmHg carbon dioxide, normal level is 40 mmHg) and during the rebreathing phase of the breathing test by monitoring the expired level of carbon dioxide and terminating the test if the level of carbon dioxide increases above 60 mmHg (normal level is 40 mmHg), and by monitoring the level of oxygen and terminating the test if the level of oxygen drops below 40 mm Hg (normal level is 100 mmHg). Further, oxygen saturation (level of
oxygen in the blood) will be monitored using a pulse oximeter (a clamp that gently fits over a finger) and the test will be terminated if the oxygen saturation drops below 60% (normal level is 100%). Nevertheless, if any of these symptoms arise despite the measures undertaken by the researchers, the test will immediately be terminated.

3. The researcher will control for the risks involved in the cycle ergometer fitness test by (a) monitoring heart rates at all times and terminating the test if the participant’s heart rate reaches 85% of their age-predicted maximum heart rate (220 bpm-age), (b) terminating the test if the participant experiences signs of excessive discomfort, or if the subject fails to conform to the exercise protocol.

Prior to conducting the study, I will obtain written, informed consent from the swimmers and parent / guardian (if the child is under 16 years of age). There is no obligation to participate in the study and the swimmer and parent / guardian (if the child is under 16 years of age) may withdraw from the study at any time without affecting the coaching that the swimmer may receive. All information collected during the study will not identify the swimmer and parent / guardian by name and all information obtained will be kept in a secure location and will be destroyed 7 years following the completion of the study.

Thank you for your interest in hearing about the study.

Sincerely,

Greg Wells
Master of Science Student
Graduate Department of Exercise Science
University of Toronto
Tel: (416) 573-2258
VOLUNTEER CONSENT FORM — COMPETITIVE SWIMMER

Project Title: Changes in the Chemoreflex Response with Training in Competitive Swimmers and Non-Athletes

Investigators: Greg Wells, Master of Science Candidate, Graduate Department of Exercise Science
Dr. Michael Plyley, Faculty of Physical Education and Health, University of Toronto
Dr. James Duffin, Department of Physiology, University of Toronto

Greg Wells, a Master of Science student in the Graduate Program in Exercise Science at the University of Toronto, is conducting a study about the effect of the intense exercise involved with competitive swimming training on the urge to breathe in swimmers. Dr. Michael Plyley, University of Toronto School of Physical Education and Health and Dr. James Duffin, University of Toronto Department of Physiology are supervising the study. Dr. Plyley can be reached at 978-8563, Dr. Duffin can be reached at 978-6379, and Greg Wells can be reached at 573-2258. Permission to conduct the study has been granted from the Office of Research Services at the University of Toronto (protocol number 3670).

The study will be conducted over a total period of six months (one of short course or long course seasons). At the beginning of the study, the swimmers will be asked to complete a physical activity readiness questionnaire. As well, we will be asking them for information about their date of birth and competitive swimming history.

On four separate occasions during the six months, we will ask the participants to come to the University of Toronto for testing. On the first occasion only, they will be asked to complete a short cycle ergometer fitness assessment. On each of the four occasions, the swimmers will have their height, arm length, foot length measured. There are two aspects involved in the testing protocol; a swimming test and a breathing test. Both will be conducted within 7 days of each other. The swimming test will consist of one usual practice lasting approximately 2 hours during which the swimmer’s fitness and technique will be assessed. The assessment will include the determination of blood lactate levels by finger prick tests, measurement of heart rates using a heart rate monitor, and measurement of stroke counts and breathing patterns. The second aspect of the research
involves assessing the swimmer’s breathing response. To this end, the swimmers will be asked to attend a habituation session to familiarize the swimmers to the breathing test and apparatus. On a separate occasion, the swimmers will complete 3 – 10 minute breathing tests separated by a minimum of 30 minutes between each test to assess the swimmer’s breathing response. The breathing test consists of approximately 5 minutes of deep breathing of room air (hyperventilation) followed by 4 minutes of breathing a mixture of low (but constant) level of oxygen and a gradually higher level of carbon dioxide by asking the swimmer to breathe into and out of a bag (rebreathing). The total time for the breathing test session will be approximately two hours. Participants will refrain from drinking caffeinated beverages for at least 2 hours prior to the rebreathing test.

We hope that the results will contribute to our understanding of the control of breathing in exercise and improve the future performances of athletes. Further, the knowledge gained from the study may assist the coaches and athletes in the design, planning and implementation of future training programs. Also, information gained in this study would, in the future, allow those athletes with low/high ventilatory responses to carbon dioxide to be identified and then placed in the appropriate distance whereby this natural ability would enhance the athlete’s potential performance.

There may be certain risks associated with participation in the study. The risks are:

1. Bruising and infection of the fingers may occur as a result of the finger prick during the lactate test.

2. During the breathing test, the swimmer could experience dizziness, headaches, nausea, tightness of the chest, a sense of smothering, feelings of apprehension, heart palpitations, abdominal pains, faintness, or impaired consciousness.

3. During the cycle fitness test, the swimmer could experience muscle discomfort, and shortness of breath.

The above risks are considered rare and will be controlled by the researcher. The measures are:

1. Finger bruising and infection will be prevented by adhering to proper technique (disinfecting and cleansing of the fingers before and after the finger prick) for finger pricking.
2. The risks of the breathing test will be prevented from arising during the hyperventilation phase of the breathing test by monitoring the expired level of carbon dioxide to prevent it from going too low (below 20 mmHg carbon dioxide, normal level is 40 mmHg) and during the rebreathing phase of the breathing test by monitoring the expired level of carbon dioxide and terminating the test if the level of carbon dioxide increases above 60 mmHg (normal level is 40 mmHg), and by monitoring the level of oxygen and terminating the test if the level of oxygen drops below 40 mm Hg (normal level is 100 mmHg). Further, oxygen saturation (level of oxygen in the blood) will be monitored using a pulse oximeter (a clamp that gently fits over a finger) and the test will be terminated if the oxygen saturation drops below 60% (normal level is 100%). Nevertheless, if any of these symptoms arise despite the measures undertaken by the researchers, the test will immediately be terminated.

3. The researcher will control for the risks involved in the cycle ergometer fitness test by (1) monitoring heart rates at all times and terminating the test if the participant’s heart rate reaches 85% of their age-predicted maximum heart rate (220 bpm-age), (2) terminating the test if the participant experiences signs of excessive discomfort, or (3) if the subject fails to conform to the exercise protocol.

There is no obligation to participate in the study and the swimmer and parent / guardian (if the child is under 16 years of age) may withdraw from the study at any time without affecting the coaching that the swimmer may receive. All information collected during the study will not identify the swimmer and parent / guardian by name and all information obtained will be kept in a secure location and will be destroyed 7 years following the completion of the study.

I acknowledge that I have read this form and I understand that my consent is voluntary and has been given under circumstances in which I can exercise free power of choice. I have been informed that I may ask further questions at any time, that at any time, I can revoke my consent, and that I am free to withdraw from the study at any time.

NAME (please print) ____________________________________________________________
DATE ___________________________
PARTICIPANT ______________________________________________________________
WITNESS _________________________
I agree to permit my son/daughter to participate in the above study to be conducted by Greg Wells. I so-doing understand the following: (1) my son/daughter's participation in the study will involve completing the rebreathing and swimming tests, described in the information sheet, on four separate occasions over a 6 month period. (2) any information or data related to my son/daughter will be kept confidential and anonymous and that only Greg Wells and the research committee will have access to the information. (3) I can withdraw my son/daughter from the study at any time.

NAME (please print) ____________________________________________________________

DATE ___________________________________________

PARENT'S SIGNATURE __________________________________________________________

WITNESS ___________________________________________
TEXT FOR ADVERTISEMENT FOR NON-ATHLETE COMPARISON GROUP

The department of Exercise Science at the University of Toronto is undertaking a study compare the breathing responses of swimmers and non-athletes. Mr. Greg Wells will be conducting the study as a Master of Science student. He can be reached at (416) 573-2258. Dr. Michael Plyley, University of Toronto Faculty of Physical Education and Health and Dr. James Duffin, University of Toronto Department of Physiology are supervising Greg. Dr. Plyley can be reached at 978-8563, Dr. Duffin can be reached at 978-6379. Permission to conduct the study has been granted from the Office of Research Services at the University of Toronto (protocol number 3670).

We are asking for you to volunteer for the study if you are:

a. healthy,
b. a post-pubescent non-athlete between the ages of 13-22 years of age.
c. have not been involved in regular training programs over the previous 12 months.

This means that you will not have exercised more than 3 times per week for no more than 40 minutes over the previous year.

Involvement in the study consists of two visits to the University of Toronto. Each visit will last about 2 hours and the visits will be separated by 6 months. At the end of the six months we will provide you with a personal exercise program.

We greatly appreciate your interest in the study. If you are interested in hearing more about the study, could you please provide your name and phone number to Mr. Greg Wells who will then contact you to describe the research in detail.

Greg Wells
Graduate Department of Exercise Science
University of Toronto
(416) 573-2258
greg.wells@utoronto.ca
DETAILED LETTER OF EXPLANATION OF THE STUDY –
NON-ATHLETES AND THEIR PARENTS / GUARDIANS

Date: __________________________

Dear __________________________ (participant; parents / guardians),

I am a Master of Science student in the Graduate Program in Exercise Science at the University of Toronto. As part of my Master’s thesis I am conducting a study about the effect of the intense exercise involved with competitive swimming training on the urge to breathe in swimmers. The study involves comparing the breathing responses of swimmers and non-athletes. Dr. Michael Plyley, University of Toronto School of Physical Education and Health and Dr. James Duffin, University of Toronto Department of Physiology are supervising the study. Dr. Plyley can be reached at 978-8563, and Dr. Duffin can be reached at 978-6379. Permission to conduct the study has been granted from the Office of Research Services at the University of Toronto (protocol number 3670).

On two separate occasions during the six months, we will ask the participants to come to the University of Toronto for testing. At the beginning of the study, the participants will be asked to complete a physical activity readiness questionnaire, and we will be asking them for information about their exercise history. On each of the two separate visits, the participants will have their height, arm length, foot length measured. Before the first breathing test, the participants will be asked to complete one 10-minute fitness test on a stationary exercise cycle. The test protocol involves a breathing test. The research involves assessing the participant’s breathing response. To this end, the participants will be asked to attend a habituation session to familiarize them to the breathing test and apparatus. On a separate occasion, the participants will complete 3 – 10 minute breathing tests separated by a minimum of 30 minutes between each test to assess the participant’s breathing response. The breathing test consists of approximately 5 minutes of deep breathing of room air (hyperventilation) followed by 4 minutes of breathing a mixture of low (but constant) level of oxygen and a gradually higher level of carbon dioxide by asking the participant to breathe into and out of a bag (rebreathing). The total time for
the breathing test session will be approximately two hours. Participant will refrain from drinking caffeinated beverages for at least 2 hours prior to the rebreathing test.

We hope that the results will contribute to our understanding of the control of breathing in exercise and improve the future performances of athletes. Further, the knowledge gained from the study may assist the coaches and athletes in the design, planning and implementation of future training programs. Also, information gained in this study would, in the future, allow those athletes with low/high ventilatory responses to carbon dioxide to be identified and then placed in the appropriate distance whereby this natural ability would enhance the athlete's potential performance. Also, the researchers will provide the non-athletes with a recommended exercise program following the research project.

There may be certain risks associated with participation in the study. During the breathing test, the participant could experience dizziness, headaches, nausea, tightness of the chest, a sense of smothering, feelings of apprehension, heart palpitations, abdominal pains, faintness, or impaired consciousness. During the fitness test, the participant may experience muscle discomfort, and shortness of breath.

The above risks are considered rare and will be controlled by the researcher. The risks of the breathing test will be prevented from arising during the hyperventilation phase of the breathing test by monitoring the expired level of carbon dioxide to prevent it from going too low (below 20 mmHg carbon dioxide, normal level is 40 mmHg) and during the rebreathing phase of the breathing test by monitoring the expired level of carbon dioxide and terminating the test if the level of carbon dioxide increases above 60 mmHg (normal level is 40 mmHg), and by monitoring the level of oxygen and terminating the test if the level of oxygen drops below 40 mm Hg (normal level is 100 mmHg). Further, oxygen saturation (level of oxygen in the blood) will be monitored using a pulse oximeter (a clamp that gently fits over a finger) and the test will be terminated if the oxygen saturation drops below 60% (normal level is 100%). Nevertheless, if any of these symptoms arise despite the measures undertaken by the researchers, the test will
immediately be terminated. The researcher will control for the risks involved with the cycle ergometer fitness test by (a) monitoring heart rates at all times and terminating the test if the participant’s heart rate reaches 85% of their age-predicted maximum heart rate (220 bpm-age), (b) terminating the test if the participant experiences signs of excessive discomfort, or if the subject fails to conform to the exercise protocol.

Prior to conducting the study, I will obtain written, informed consent from the participants and parent / guardian (if the child is under 16 years of age). There is no obligation to participate in the study and the participant and parent / guardian (if the child is under 16 years of age) may withdraw from the study at any time with no repercussions. All information collected during the study will not identify the participant and parent / guardian by name and all information obtained will be kept in a secure location and will be destroyed 7 years following the completion of the study.

Thank you for your interest in hearing about the study.

Sincerely,

Greg Wells
Master of Science Student
Graduate Department of Exercise Science
University of Toronto
Tel: (416) 573-2258
VOLUNTEER CONSENT FORM --- NON-ATHLETE

Project Title: Changes in the Chemoreflex Response with Training in Competitive Swimmers and Non-Athletes

Investigators: Greg Wells, Master of Science Candidate, Graduate Department of Exercise Science
Dr. Michael Plyley, Faculty of Physical Education and Health, University of Toronto
Dr. James Duffin, Department of Physiology, University of Toronto

Greg Wells, a Master of Science student in the Graduate Program in Exercise Science at the University of Toronto, is conducting a study about the effect of the intense exercise involved with competitive swimming training on the urge to breathe in swimmers. Dr. Michael Plyley, University of Toronto School of Physical & Health Education and Dr. James Duffin, University of Toronto Department of Physiology are supervising the study. Dr. Plyley can be reached at 978-8563, Dr. Duffin can be reached at 978-6379, and Greg Wells can be reached at 573-2258. Permission to conduct the study has been granted from the Office of Research Services at the University of Toronto (protocol number 3670).

At the beginning of the study, the participants will be asked to complete a physical activity readiness questionnaire, and we will be asking them for information about their exercise history. On two separate occasions during the six months, we will ask the participants to come to the University of Toronto for testing. Before the first breathing test, the participants will be asked to complete one 10-minute fitness test on a stationary exercise cycle. On each of the two separate visits, the participants will have their height, arm length, foot length measured. The test protocol involves a breathing test. The research involves assessing the participant’s breathing response. To this end, the participants will be asked to attend a habituation session to familiarize them to the breathing test and apparatus. On a separate occasion, the participants will complete 3 – 10 minute breathing tests separated by a minimum of 30 minutes between each test to assess the participant’s breathing response. The breathing test consists of approximately 5 minutes of deep breathing of room air (hyperventilation) followed by 4 minutes of
breathing a mixture of low (but constant) level of oxygen and a gradually higher level of carbon dioxide by asking the participant to breathe into and out of a bag (rebreathing). The total time for the breathing test session will be approximately two hours. Participant will refrain from drinking caffeinated beverages for at least 2 hours prior to the rebreathing test.

We hope that the results will contribute to our understanding of the control of breathing in exercise and improve the future performances of athletes. Further, the knowledge gained from the study may assist the coaches and athletes in the design, planning and implementation of future training programs. Also, information gained in this study would, in the future, allow those athletes with low/high ventilatory responses to carbon dioxide to be identified and then placed in the appropriate distance whereby this natural ability would enhance the athlete's potential performance. Also, the researchers will provide the non-athletes with a recommended exercise program following the research project.

There may be certain risks associated with participation in the study. The risks are:
1. During the breathing test, the participant could experience dizziness, headaches, nausea, tightness of the chest, a sense of smothering, feelings of apprehension, heart palpitations, abdominal pains, faintness, or impaired consciousness.
2. During the fitness test, the participant may experience muscle discomfort, and shortness of breath.

The above risks are considered rare and will be controlled by the researcher. The measures are:
1. The risks of the breathing test will be prevented from arising during the hyperventilation phase of the breathing test by monitoring the expired level of carbon dioxide to prevent it from going too low (below 20 mmHg carbon dioxide, normal level is 40 mmHg) and during the rebreathing phase of the breathing test by monitoring the expired level of carbon dioxide and terminating the test if the level of carbon dioxide increases above 60 mmHg (normal level is 40 mmHg), and by monitoring the level of oxygen and terminating the test if the level of oxygen drops
below 40 mm Hg (normal level is 100 mmHg). Further, oxygen saturation (level of oxygen in the blood) will be monitored using a pulse oximeter (a clamp that gently fits over a finger) and the test will be terminated if the oxygen saturation drops below 60% (normal level is 100%). Nevertheless, if any of these symptoms arise despite the measures undertaken by the researchers, the test will immediately be terminated.

2. The researcher will control for the risks involved with the cycle ergometer fitness test by (a) monitoring heart rates at all times and terminating the test if the participant’s heart rate reaches 85% of their age-predicted maximum heart rate (220 bpm-age), (b) terminating the test if the participant experiences signs of excessive discomfort, or if the subject fails to conform to the exercise protocol.

There is no obligation to participate in the study and the participant and parent/guardian (if the child is under 16 years of age) may withdraw from the study at any time with no repercussions. All information collected during the study will not identify the participant and parent/guardian by name and all information obtained will be kept in a secure location and will be destroyed 7 years following the completion of the study.

I acknowledge that I have read this form and I understand that my consent is voluntary and has been given under circumstances in which I can exercise free power of choice. I have been informed that I may ask further questions at any time, that at any time, I can revoke my consent, and that I am free to withdraw from the study at any time.

NAME (please print) ____________________________________________________________
DATE __________________________
PARTICIPANT ______________________________________________________________
WITNESS ________________________________________________________________

I agree to permit my son/daughter to participate in the above study to be conducted by Greg Wells. I so-doing understand the following: (1) my son/daughter's participation in the study will involve completing the rebreathing tests, described in the information sheet, on four separate occasions over a 6 month period. (2) any information or data related to my son/daughter will be kept confidential and anonymous and that only Greg
Wells and the research committee will have access to the information. (3). I can withdraw my son/daughter from the study at any time.

NAME (please print) ____________________________________________
DATE ____________________________________
PARENT'S SIGNATURE __________________________________________
WITNESS ______________________________
PAR-Q ASSESSMENT FORM TEXT

Before you participate in this research, please read carefully and check (√) “Yes” or “No” to the questions below.

1. Has your doctor said you have any heart problems? Yes ___ No ___
2. Do you frequently suffer from chest pains? Yes ___ No ___
3. Do you ever experience an irregular heart rate during exercise or at rest? Yes ___ No ___
4. Do you ever feel faint or have spells of dizziness? Yes ___ No ___
5. Has a doctor ever said that your blood pressure is too high? Yes ___ No ___
6. Do you often have difficulty breathing? Yes ___ No ___
7. Do you have asthma? Yes ___ No ___
8. Is there a good physical reason through your doctor’s advice or your own experience, why you should not follow an exercise program? Yes ___ No ___
9. Are you diabetic? Yes ___ No ___
10. Are you pregnant or have you given birth in the last 2 months? Yes ___ No ___

11. Do you currently have an illness or an infection? If yes, please explain. Yes ___ No ___

Name: ____________________________________________
Date: ____________________________________________
Signature: ________________________________________ (parent or guardian if under 16).
## Appendix B - Swimming Test Data

<table>
<thead>
<tr>
<th>Subject</th>
<th>Test</th>
<th>m/s</th>
<th>( V_{w} )</th>
<th>mmol/L</th>
<th>m/stroke</th>
<th>sec/str</th>
<th>m/breath</th>
<th>breath/sec</th>
<th>m/breath</th>
<th>breath/( m/s )</th>
</tr>
</thead>
<tbody>
<tr>
<td>A2</td>
<td>1</td>
<td>1.55</td>
<td>1.57</td>
<td>10.70</td>
<td>1.09</td>
<td>-0.97</td>
<td>3.13</td>
<td>-3.32</td>
<td>-2.81</td>
<td>33.87</td>
</tr>
<tr>
<td>A3</td>
<td>1</td>
<td>1.42</td>
<td>1.43</td>
<td>8.80</td>
<td>1.02</td>
<td>-2.09</td>
<td>2.27</td>
<td>-6.42</td>
<td>-6.26</td>
<td>73.62</td>
</tr>
<tr>
<td>A4</td>
<td>1</td>
<td>1.35</td>
<td>1.27</td>
<td>3.70</td>
<td>1.11</td>
<td>-1.14</td>
<td>3.33</td>
<td>-2.03</td>
<td>27.12</td>
<td></td>
</tr>
<tr>
<td>A5</td>
<td>1</td>
<td>1.38</td>
<td>1.65</td>
<td>9.40</td>
<td>1.16</td>
<td>-0.93</td>
<td>2.27</td>
<td>-5.57</td>
<td>-5.49</td>
<td>65.89</td>
</tr>
<tr>
<td>A6</td>
<td>1</td>
<td>1.38</td>
<td>1.41</td>
<td>8.60</td>
<td>1.14</td>
<td>-0.67</td>
<td>2.50</td>
<td>-3.54</td>
<td>-3.51</td>
<td>46.97</td>
</tr>
<tr>
<td>A7</td>
<td>1</td>
<td>1.48</td>
<td>1.45</td>
<td>8.50</td>
<td>1.06</td>
<td>-1.14</td>
<td>2.78</td>
<td>-4.81</td>
<td>-4.18</td>
<td>45.47</td>
</tr>
<tr>
<td>A8</td>
<td>1</td>
<td>1.43</td>
<td>1.47</td>
<td>12.50</td>
<td>1.02</td>
<td>-0.94</td>
<td>3.33</td>
<td>-5.80</td>
<td>-5.86</td>
<td>45.82</td>
</tr>
<tr>
<td>A9</td>
<td>1</td>
<td>1.37</td>
<td>1.42</td>
<td>3.70</td>
<td>1.14</td>
<td>-1.27</td>
<td>2.78</td>
<td>-6.68</td>
<td>-4.70</td>
<td>49.19</td>
</tr>
<tr>
<td>A11</td>
<td>1</td>
<td>1.27</td>
<td>1.29</td>
<td>11.80</td>
<td>1.04</td>
<td>-1.84</td>
<td>2.50</td>
<td>-3.63</td>
<td>-3.64</td>
<td>53.47</td>
</tr>
<tr>
<td>A13</td>
<td>1</td>
<td>1.41</td>
<td>1.46</td>
<td>11.80</td>
<td>1.04</td>
<td>-0.56</td>
<td>2.78</td>
<td>-4.48</td>
<td>-2.78</td>
<td>33.85</td>
</tr>
<tr>
<td>A14</td>
<td>1</td>
<td>1.26</td>
<td>1.31</td>
<td>12.80</td>
<td>1.04</td>
<td>-0.39</td>
<td>2.50</td>
<td>-3.49</td>
<td>-3.34</td>
<td>35.06</td>
</tr>
<tr>
<td>A15</td>
<td>1</td>
<td>1.42</td>
<td>1.53</td>
<td>10.90</td>
<td>1.16</td>
<td>-1.80</td>
<td>2.08</td>
<td>-4.72</td>
<td>-4.72</td>
<td>45.82</td>
</tr>
<tr>
<td>A16</td>
<td>1</td>
<td>1.30</td>
<td>1.33</td>
<td>12.30</td>
<td>1.02</td>
<td>-0.97</td>
<td>2.50</td>
<td>-4.28</td>
<td>-4.25</td>
<td>51.55</td>
</tr>
<tr>
<td>A17</td>
<td>1</td>
<td>1.42</td>
<td>1.49</td>
<td>13.50</td>
<td>1.11</td>
<td>-0.30</td>
<td>2.63</td>
<td>-5.41</td>
<td>-5.41</td>
<td>48.82</td>
</tr>
<tr>
<td>A21</td>
<td>1</td>
<td>1.61</td>
<td>1.65</td>
<td>10.30</td>
<td>1.28</td>
<td>-1.07</td>
<td>3.33</td>
<td>-0.35</td>
<td>20.24</td>
<td></td>
</tr>
<tr>
<td>A2</td>
<td>3</td>
<td>1.63</td>
<td>1.58</td>
<td>5.20</td>
<td>1.09</td>
<td>-1.07</td>
<td>3.13</td>
<td>-3.35</td>
<td>-3.34</td>
<td>39.04</td>
</tr>
<tr>
<td>A3</td>
<td>3</td>
<td>1.57</td>
<td>1.53</td>
<td>5.10</td>
<td>1.11</td>
<td>-0.81</td>
<td>3.33</td>
<td>-2.69</td>
<td>-2.70</td>
<td>32.52</td>
</tr>
<tr>
<td>A4</td>
<td>3</td>
<td>1.33</td>
<td>1.33</td>
<td>6.20</td>
<td>1.11</td>
<td>-0.47</td>
<td>3.33</td>
<td>-0.59</td>
<td>20.72</td>
<td></td>
</tr>
<tr>
<td>A5</td>
<td>3</td>
<td>1.66</td>
<td>1.72</td>
<td>11.00</td>
<td>1.14</td>
<td>-1.22</td>
<td>2.78</td>
<td>-5.43</td>
<td>-5.42</td>
<td>49.58</td>
</tr>
<tr>
<td>A6</td>
<td>3</td>
<td>1.53</td>
<td>1.40</td>
<td>6.90</td>
<td>1.05</td>
<td>-0.53</td>
<td>2.78</td>
<td>-2.45</td>
<td>-2.47</td>
<td>37.50</td>
</tr>
<tr>
<td>A7</td>
<td>3</td>
<td>1.50</td>
<td>1.49</td>
<td>10.10</td>
<td>1.04</td>
<td>-1.02</td>
<td>3.57</td>
<td>-3.50</td>
<td>-3.47</td>
<td>32.29</td>
</tr>
<tr>
<td>A8</td>
<td>3</td>
<td>1.47</td>
<td>1.51</td>
<td>16.00</td>
<td>1.09</td>
<td>-0.26</td>
<td>3.57</td>
<td>-3.78</td>
<td>-3.78</td>
<td>34.57</td>
</tr>
<tr>
<td>A9</td>
<td>3</td>
<td>1.47</td>
<td>1.46</td>
<td>6.20</td>
<td>1.11</td>
<td>-0.99</td>
<td>2.50</td>
<td>-3.29</td>
<td>-3.02</td>
<td>49.46</td>
</tr>
<tr>
<td>A11</td>
<td>3</td>
<td>1.29</td>
<td>1.29</td>
<td>4.90</td>
<td>0.89</td>
<td>-2.00</td>
<td>3.33</td>
<td>-5.99</td>
<td>-5.80</td>
<td>42.50</td>
</tr>
<tr>
<td>A13</td>
<td>3</td>
<td>1.56</td>
<td>1.60</td>
<td>13.00</td>
<td>1.25</td>
<td>-1.35</td>
<td>3.13</td>
<td>-10.22</td>
<td>-10.45</td>
<td>49.88</td>
</tr>
<tr>
<td>A14</td>
<td>3</td>
<td>1.30</td>
<td>1.37</td>
<td>13.00</td>
<td>1.04</td>
<td>-0.40</td>
<td>2.78</td>
<td>-3.39</td>
<td>-3.16</td>
<td>42.79</td>
</tr>
<tr>
<td>A15</td>
<td>3</td>
<td>1.66</td>
<td>1.50</td>
<td>11.60</td>
<td>1.28</td>
<td>-1.21</td>
<td>2.50</td>
<td>-8.40</td>
<td>-8.47</td>
<td>63.53</td>
</tr>
<tr>
<td>A16</td>
<td>3</td>
<td>1.31</td>
<td>1.33</td>
<td>8.90</td>
<td>1.02</td>
<td>-0.63</td>
<td>2.50</td>
<td>-3.51</td>
<td>-3.51</td>
<td>53.90</td>
</tr>
<tr>
<td>A17</td>
<td>3</td>
<td>1.51</td>
<td>1.55</td>
<td>16.60</td>
<td>1.14</td>
<td>-0.37</td>
<td>2.94</td>
<td>-3.74</td>
<td>-3.72</td>
<td>41.59</td>
</tr>
<tr>
<td>A21</td>
<td>3</td>
<td>1.52</td>
<td>1.51</td>
<td>9.00</td>
<td>1.11</td>
<td>-1.14</td>
<td>2.94</td>
<td>-5.32</td>
<td>-4.44</td>
<td>42.07</td>
</tr>
<tr>
<td>A8</td>
<td>4</td>
<td>1.50</td>
<td>1.54</td>
<td>8.90</td>
<td>1.22</td>
<td>-0.95</td>
<td>2.94</td>
<td>-4.19</td>
<td>-3.12</td>
<td>39.97</td>
</tr>
<tr>
<td>A9</td>
<td>4</td>
<td>1.51</td>
<td>1.54</td>
<td>8.90</td>
<td>1.22</td>
<td>-0.95</td>
<td>2.94</td>
<td>-2.80</td>
<td>-2.84</td>
<td>38.84</td>
</tr>
<tr>
<td>A11</td>
<td>4</td>
<td>1.34</td>
<td>1.34</td>
<td>5.80</td>
<td>1.09</td>
<td>-1.46</td>
<td>3.85</td>
<td>0.00</td>
<td>-0.88</td>
<td>19.11</td>
</tr>
<tr>
<td>A13</td>
<td>4</td>
<td>1.45</td>
<td>1.60</td>
<td>11.60</td>
<td>1.28</td>
<td>-1.05</td>
<td>5.56</td>
<td>-1.88</td>
<td>-1.92</td>
<td>14.84</td>
</tr>
<tr>
<td>A14</td>
<td>4</td>
<td>1.35</td>
<td>1.50</td>
<td>9.30</td>
<td>0.96</td>
<td>-0.97</td>
<td>2.38</td>
<td>-4.98</td>
<td>-2.62</td>
<td>43.16</td>
</tr>
<tr>
<td>A15</td>
<td>4</td>
<td>1.60</td>
<td>1.53</td>
<td>9.80</td>
<td>1.22</td>
<td>-1.12</td>
<td>3.33</td>
<td>-6.43</td>
<td>-7.25</td>
<td>43.44</td>
</tr>
<tr>
<td>A16</td>
<td>4</td>
<td>1.32</td>
<td>1.36</td>
<td>8.70</td>
<td>1.06</td>
<td>-0.73</td>
<td>2.94</td>
<td>-1.63</td>
<td>-1.64</td>
<td>31.54</td>
</tr>
<tr>
<td>A17</td>
<td>4</td>
<td>1.52</td>
<td>1.51</td>
<td>15.1</td>
<td>1.22</td>
<td>-0.28</td>
<td>2.94</td>
<td>-6.14</td>
<td>-6.13</td>
<td>55.33</td>
</tr>
<tr>
<td>A21</td>
<td>4</td>
<td>1.62</td>
<td>1.68</td>
<td>12.3</td>
<td>1.28</td>
<td>-0.46</td>
<td>3.33</td>
<td>-0.60</td>
<td>-0.60</td>
<td>21.92</td>
</tr>
</tbody>
</table>
### Appendix C - Rebreathing Test Data

<table>
<thead>
<tr>
<th>Subject</th>
<th>PO2</th>
<th>Athlete</th>
<th>test</th>
<th>CO2</th>
<th>VE</th>
<th>V T</th>
<th>VE T</th>
<th>VE S</th>
<th>VT S</th>
<th>VE T</th>
<th>VT S</th>
<th>Fb</th>
<th>F T1</th>
<th>F T2</th>
<th>F S1</th>
<th>F S2</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1</td>
<td>150</td>
<td>Y</td>
<td>1</td>
<td>0.0653</td>
<td>4.49</td>
<td>41.51</td>
<td>2.26</td>
<td>264.21</td>
<td>41.12</td>
<td>97.20</td>
<td>17.12</td>
<td>41.00</td>
<td>0.43</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A1</td>
<td>150</td>
<td>Y</td>
<td>2</td>
<td>0.0972</td>
<td>4.47</td>
<td>47.39</td>
<td>7.15</td>
<td>555.00</td>
<td>41.07</td>
<td>18.17</td>
<td>18.38</td>
<td>38.98</td>
<td>0.10</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A2</td>
<td>150</td>
<td>Y</td>
<td>3</td>
<td>0.0709</td>
<td>4.22</td>
<td>45.49</td>
<td>1.83</td>
<td>534.97</td>
<td>43.21</td>
<td>102.84</td>
<td>16.67</td>
<td>0.03</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A3</td>
<td>150</td>
<td>Y</td>
<td>4</td>
<td>0.0842</td>
<td>4.27</td>
<td>44.92</td>
<td>1.28</td>
<td>139.12</td>
<td>42.50</td>
<td>63.71</td>
<td>16.30</td>
<td>43.00</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A4</td>
<td>150</td>
<td>Y</td>
<td>5</td>
<td>0.0970</td>
<td>4.30</td>
<td>42.65</td>
<td>5.00</td>
<td>1.81</td>
<td>4.06</td>
<td>209.95</td>
<td>43.11</td>
<td>54.60</td>
<td>122.16</td>
<td>45.40</td>
<td>18.55</td>
<td>52.30</td>
</tr>
<tr>
<td>NA10</td>
<td>150</td>
<td>N</td>
<td>1</td>
<td>0.0782</td>
<td>4.96</td>
<td>43.59</td>
<td>6.35</td>
<td>680.50</td>
<td>42.92</td>
<td>275.23</td>
<td>14.37</td>
<td>42.66</td>
<td>0.77</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NA10</td>
<td>150</td>
<td>N</td>
<td>2</td>
<td>0.0886</td>
<td>4.66</td>
<td>43.37</td>
<td>1.99</td>
<td>273.05</td>
<td>41.05</td>
<td>78.69</td>
<td>10.22</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NA10</td>
<td>150</td>
<td>N</td>
<td>3</td>
<td>0.0794</td>
<td>4.71</td>
<td>41.03</td>
<td>4.25</td>
<td>3.19</td>
<td>0.99</td>
<td>499.57</td>
<td>40.91</td>
<td>48.44</td>
<td>149.68</td>
<td>122.83</td>
<td>12.28</td>
<td>47.18</td>
</tr>
<tr>
<td>NA10</td>
<td>150</td>
<td>N</td>
<td>4</td>
<td>0.0842</td>
<td>4.27</td>
<td>44.92</td>
<td>1.28</td>
<td>139.12</td>
<td>42.50</td>
<td>63.71</td>
<td>16.30</td>
<td>43.00</td>
<td>0.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NA10</td>
<td>150</td>
<td>N</td>
<td>5</td>
<td>0.0970</td>
<td>4.30</td>
<td>42.65</td>
<td>5.00</td>
<td>1.81</td>
<td>4.06</td>
<td>209.95</td>
<td>43.11</td>
<td>54.60</td>
<td>122.16</td>
<td>45.40</td>
<td>18.55</td>
<td>52.30</td>
</tr>
</tbody>
</table>

**100**