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# The acute effects of controlled breathing training in swimming on glycolytic metabolism.

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# THE ACUTE EFFECTS OF CONTROLLED BREATHING TRAINING IN SWIMMING ON GLYCOLYTIC METABOLISM

By

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ABSTRACT

THE ACUTE EFFECTS OF CONTROLLED BREATHING TRAINING IN SWIMMING ON GLYCOLYTIC METABOLISM

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The term Hypoxic Training has been popular in swimming for the past few years. It is actually more correctly called Controlled Breathing Training (CBT). The purpose of this study was to investigate the acute effects of CBT on the blood parameters of glucose (GL) and lactate (LA), the acid-base parameters of pH and $P_vCO_2$, and recovery oxygen uptake. Six male swimmers were studied in two separate swimming exercise sessions using two breathing patterns - free breathing (FB), and 7-stroke breathing (7B). The swimming intensity for all swims was determined to be at 80 to 88% of each individual's best 200-yard time. For each session venous blood samples were drawn prior to the swim (Rest), immediately after the swim (IA), and during the 6th minute of recovery (6-R). Analyses for GL, LA, pH, and $P_vCO_2$ were done, and recovery $\dot{V}O_2$ (20 minutes) was calculated. Significant increases due to the exercise sessions themselves were found in both GL ($p < .05$), and LA ($p < .05$) concentrations. Between the two swim protocols significant difference was found in only one of the variables, that of $CO_2$ production. The $CO_2$ produced during the first 30 seconds of recovery was greater after the 7B than after the FB ($p < .05$). These results indicated that the acute CBT did not induce more stress on the glycolytic metabolism than did the normal training. From these data it appears that decreases in sprinting time attributed to hypoxic

training may not be the result of physiological change, but rather due to other changes such as biomechanical efficiency or psychological mind set.

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# DEDICATION

To Sally

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## LIST OF ABBREVIATIONS

CBT = controlled breathing training in swimming.

FB = free breathing pattern during swimming.

7B = 7-stroke breathing pattern during swimming.

REST = prior to the swimming exercise session.

IA = immediately after the exercise session.

6-R = at the 6th minute of recovery.

NR-20 = net recovery $O_2$ uptake for 20 minutes.

GL = glucose

LA = lactate

$P_VCO_2$ = partial pressure of $CO_2$ in venous blood.

pH = the negative log of the hydrogen ion concentration in a solution which determines its acidity.

$\dot{V}O_2$ = oxygen uptake per unit time measured.

$F_IO_2$ = fraction of oxygen in the inspired air (.2093 at sea level).

$F_EO_2$ = fraction of oxygen in the expired gas.

$F_ICO_2$ = fraction of carbon dioxide in the inspired air.

$F_ECO_2$ = fraction of carbon dioxide in the expired gas.

STPD = standard temperature ($0^{\circ}C$), pressure (760 mmHg), and dry.

# CHAPTER I

## INTRODUCTION

The goal of athletic conditioning is to improve physical fitness and performance. From a physiological point of view, in order to achieve this goal the metabolic aspects of a training program must be considered. The maximum training effect can only be obtained from optimal metabolic response. The two major metabolic pathways which may be stressed in training are the glycolytic (anaerobic) pathway and the oxidative (aerobic) pathway. Both of these pathways are able to function at the same time for ATP production in order to perform work.

In competitive swimming, conditioning methods such as Repetition Training, Overdistance Training, Sprint Training, Fartlek Training, and Interval Training have dominated the training program for the past few decades (Counsilman, 1977). The credibility of these training methods has been fairly well documented (Counsilman, 1968, 1977; Houston, 1978). A relatively new method of training in swimming called "Hypoxic Training" has gained in popularity in the past several years (Counsilman, 1975; Shaw, 1979). However, very little scientific investigation has been directed toward Hypoxic Training, so that little is actually known about its metabolic consequences.

# CHAPTER II

## REVIEW OF RELATED LITERATURE

### A. Controlled Breathing Training (CBT)

The so-called "Hypoxic Training" in swimming is more accurately described as Controlled Breathing Training (or skipped breathing training), since the swimmers are instructed to breathe less frequently than normal. Without scientific investigation, it was assumed that these less frequent breathing patterns would decrease the oxygen supply to the body, and thus create hypoxia within the working cells. For this reason the term "Hypoxic Training" was adopted.

If the assumption which was made regarding controlled breathing training is true, the chronic effect of this type of training which occurred at sea level should be similar to that experienced by athletes who have trained at a high altitude and returned to sea level. The altitude training effects have been summarized by Stiles (1974), Colwin (1975), and Woodburg (1978). They stated that altitude training will increase aerobic capacity due to: 1) increased pulmonary ventilation, 2) increased cardiac output, 3) increased oxygen carrying capacity, and 4) increased vascularization. However, these statements seem to conflict with the reports by Patton and his co-workers (1979). They reported that only the "lactacid" part (borrowed energy sources) of the anaerobic capacity was improved through hypoxic training in swimming. Bonen (1979) also stated that hypoxic training is purely anaerobic and that there is no improvement in aerobic capacity.

There have been some studies using a reduced fraction of inspired oxygen ($F_IO_2$) which have questioned this hypoxic effect. Davis and

Sargeant (1974) found no clear evidence that training, in reduced $F_IO_2$, enhanced the improvement of $\dot{V}O_2$ max as observed under normoxic conditions. Jones et al. (1972) showed that hypoxia ($F_IO_2$ = .11 ~ .13) was associated with high blood lactate levels. Furthermore, Kinnula (1976) and Kinnula and Hassinen (1977) found similar results showing that hypoxia was not considered a stimulus for increases in the concentration of mitochondrial oxidative enzymes. In any case, other factors which determine the chronic hypoxic effects, such as the duration of exposure to hypoxia and the age at which it was initiated should be considered when interpreting these types of data (Frisancho et al., 1973).

As mentioned earlier, it was assumed that controlled breathing training created hypoxia within the working cells. In fact, Wilson (1976) reported that controlled breathing patterns in swimming created some degree of hypoxia within the lung, and that this hypoxia could have affected anaerobic and aerobic systems. However, studies by Craig (1979) and Dicker et al. (1980) showed that controlled breathing during exercise created "hypercapnia" (high $P_ACO_2$) rather than "hypoxia". Craig (1979) suggested that the so-called "hypoxic training" should be called "hypercapnic training". Curran (1977) suggested that it was likely that muscle cells were already hypoxic during intense exercise. Therefore, it was questionable whether inducing muscle hypoxia would have any further effect.

Regardless of whether controlled breathing during exercise would induce hypoxia or not, the other effects of this training which have been reported cannot be ignored. Counsilman (1975) reported that "hypoxic training" increased heart rate, oxygen debt, and lactate tolerance levels. Vaser and Laidre (1975), and Kedrowsky (1976) found

that controlled breathing training workouts increased cardiopulmonary efficiency and also increased heart rate nearly 10% more than did normal breathing training. Shaw (1979) stated that "hypoxic training" forced the body to work under stress without a sufficient amount of oxygen, and thus increased the number of red blood cells as a chronic effect.

Patton et al. (1979) found that "hypoxic" swimming training significantly improved sprinting performance for 100 yards but not for 50 yards. Sparks (1973) found that swimming training using controlled breathing would benefit the swimmer by improving his ability to extract more oxygen per unit volume ventilated. This result did agree with the study by Hsieh (1979), and Dicker et al. (1980). Both studies showed that the swimmers who used a more controlled breathing pattern (lower breathing frequency) had higher oxygen extraction than the swimmers who used a less controlled pattern (higher breathing frequency), even though no difference in $O_2$ consumption was found for a given work load. Dicker et al. (1980) also showed that a more controlled breathing pattern caused hypercapnia rather than hypoxia. These data agreed with the report by Craig (1979).

## B. Exercise Related Glycolytic Metabolism

### Blood Glucose

Research has shown that blood glucose and free fatty acid were the major substrates for metabolism in exercising muscle (Ahlborg, 1974). The normal venous blood glucose concentration is 70-105 mg/100 ml. However, blood glucose values have been found to be noticeably influenced by the previous day's food intake (Stock, 1980). Diet high in carbohydrates not only elevated the blood glucose value, but also increased the liver and muscle glycogen concentrations (Hultman and Nilsson, 1975). Physical

performance time has been positively associated with high levels of these substrates in these tissues (Hultman, 1967; Hultman and Nilsson, 1975). Furthermore, carbohydrates have been shown to be the exclusive fuel for working muscles at work loads which were greater than 85-90% $\dot{V}O_2$ max (Bergstrom and Hultman, 1972). It has also been reported that during hard exercise the glucose output from the liver may increase up to seven times the resting value (Hultman and Nilsson, 1975).

Blood Lactate

The end-product of glycolytic metabolism is lactate. The normal blood lactate concentration for a healthy adult at rest is 3-12 mg/100 ml. Under anaerobic exercise conditions, lactate has to build up in order to ensure sufficient NAD co-factor for glycolysis to continue (Lehninger, 1977). Lactate is an easily diffusible by-product. A study by Diamant et al. (1968) indicated that the lactate concentration in the skeletal muscle was much higher than in the blood during maximum exercise. However, shortly after the exercise was stopped (approximately 6~7 minutes), the peak blood lactate concentration was reached, and this level was very close to the concentration in the muscle. Blood lactate as high as 198 mg/100 ml (or 22 mM/liter) has been reported after exhausting exercise (Hermansen, et al., 1973). Because of the rapid distribution of lactate, valuable information about changes taking place in the muscle were able to be detected in the blood. These values are significant since it has been suggested that both muscle and blood lactate concentrations should be regarded as an indication of the extent to which the glycolytic process is activated (Gollnick and Hermansen, 1973; Jensen and Fisher, 1979).

## Blood Gases

Hypoventilation during exercise such as breathholding in swimming can result in an elevated arterial $PCO_2$ above normal limits (35-45 mm Hg). It has been stated that high $PCO_2$ was usually associated with respiratory acidosis which caused a drop in blood pH values (West, 1976). Respiratory acidosis plus a high concentration of intracellular lactic acid during muscular activity have been shown to arrest glycolytic metabolism within a few seconds (Mader, et al. 1978; Sutton, et al. 1976). The activity of the regulatory enzyme in glycolysis, PFK, was inhibited if the cellular pH value dropped from 7.4 by 0.6-0.7 units (Mader, et al. 1978). This acidosis can be buffered by the blood bicarbonate, and by respiratory hyperventilation.

It is believed that arterial blood is the most valuable for blood gases analysis. However, recently research has shown that venous blood could be used in most cases in place of arterialized capillary or arterial blood to determine pH, total $CO_2$ content, and $PCO_2$ (Race, 1978). For these 3 parameters, very little variation has been found between arterial and venous blood samples. In fact, it has been shown that venous blood pH better represented the total body acid-base status, since arterial pH could be rapidly changed by emotion and respiration during the drawing of the blood samples (Gambino, 1966).

## Recovery $O_2$ Uptake

The resting oxygen uptake ($\dot{V}O_2$) of a normal adult is around 0.2-0.4 liter/minute. However, $\dot{V}O_2$ can be increased rapidly at the onset of exercise. A $\dot{V}O_2$ value of 5.0 liter/minute was not uncommon during maximum exercise for a well trained athlete. Following exercise, the

$O_2$ consumed above the resting level was referred to as net recovery $O_2$ uptake. The volume of this recovery $O_2$ uptake was generally caused by the intensity and duration of the exercise, and also by the condition of the athlete (Hagberg, et al. 1980; Mathews, and Fox, 1976).

# CHAPTER III

## PURPOSE OF STUDY

If increased $O_2$ extraction, increased $P_ACO_2$, combined with no change in $O_2$ uptake are the major effects of controlled breathing, several questions remain unanswered. Will this increased $P_ACO_2$ be high enough to induce respiratory acidosis and drop the blood pH values (Craig, 1979; Dicker et al., 1980)? Will the glycolytic metabolism be over stressed by a decreased blood glucose concentration and cause a high lactate level (Houston, 1978; Bonen, 1979)? Furthermore, will this controlled breathing result in greater recovery $O_2$ uptake to the body as a whole (Counsilman, 1977; Patton et al., 1979)?

It was the purpose of this study to compare the acute effects of controlled breathing training and free breathing training on venous blood gases parameters, blood glucose and lactate concentrations, and the recovery $O_2$ uptake.

As a result the following hypotheses were made:

Null Hypothesis:

There will be no difference (designated by =) in effect on blood chemistry and recovery $O_2$ uptake between the free breathing (FB) and the 7-stroke breathing (7B) patterns.

$$H_0 : FB = 7B$$

Experimental Hypothesis:

There will be a difference (designated by $\neq$) in effect on blood chemistry and recovery $O_2$ uptake between the free breathing (FB) and the 7-stroke breathing (7B) patterns.

$$H : FB \neq 7B$$

# CHAPTER IV

# METHODS

## Subjects

Six volunteer male swimmers from Windsor Aquatic Club were the subjects for this study. They were all healthy competitive swimmers, and were familiar with controlled breathing training. Prior to the experiment, the purpose and procedures were both explained, and a consent form (see Appendix A) was signed by each subject. The signature of a parent was also obtained for those who were under 16 years of age.

The physical characteristics of these subjects are shown in Table 1. The percent body fat was measured with John Bull skin fold calipers (British Indicators Ltd., England) using the method of Forsyth et al. (1973). The maximum oxygen uptake (max $\dot{V}O_2$) was determined on a Monark bicycle ergometer (Stockholm, Sweden) combined with a hand crank ergometer (modified from the Quinton bicycle ergometer, Seattle, Washington). The open circuit spirometry method was used for this $\dot{V}O_2$ test (Consolazio, 1963). These two tests were done prior to the experiment in conjunction with a longitudinal research program involving the Windsor Aquatic Club swimmers. This research program was funded by a University of Windsor research grant (#900A-15). The glucose (GL), lactate (LA), and hemoglobin (Hb) values were obtained during the experiment, and were calculated as the mean of the 2 resting blood samples.

## Procedure

A standardized experimental protocol (see Appendix B) was followed throughout the experiment. A physician had agreed to be available for assistance at any time during the testing sessions. The order of testing

TABLE 1

PHYSICAL CHARACTERISTICS OF SUBJECTS

| Subject | Age (year) | Weight (kg) | Height (cm) | Body Fat (%) | $\dot{V}O_2$ Max (ml/m/kg) | Blood Glucose (mg%) | Blood Lactate (mg%) | Blood Hb (g%) | Best 200 Yard Time (min:sec) |
|---|---|---|---|---|---|---|---|---|---|
| J.S. | 16 | 77.7 | 180.3 | 10.27 | 58.62 | 81.23 | 10.25 | 15.9 | 2:05 |
| B.M. | 20 | 89.4 | 193.0 | 10.83 | 71.07 | 83.10 | 5.85 | 16.9 | 1:46 |
| E.J. | 15 | 65.3 | 176.5 | 8.72 | 57.26 | 84.17 | 10.32 | 16.2 | 1:52 |
| R.B. | 15 | 66.2 | 172.0 | 9.58 | 65.58 | 81.88 | 8.34 | 15.9 | 2:06 |
| R.H. | 15 | 61.4 | 176.5 | 8.37 | 56.67 | 86.59 | 9.26 | 16.6 | 1:52 |
| T.M. | 18 | 77.9 | 186.7 | 10.91 | 67.82 | 92.73 | 9.57 | 17.0 | 1:49 |
| $\bar{X}$ | 16.50 | 72.98 | 180.83 | 9.78 | 62.84 | 84.95 | 8.93 | 16.42 | 1:55 |
| $\pm S\bar{x}$ | ±0.84 | ±4.30 | ±3.15 | ±0.44 | ±2.50 | ±1.74 | ±.68 | ±.20 | ±0:03 |

was chosen at random. However, slight adjustments were made in order to accommodate previously scheduled swimming meets. Each subject was exposed to two different experimental conditions - free breathing, and 7-stroke breathing. The testing was done in the morning during regular training sessions. The tests were performed at the Human Kinetics Pool, University of Windsor. The pool was 25 yards long, and the water temperature during the experiment was between 28°C-29°C.

Control of Diet and Physical Activity

The subjects were instructed to maintain a balanced diet and their usual pattern of physical activity for 3 days prior to each test. The parents were also informed of these instructions. A Canada's Food Guide, and a diet and activity record sheet (see Appendix C) were given to each subject. The daily record sheet was submitted to the investigator, and was checked before testing began. The subjects were also required to fast overnight. The control described above was necessary in order to minimize the fluctuation of blood metabolites and physical work capacity (Kelman, et al. 1975; Stock, 1980).

Exercise Session

The exercise session consisted of 15 discontinuous 200-yard swims with a 30-45 second rest between each swim. The total work time was approximately 30 minutes for each session. Each swimmer was required to swim at a speed of 85% of his best time using the front crawl stroke. This workout was designed to ensure that enough stress was put on the subject in order for the metabolic changes to take place. However, care was taken to detect any signs of discomfort which could be caused by the 7-stroke breathing pattern (Counsilman, 1977). Each subject used only

the pre-assigned breathing pattern for the entire training session.

Blood Samples

Blood samples were drawn from a cubital vein with multi sampling needles (Jelco Laboratories, N.J.) into 10 cc vacuum tubes coated with sodium heparin (Vacutainer, B-D & Co.,Canada). Vacuum tubes have been proven to be adequate for blood gases analysis (Moran, 1979). Samples were collected prior to the swim (REST), immediately after the exercise session (IA), and at the 6th minute of recovery (6-R). It has been reported that after intense exercise a blood sample taken at the 6th minute of recovery has the highest lactate concentration (Diamant, et al. 1968).

Two tubes of blood were drawn at each sampling time. The first tube was filled as full as possible, and was handled anaerobically. It was kept submerged in ice water and was analyzed within 2 hours with the ABL 2 blood gases analyzer (Acid Base Laboratory, Radiometer, Copenhagen, Denmark) at the Windsor Western Hospital. The ABL 2 blood gases analyzer was calibrated daily by the hospital laboratory staff.

The blood in the second tube was immediately treated with 4% sodium fluoride to prevent further glycolysis (Sigma, 1978; Sazama, et al. 1979). This sample was stored at $0^{\circ}$-$5^{\circ}$C and was analyzed within a few hours for the glucose and lactate concentrations. Two millilitres of this sample were separated and were deproteinized with 4.0 ml of 8% cold perchloric acid. The supernatant obtained after centrifugation at 3000 RPM was analyzed immediately for lactate concentration using the lactate dehydrogenase method (see Appendix D). The remainder of the blood sample in the second tube was centrifuged, and the plasma obtained

was used to determine the glucose concentration using the o-toluidine reagent method (see Appendix E).

A Beckman DB-G Spectrophotometer (Beckman Instruments Inc., U.S.A.), and a Unicam SP 1700 Ultraviolet Spectrophotometer (Pye Unicam Ltd., Cambridge, England) were used to read the optical density in order to determine the substrate concentration. Both the glucose and the lactate assays were done in triplicate and the mean value of each was used for statistical analysis.

Oxygen Uptake

Oxygen uptake ($\dot{V}O_2$) was measured using the open circuit spirometry method (Consolazio, 1963). The gas collecting system used in this study is shown in Figure 1. The respiratory valve of the apparatus was connected with plastic tubing to a 5-way metal valve which was attached to a 120-liter Chain-Compensated Gasometer (Warren E. Collins Incorporated, Mass., U.S.A.). Two 3-way valves were connected to the 5-way valve at the end opposite to the gasometer. By this arrangement, four 120-liter meteorological balloons (bags) could fit onto the various valves at one time. This system allowed for continuous collection of the expired gas.

The time periods (in minutes) over which the gas samples were collected were as follows: 1) at rest: 0-2.0; 2) during recovery: 0-0.5, 0.5-3.0, 3.0-6.0, 6.0-12.0, 12.0-18.0, and 18.0-20.0. The gases collected in the bags were pumped into the gasometer by a motor blower (Collins). The volume of the gas was then determined. At this time also the temperature and barometric pressure were recorded in order to convert the volume under STPD conditions. The gas was then mixed well and a small sample was drawn off for the analysis of $O_2$ and $CO_2$ concentrations. This analysis was done with a S-AS Oxygen Analyzer and a CD-2 $CO_2$ Analyzer

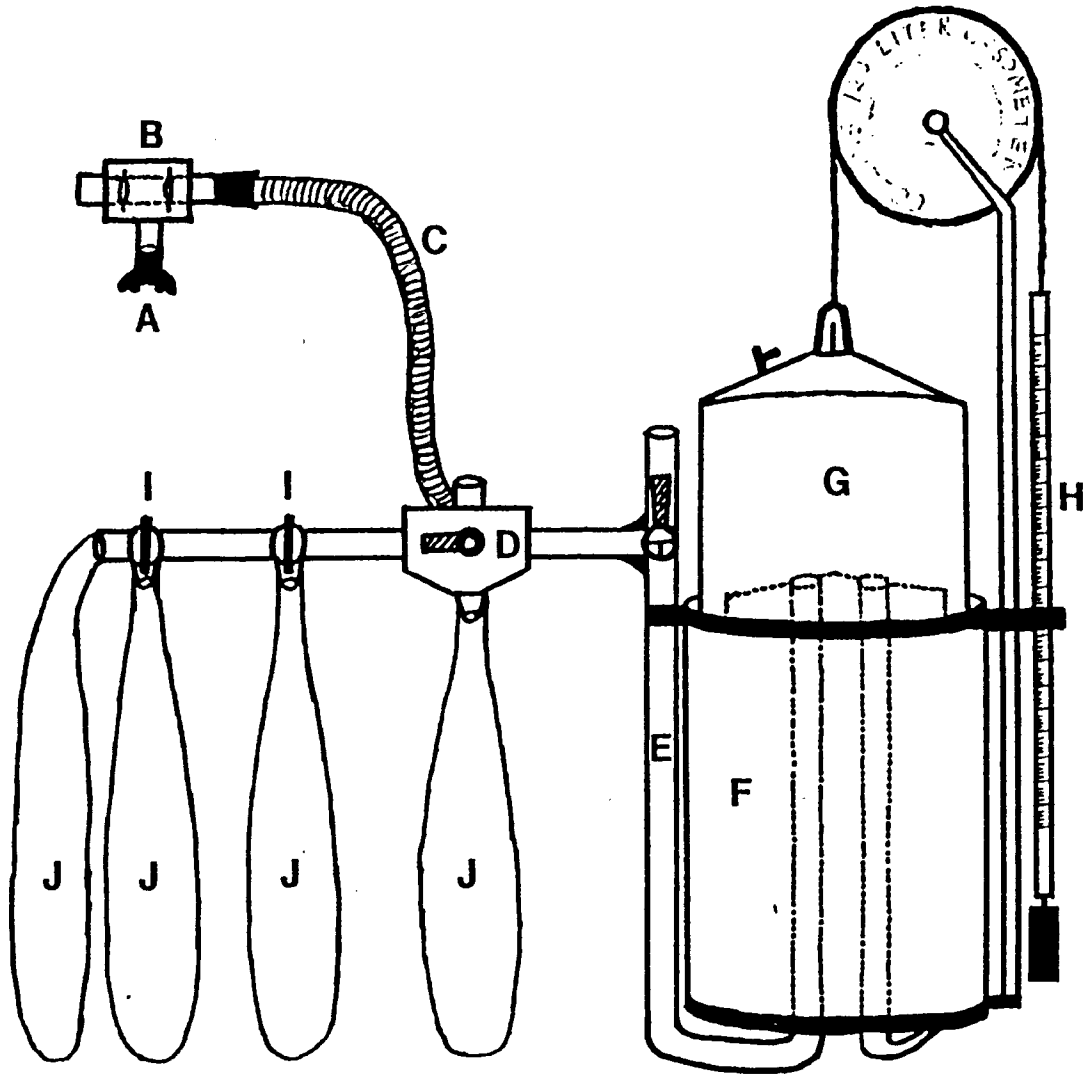


FIGURE 1. GAS COLLECTION SYSTEM

A = MOUTH PIECE. C = PLASTIC TUBING. E = AIR PIPE. G = GASOMETER BELL. I = 3-WAY VALVE.

B = RESPIRATORY VALVE. D = 5-WAY VALVE. F = GASOMETER TANK. H = VOLUME METER. J = METEOROLOGICAL BALLOON.

(Applied Electrochemistry Inc., Sunnyvale, California). Oxygen uptake was then calculated (see Appendix F). Each time prior to the gas analysis, the analyzers were calibrated with reference gases, the concentrations of which had been pre-determined with a Lloyd-Gallenkamp Gas Analyzer (A. Gallenkamp and Co. Ltd., England).

Statistical Analysis

In order to determine the effectiveness of the diet and physical activity control, a correlated Student t-test as well as a Pearson Product-Moment correlation coefficient (Ferguson, 1976) were performed on the resting valves. The purpose of this was to detect the baseline difference between the 2 different test days. The t-test for the $O_2$ extraction and the $CO_2$ production were also performed. This analysis was done manually using an electronic calculator (EL-8128, Sharp, Japan).

A two-way analysis of variance (ANOVA) with repeated measures on two factors followed by the Post-Hoc Duncan's multiple range test were used to analyze the main effects and the interaction. This was performed in the Computer Center at the University of Windsor using the ANOVA Procedure of the SAS System (Barr, et al., 1979). The results from the computer were checked with the Scheffé's method for Post-Hoc multiple comparisons which was performed manually.

# CHAPTER V

## RESULTS

The resting values of the glucose, lactate, pH, $P_vCO_2$ and $\dot{V}O_2$ of the 2 different test days were shown to be highly correlated, and no significant differences could be found in the t-test (Table 2). These homogeneous baseline data validated the further analysis of the treatment effects. Furthermore, the calibration curves of the lactate (in Appendix D), and glucose (in Appendix E) were almost perfectly linear. These curves demonstrated that there was a high degree of technique reliability, and also that the reproducibility of the biochemical procedures was satisfactory.

TABLE 2

CORRELATION COEFFICIENT ($\gamma$) AND t-TEST OF THE RESTING VALUES BETWEEN THE FREE BREATHING AND 7-STROKE BREATHING PATTERNS

| | $\gamma$ | t-test |
|---|---|---|
| Glucose | .99 | $p > .01$ |
| Lactate | .95 | $p > .01$ |
| pH | 1.00 | $p > .01$ |
| $P_vCO_2$ | .99 | $p > .01$ |
| $\dot{V}O_2$ | .99 | $p > .01$ |

The swimming intensity of the exercise sessions was measured to be at 80-88% (84.33, $\pm S\bar{x}$ 0.84) of each individual's best 200-yard time (Table 3).

TABLE 3

SWIMMING INTENSITY (%)* OF THE EXERCISE SESSIONS

| Subject | Free Breathing | 7-Stroke Breathing |
|---|---|---|
| J.S. | 80 | 80 |
| B.M. | 85 | 84 |
| E.J. | 85 | 87 |
| R.B. | 88 | 88 |
| R.H. | 84 | 81 |
| T.M. | 85 | 85 |
| $\bar{X} \pm S\bar{x}$ | 84.50 ± 1.06 | 84.16 ± 1.30 |
| Total $\bar{X} \pm S\bar{x}$ | 84.33 ± 0.84 | |

* The individual's best 200-yard time was considered as the 100% intensity.

Raw data collected from this study can be found in Appendix G. Mean values with standard error of mean ($S\bar{x}$) of these data are graphically presented in Figure 2 (glucose), Figure 3 (lactate), Figure 4 (pH), Figure 5 ($P_vCO_2$), and Figure 6 ($\dot{V}O_2$).

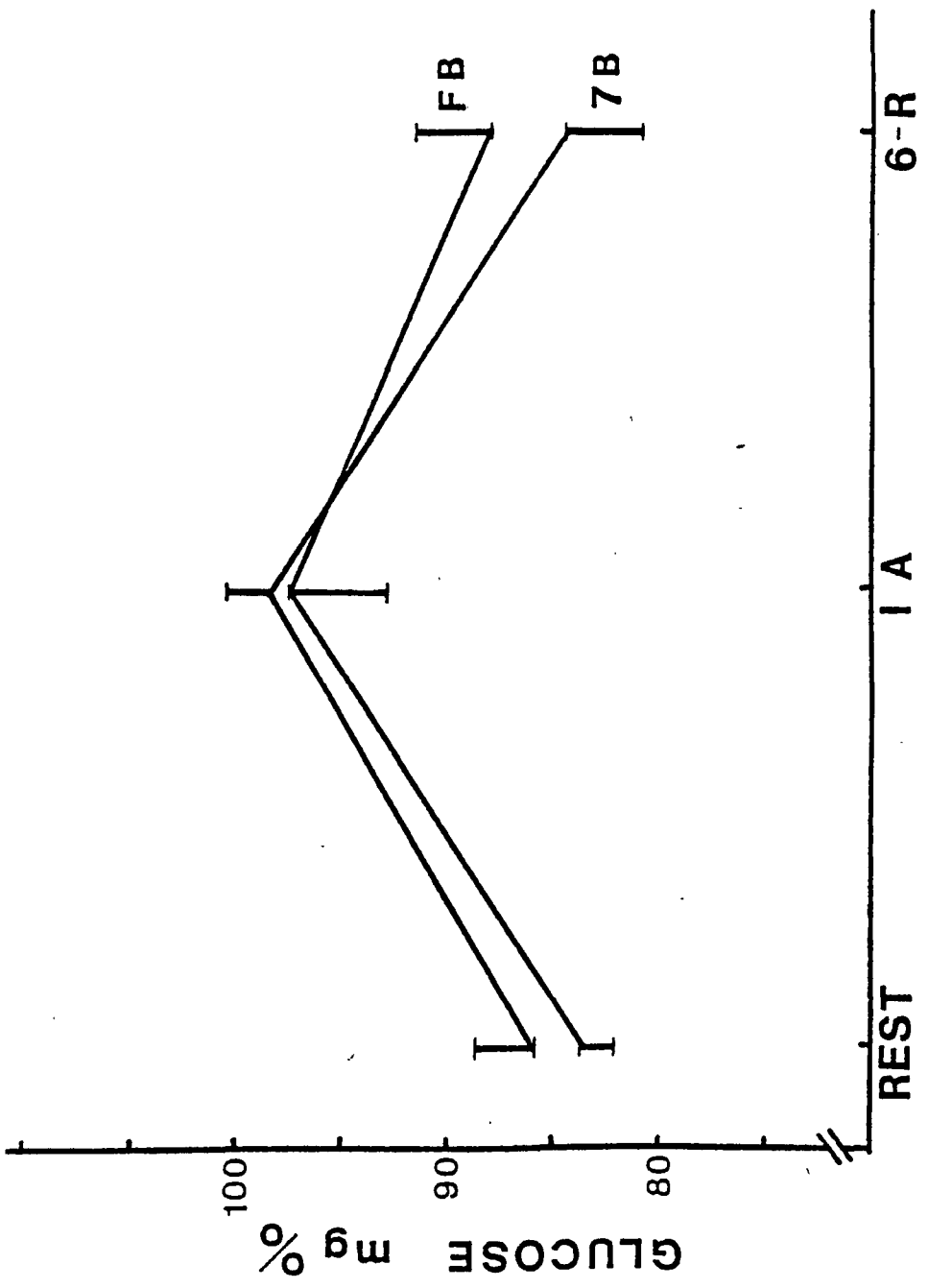


FIGURE 2. EFFECT OF FREE BREATHING (FB), AND 7-STROKE BREATHING (7B) ON VENOUS BLOOD GLUCOSE CONCENTRATIONS PRIOR TO THE SWIM (REST), IMMEDIATELY AFTER THE EXERCISE SESSION (IA), AND AT THE 6TH MINUTE OF RECOVERY (6-R).

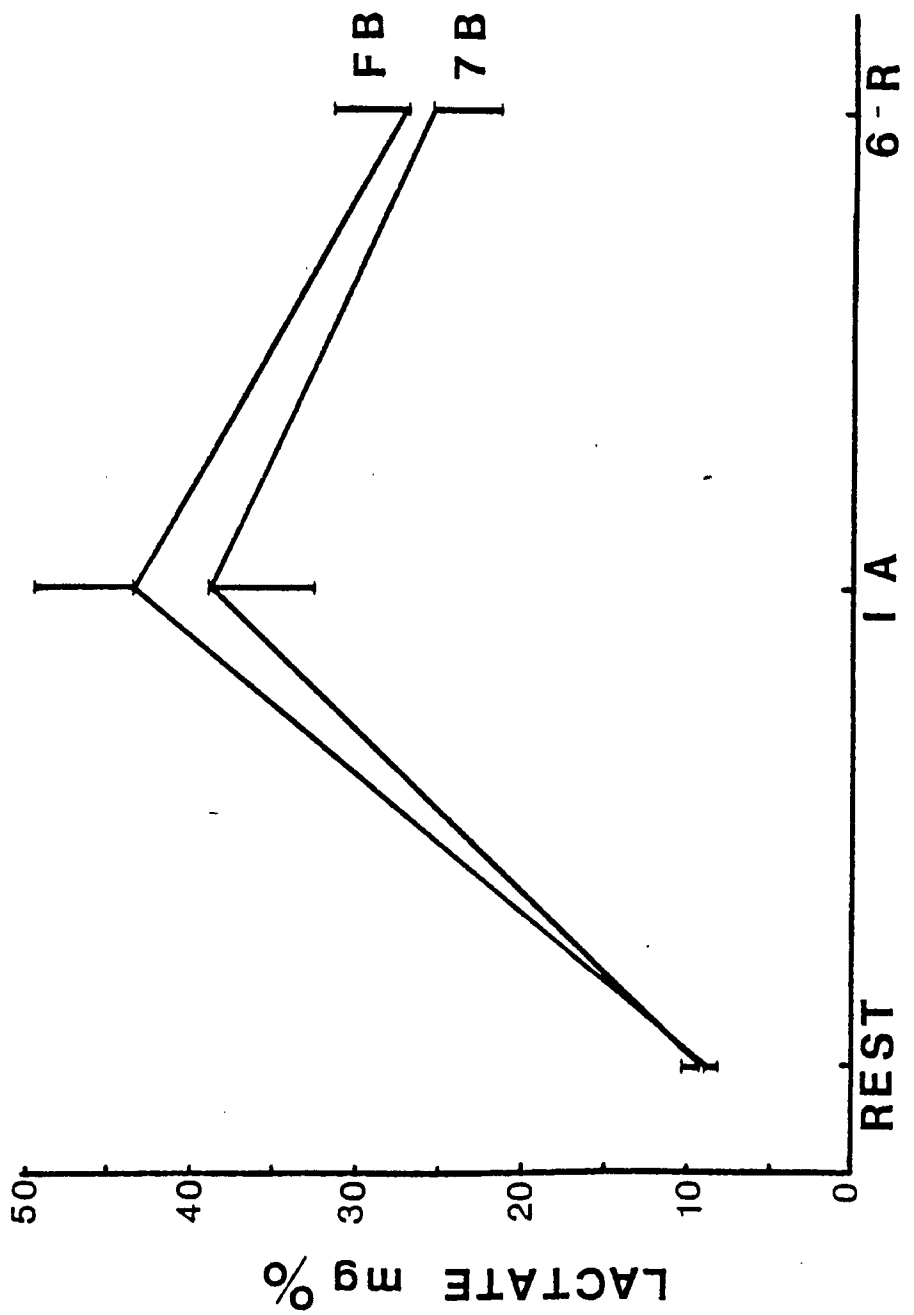


FIGURE 3. EFFECT OF FREE BREATHING (FB), AND 7-STROKE BREATHING (7B) ON VENOUS BLOOD LACTATE CONCENTRATIONS PRIOR TO THE SWIM (REST), IMMEDIATELY AFTER THE EXERCISE SESSION (IA), AND AT THE 6TH MINUTE OF RECOVERY (6-R).

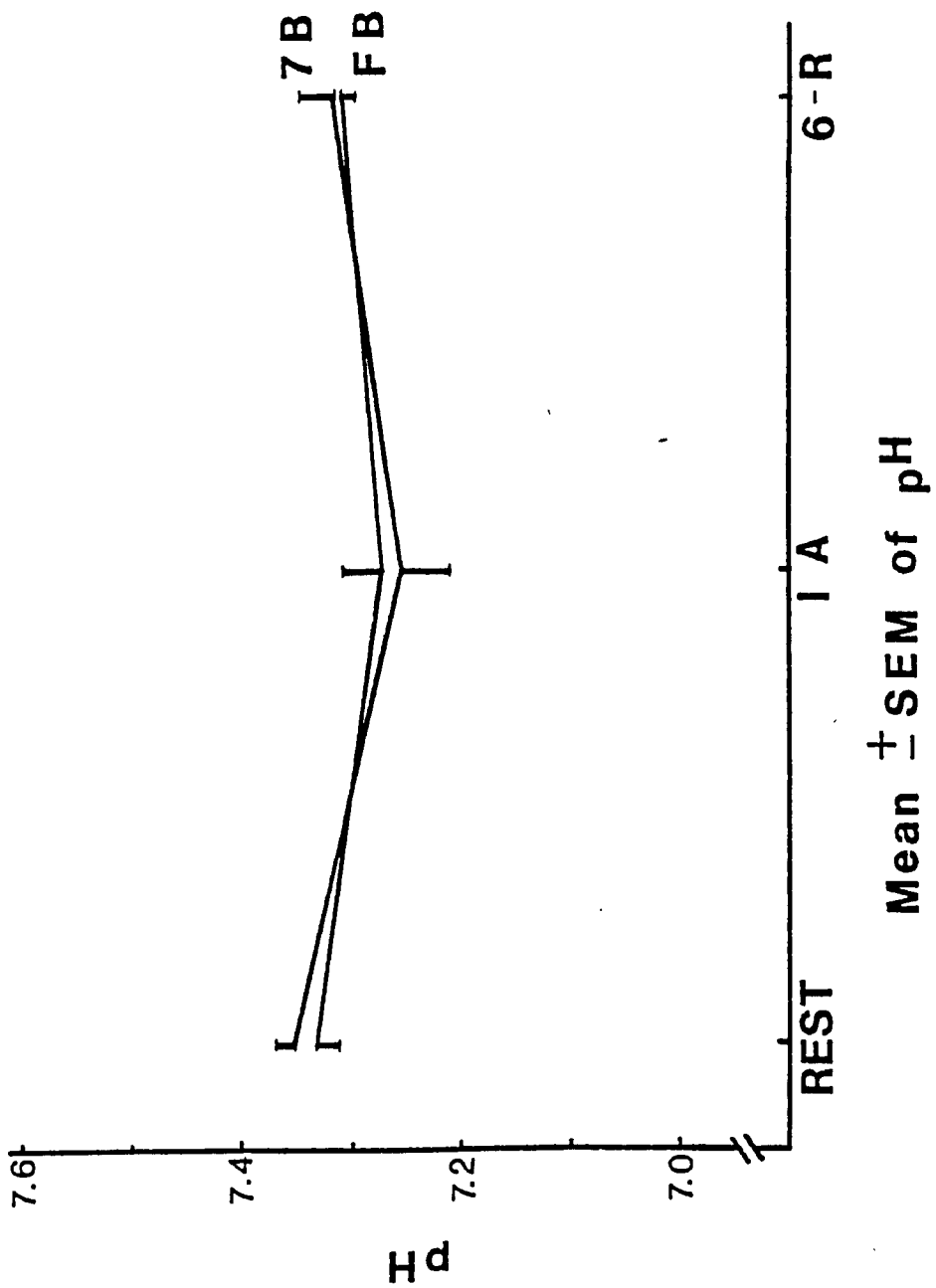


FIGURE 4. EFFECT OF FREE BREATHING (FB), AND 7-STROKE BREATHING (7B) ON VENOUS BLOOD pH VALUES PRIOR TO THE SWIM (REST), IMMEDIATELY AFTER THE EXERCISE SESSION (IA), AND AT THE 6TH MINUTE OF RECOVERY (6-R).

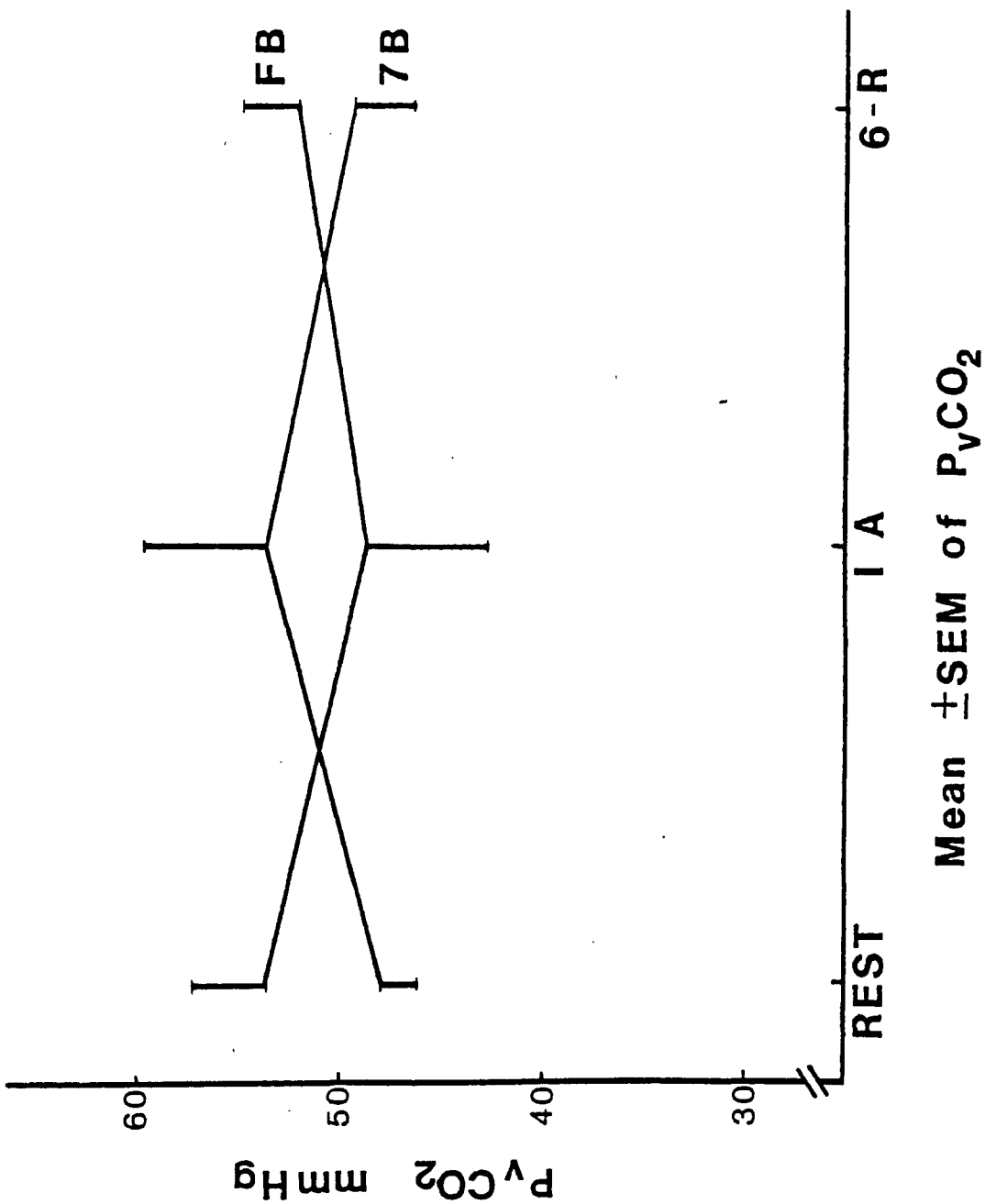


FIGURE 5. EFFECT OF FREE BREATHING (FB), AND 7-STROKE BREATHING (7B) ON VENOUS BLOOD $P_vCO_2$ VALUES PRIOR TO THE SWIM (REST), IMMEDIATELY AFTER THE EXERCISE SESSION (IA), AND AT THE 6TH MINUTE OF RECOVERY (6-R).

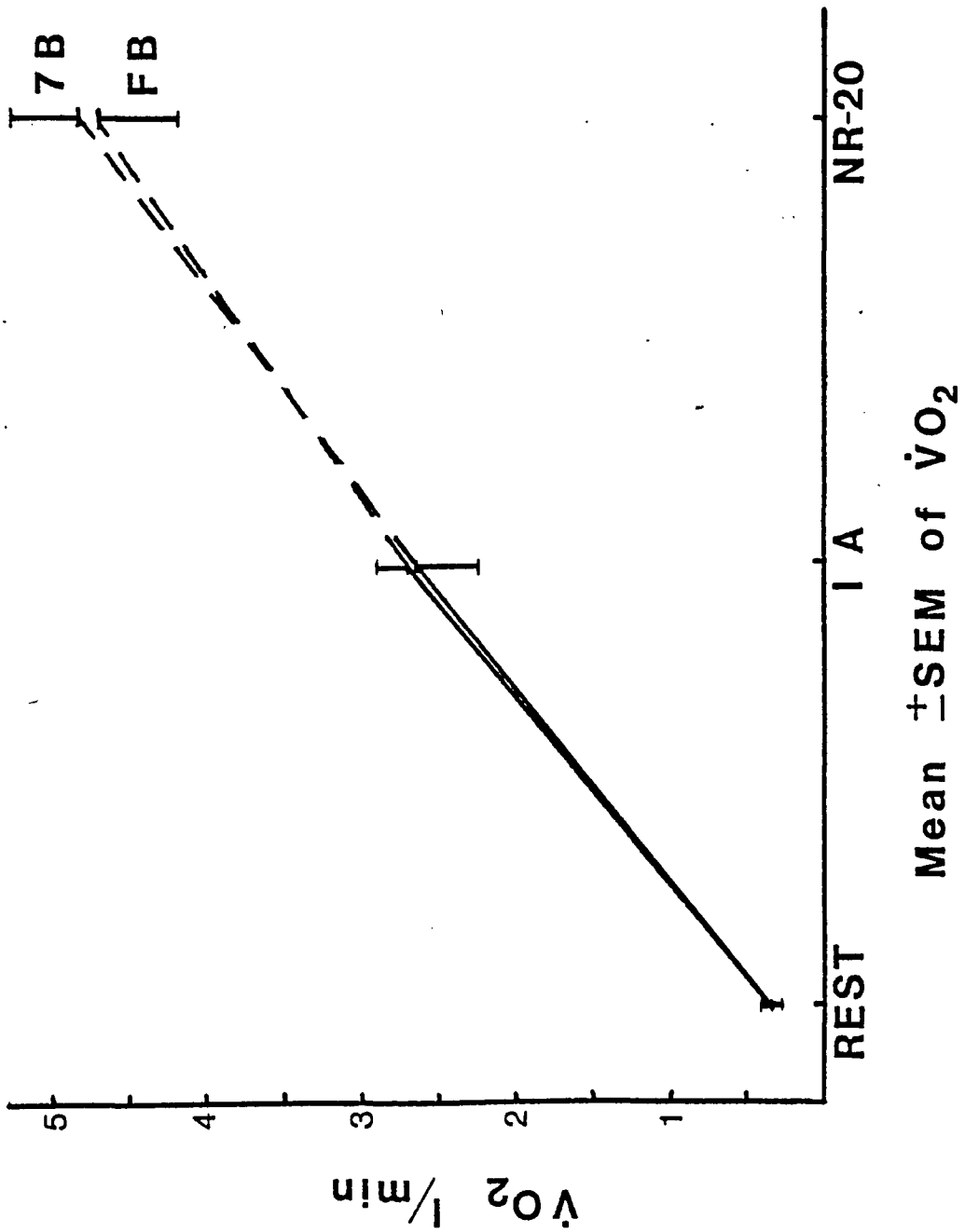


FIGURE 6. EFFECT OF FREE BREATHING (FB), AND 7-STROKE BREATHING (7B) ON $\dot{V}O_2$ PRIOR TO THE SWIM (REST), IMMEDIATELY AFTER THE EXERCISE SESSION (IA), AND DURING THE FIRST 20 MINUTES OF NET RECOVERY $O_2$ UPTAKE (NR-20).

The results of analysis of variance can be found in Appendix H, and are summarized in Table 4, A and B. Significant changes ($p < .05$) due to the exercise protocols were found only in GL, LA, and $\dot{V}O_2$ among the three different times measured. However, no significant differences were observed in GL, LA, pH, $P_VCO_2$, and $O_2$ uptake between the FB and 7B swims.

The $O_2$ uptake during the recovery periods is shown in Table 5. The recovery $\dot{V}O_2$ patterns did not show any difference between the 2 breathing protocols (Figure 7). The $O_2$ extraction ($F_IO_2 - F_EO_2$), and the $CO_2$ production ($F_ECO_2 - F_ICO_2$) during the first 30 seconds of recovery are shown in Table 6, and are presented in Figure 8. No difference was found in $O_2$ extraction, but significant difference ($p < .05$) was found in $CO_2$ production between the 2 swim protocols.

TABLE 4

SUMMARY OF THE RESULTS FROM ANOVA

(A) TREATMENT EFFECTS BETWEEN THE FREE AND 7-STROKE BREATHING PATTERNS

| | IA | 6-R | Interaction |
|---|---|---|---|
| Glucose | N.S. | N.S. | NO |
| Lactate | N.S. | N.S. | NO |
| pH | N.S. | N.S. | NO |
| $P_vCO_2$ | N.S. | N.S. | NO |
| $\dot{V}O_2$ | N.S. | N.S.* | NO |

N.S. = Not statistically significant at the 0.05 level.

* Net recovery $O_2$ uptake for 20 minutes was used instead of the $\dot{V}O_2$ at the 6th minute of recovery.

(B) EFFECTS OF THE EXERCISE PROTOCOL ON THE 3 DIFFERENT TIMES MEASURED (DUNCAN'S MULTIPLE RANGE TEST)*

| | Rest - IA | Rest - 6-R | IA - 6-R |
|---|---|---|---|
| Glucose | $P < .05$ | N.S. | $P < .05$ |
| Lactate | $P < .05$ | $P < .05$ | $P < .05$ |
| pH | N.S. | N.S. | N.S. |
| $P_vCO_2$ | N.S. | N.S. | N.S. |
| $\dot{V}O_2$ | $P < .05$ | | |

N.S. = Not statistically significant at the 0.05 level.

* Since no interaction was found in ANOVA, the values of the FB, and the 7B were pooled together for the Duncan's test.

TABLE 5

MEANS AND STANDARD ERROR OF MEANS OF $\dot{V}O_2$ (1/min) DURING RECOVERY TIME PERIODS

| | Recovery Time Periods (Minutes) | | | | | | |
|---|---|---|---|---|---|---|---|
| | 0-0.5 | 0.5-3.0 | 3.0-6.0 | 6.0-12 | 12-18 | 18-20 | Total |
| Free Breathing | 2.73 ±0.21 | 0.83 ±0.11 | 0.56 ±0.03 | 0.48 ±0.02 | 0.43 ±0.02 | 0.42 ±0.02 | 5.45 |
| 7-Stroke Breathing | 2.68 ±0.48 | 0.80 ±0.08 | 0.55 ±0.03 | 0.49 ±0.03 | 0.44 ±0.03 | 0.40 ±0.03 | 5.36 |

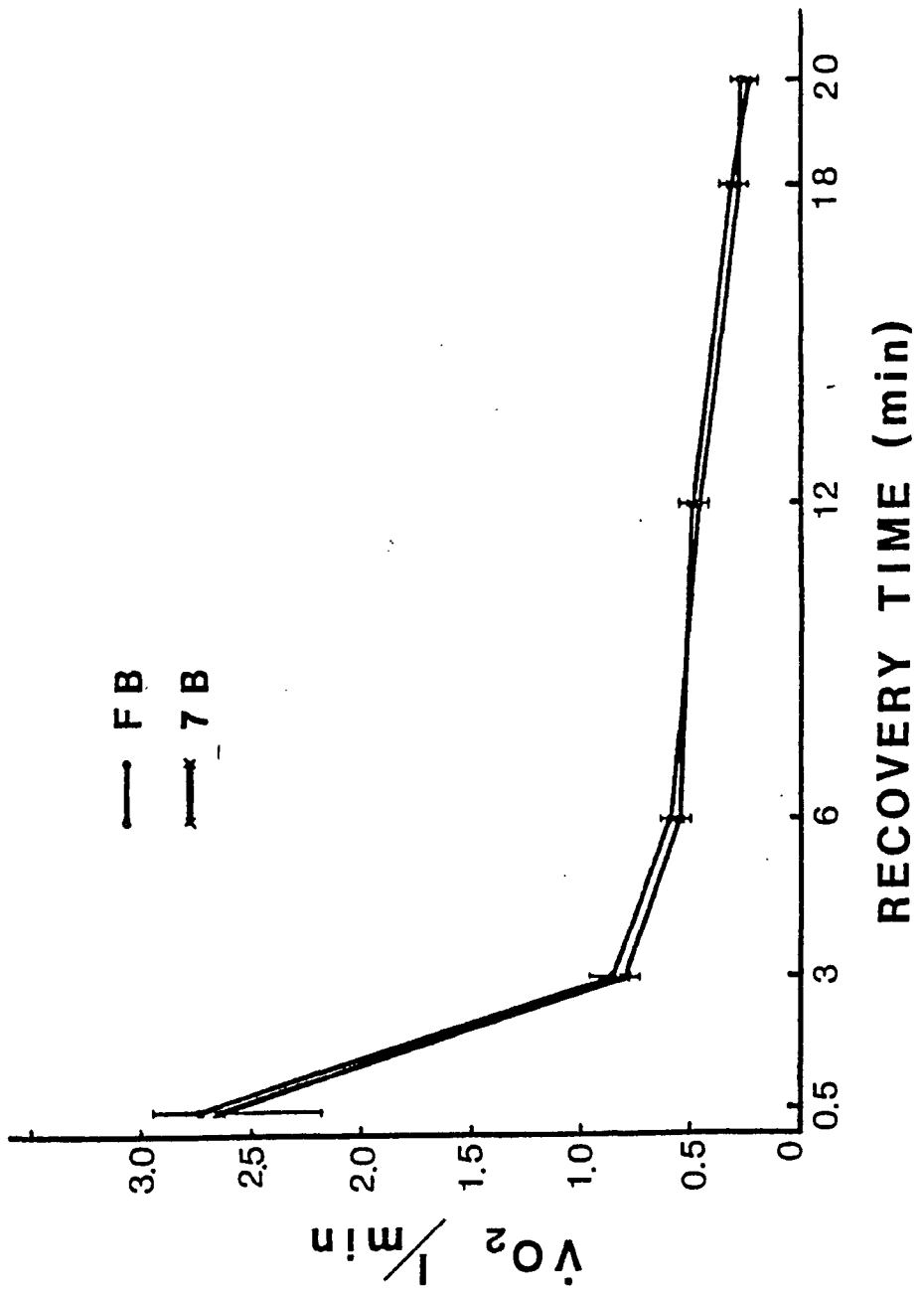


FIGURE 7. RECOVERY $O_2$ UPTAKE ($\bar{X} \pm S\bar{x}$) PATTERN OF THE FREE BREATHING (FB), AND THE 7-STROKE BREATHING (7B) DURING THE FIRST 20 MINUTES OF RECOVERY.

TABLE 6

$O_2$ EXTRACTION AND $CO_2$ PRODUCTION DURING THE FIRST 30 SECONDS OF RECOVERY

| Subject | Free Breathing | | 7-Stroke Breathing | |
|---|---|---|---|---|
| | $O_2$ Extraction (%) | $CO_2$ Production (%) | $O_2$ Extraction (%) | $CO_2$ Production (%) |
| J.S. | 4.12 | 3.73 | 3.94 | 3.80 |
| B.M. | 3.78 | 3.40 | 4.29 | 4.32 |
| E.J. | 3.50 | 3.22 | 3.71 | 3.62 |
| R.B. | 3.99 | 3.52 | 3.84 | 3.79 |
| R.H. | 3.93 | 3.47 | 4.18 | 3.73 |
| T.M. | 4.55 | 3.83 | 4.21 | 3.90 |
| $\bar{X} \pm S\bar{x}$ | 3.98 ± 0.17 | 3.53* ± 0.09 | 4.03 ± 0.09 | 3.86* ± 0.09 |

* The difference was statistically significant at the 0.05 level.

$O_2$ Extraction (%) = $(F_IO_2 - F_EO_2) \times 100$.

$CO_2$ Production (%) = $(F_ECO_2 - F_ICO_2) \times 100$.

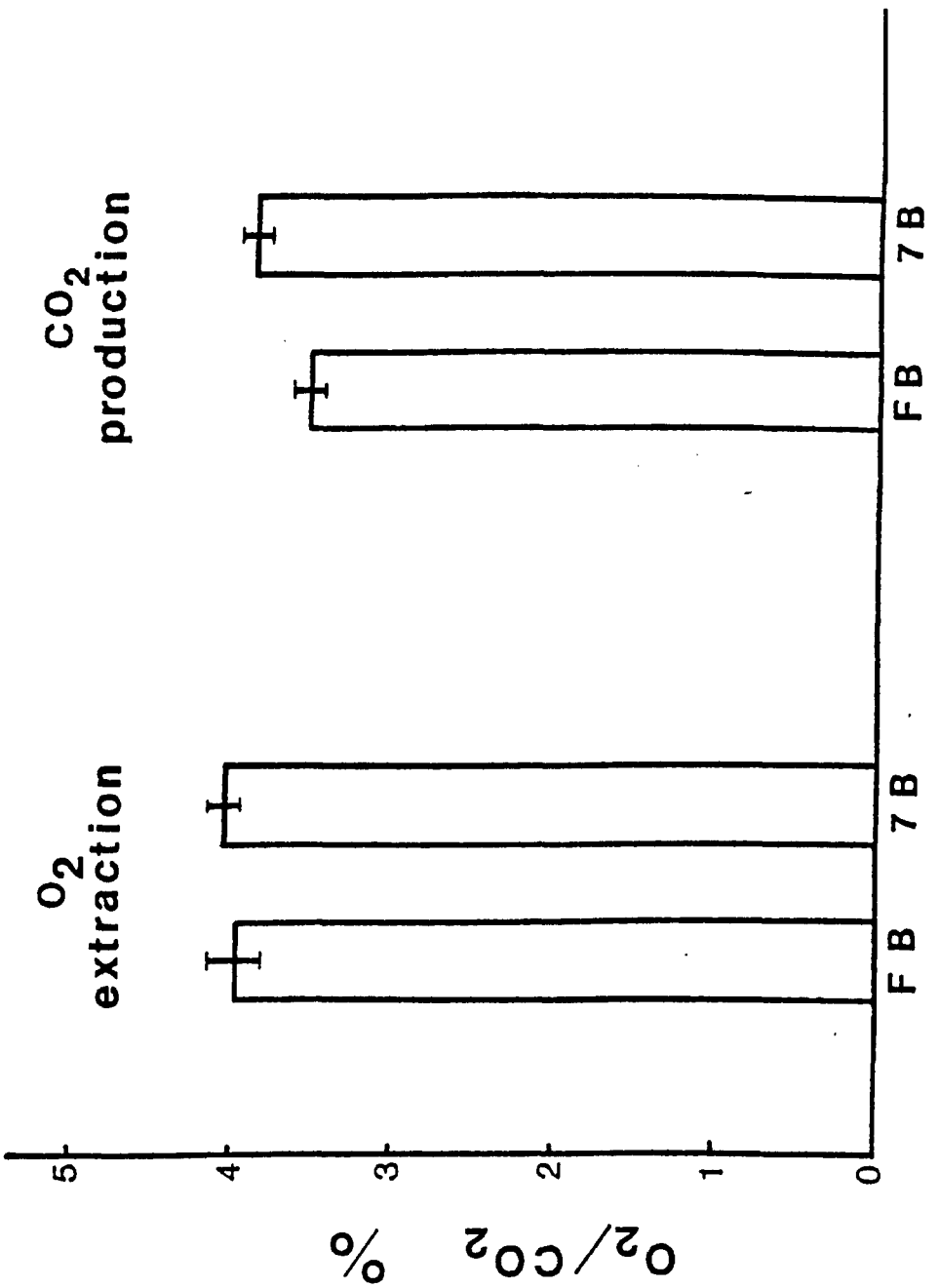


FIGURE 8. EFFECT OF FREE BREATHING (FB), AND 7-STROKE BREATHING (7B) ON $O_2$ EXTRACTION AND $CO_2$ PRODUCTION DURING THE FIRST 30 SECONDS OF RECOVERY.

# CHAPTER VI

## DISCUSSION

The changes of GL, LA, pH, $P_vCO_2$, and $\dot{V}O_2$ values of this study can be said to be caused by the training protocol, and not due to uncontrolled fluctuation of these physiological values as Table 2 indicates. In addition, swimming at 85% of each individual's best 200-yard time was an adequate, but not the optimum, metabolic stress to the swimmers (Table 4-B).

From the GL, LA, pH, $P_vCO_2$, and recovery $O_2$ uptake values of this study, the 7B swim did not show a greater stress on the glycolytic metabolism than the FB swim. Thus, the previously believed theory (Counsilman, 1977; Shaw, 1979; Bonen, 1979) that "hypoxic training" can induce a greater stress than can normal training on the glycolytic metabolism, cannot be supported. If the above theory were true, the changes of the LA, the pH, as well as the recovery $O_2$ uptake after the 7B swim would be significantly greater than after the FB swim. The literature has shown that blood LA (Gollnick and Hermansen, 1973), pH (Mader, et al., 1978), and recovery $O_2$ uptake (Hagberg, et al., 1980) were very closely related to glycolytic metabolism during exercise.

The lack of difference in the recovery $O_2$ uptake between the 2 swim protocols suggests that the $\dot{V}O_2$ during the swim also did not differ. This seems to comply with the reports (Craig, 1979; Dicker, et al., 1980) that at a given intensity there was no difference in $\dot{V}O_2$ during exercise using different breathing patterns. The fact that the percentage of $O_2$ extraction during less frequent breathing in swimming was greatly increased (Sparks, 1973; Hsieh, 1979; Dicker, et al., 1980) helps to

explain the reason for similar $\dot{V}O_2$ values. This increased $O_2$ extraction compensates for the loss of $O_2$ availability due to hypoventilation during the 7B swim.

Although a gas sample was not obtained during the swims in this study, the expired gas collected during the first 30 seconds of recovery can still give indications of the gas exchange values during exercise (di Prampero, et al., 1973; Léger, et al., 1980). Even though the $O_2$ extraction during this period was not significantly different between the 2 swim protocols, a trend of greater $O_2$ extraction during the 7B swim can be observed (Figure 8). However, the significantly greater $CO_2$ production after the 7B swim ($P < .05$) seems to confirm the reports that controlled breathing caused a significantly higher $P_ACO_2$ during exercise, but did not affect $P_AO_2$ (Craig, 1979; Dicker, et al., 1980). In spite of the fact that $P_VCO_2$ was not significantly different between the 2 protocols, a hypercapnic trend can also be observed in the greater $P_VCO_2$ value after the 7B swim (Figure 5). Physiologically, hypercapnia may be able to reduce lactate production and enhance fat metabolism during exercise, as was hypothesized by Graham, et al., (1980). However, this hypothesis has not yet been proven. Since CBT cannot cause hypoxia, the term "hypoxic training" should not be used (Craig, 1979). A clear distinction should also be made between CBT and training using reduced $F_IO_2$.

The significant changes in GL, LA, and $\dot{V}O_2$ immediately after the exercise session were expected. The increased glucose output from the liver to the blood during exercise (Wahren, et al., 1971; Hultman and Nilsson, 1975) was either used for muscle glycogen biosynthesis (Lehninger, 1977; Newsholme and Start, 1979), or transported back to the liver as exercise ceased. Thus, the glucose concentration in the

blood was still elevated shortly after exercise, and then fell back to the resting value at the 6th minute of recovery. The peak LA value was observed immediately following the training session. The time of this occurrence seems to be similar to that found in the report by Diamant, et al. (1968). Hermansen, et al. (1973), and Svarc, et al. (1973) have reported that the skeletal muscles of well trained athletes have a greater ability to convert LA to pyruvate which will then enter the oxidative energy system, than those of non-trained subjects. Since the subjects in this study were well trained (the mean $\dot{V}O_2$ max. value was 62.84 m./m/kg, ±2.50), it is likely that they were able to metabolize the LA produced during exercise at a faster rate, and thus a peak value could not be found at the 6th minute of recovery. Also, Molé and Holloszy (1970) reported that well trained rats had a greater capacity to oxidize fats as an energy source and decrease glycolytic rate and lactate accumulation. This evidence may explain the relatively low lactate values (Appendix G-2) observed in this study. However, this low lactate value may also indicate that using 85% of the individual's best 200-yard time was not the same as 85% of the individual's $\dot{V}O_2$ max. Using about 86% of the subject's $\dot{V}O_2$ max. during exercise, di Prampero, et al. (1978) have reported an appreciable accumulation of LA in the blood. At any rate, using the percentage of a swimmers $\dot{V}O_2$ max. as an indicator of swimming intensity in his daily training sessions has not been practical, and coaches prefer using the best time values.

In contrast to that of the 7B swim, the $P_vCO_2$ after the FB was lowered, similar to the result in a report by Svarc, et al. (1973). They reported that the $PCO_2$ in the arterialized capillary blood dropped significantly during interval training in swimmers. However, other

reports (Magel, 1966; Dixon, et al., 1971) have shown that normal swimming tended to cause an increase in $P_ACO_2$ whereas running did not. The difference in the experimental protocol may have caused this inconsistent result. Even though the subjects of this study were considered to be homogeneous, the results of LA (Appendix G-2), and $P_VCO_2$ (Appendix G-4) reflected a great difference in response to the imposed stress among the individuals. This individual difference in response, and the small sample size (N = 6) seem to be the limitations of this study.

The conclusion by Patton, et al. (1979) that "hypoxic training" improved 100-yard swimming performance due to the improved "lactacid" part of anaerobic capacity is doubtful. The data of this study have given rise to speculation that this improvement may not be because of physiological gain, but because of biomechanical and psychological reasons. It has been proven that swimmers who breath-hold during swimming have less resistance than if they were breathing (Hay, 1978). Furthermore, swimmers trained with controlled breathing may believe that they are better prepared because they are able to swim without breathing for the last several meters. Both biomechanical efficiency and psychological readiness are as important as physiological aspects during a close race.

It is possible to hypothesize further that a 9-stroke breathing pattern, instead of a 7-stroke pattern, will result in greater glycolytic responses in swimmers than will a normal breathing pattern. However, a 9-stroke breathing pattern was believed to be very difficult to maintain during swimming at a speed and distance which can be training effective. Also, this would be similar to breathholding training for diving purposes. In fact, Michael, et al., (1964) has shown that man generally does not adapt to breathholding like other diving mammals. Furthermore, according to Holmér (1978), the strain imposed upon the respiratory system during

swimming does not interfere with gas exchange, and it is unlikely that respiration is a limiting factor in performance. A similar point of view was also expressed by Dempsey (1980) in a symposium - Limiting Factors to Exercise: Respiratory Factors. In order to make the training more effective, the key seems to be the work intensity, and not the breathing pattern. Houston (1978) has suggested that unless "hypoxic training" is performed at high speed, there will not be a sufficient challenge to the glycolytic rate. However, the muscles may already be hypoxic under intensive exercise. The idea of inducing hypoxia, such as "hypoxic training", is questionable regarding its further effect (Holloszy, 1973; Curran, 1977). At the moment, it is not clear whether CBT used under maximal exercise, will have greater effects on the swimmers than will normal breathing. It is also uncertain whether the effects of a 9-stroke breathing pattern should be tested on swimmers. Further investigation is necessary to answer these questions.

From the data of this study and the literature, it is clear that during controlled breathing swimming, the minute ventilation is decreased, the percent $O_2$ extraction is increased, but the same level of $\dot{V}O_2$ is maintained. Also, the accumulated $CO_2$ due to the hypoventilation will cause both $P_ACO_2$, and $P_VCO_2$ to be elevated, and thus will slightly affect the pH value in the body fluid. However, these temporary differences in physiological parameters do not seem to alter the total metabolic function of the body. The key to stressing the glycolytic metabolism more effectively appears to be connected with swimming intensity rather than the breathing pattern used. Nevertheless, controlled breathing training should not be dropped without first considering the biomechanical and psychological aspects involved, as well as the possible favourable hypercapnic effects provided by this type of training.

# CHAPTER VII

# SUMMARY AND CONCLUSIONS

## Purpose of Study

This study was designed to compare the acute effects of controlled breathing training (CBT) to free breathing training in swimming on the glycolytic metabolism, attempting to reveal some of the physiological consequences of the so-called "hypoxic training" in swimming.

## Methods

Six well trained male swimmers familiar with CBT were the subjects. The diet and the physical activity of these subjects were controlled for 3 days prior to the study. The experiment was conducted under 2 separate swimming exercise sessions using 2 different breathing patterns: free breathing (FB), and 7-stroke breathing (7B). The swimming intensity was determined to be 80-88% of each individual's best 200-yard time. The total work time for each session was approximately 30 minutes.

Venous blood samples were drawn prior to the swim (Rest), immediately after the exercise session (IA), and at the 6th minute of recovery (6-R). Analyses for glucose (GL, o-toluidine reagent method), lactate (LA, lactate dehydrogenase method), and pH and $P_vCO_2$ (ABL-2 Blood Gas Analyzer) were done. The resting $O_2$ uptake as well as the recovery $O_2$ uptake (20 minutes) were also measured and calculated for each session using the open circuit spirometry method. A correlated t-test as well as a Pearson Product-Moment correlation coefficient, and repeated measures of analysis of variance (ANOVA) on 2 factors were used for statistical analysis.

## Results

Between the 2 swim protocols, no significant difference could be found among the variables measured, except for the $CO_2$ production. The $CO_2$ produced during the first 30 seconds of recovery after 7B was greater than after FB ($P < .05$). Significant changes caused by the exercise session were found in the GL ($P < .05$), LA ($P < .05$), and $\dot{V}O_2$ ($P < .05$) values.

## Discussion

The results of this study tend to agree with the previous findings that CBT caused hypercapnia, and not hypoxia. However, the theory that CBT in swimming caused more stress on the glycolytic metabolism than did the normal breathing training, was not supported. It appears that the key to stressing glycolytic metabolism effectively is the work intensity and not the breathing pattern used. Also, if "hypoxic training" can improve the 100-yard swim performance, it is more likely to be due to the biomechanical and psychological benefits, and not due to physiological reasons.

## Conclusions

This study failed to reject the null hypothesis which was made before the experiment. It was concluded from the results that the acute effects of CBT at 85% of the swimmer's best time produced no more stress on glycolytic metabolism than did the normal breathing training in well trained male swimmers.

Recommendations for Further Study

1) A swimming intensity of much greater than 85% of the swimmer's best time, or a $\dot{V}O_2$ value of close to the maximum should be used if a comparison of the metabolic responses to CBT and normal breathing training was to be done again.

2) The effect of a much more controlled breathing pattern, such as 9-stroke breathing, should also be studied.

3) Since hypercapnia is associated with CBT, its acute and chronic effects on energy metabolism during exercise should be closely examined.

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# APPENDIX A

## CONSENT FORM FOR TEST SUBJECTS

I hereby consent, voluntarily, to act as a subject in the research project outlined below. I realize that I am free to withdraw from the project at any time.

The testing will involve two swimming exercise sessions using two different breathing patterns. A venous blood sample will be taken from each subject just prior to, 10 seconds after, and 6 minutes after each exercise session. These blood samples will be examined for glucose and lactate concentrations and analyzed for blood gases, in order to determine the metabolic consequences of Hypoxic Training. Oxygen consumption at rest and during recovery will also be determined.

I am aware of the fact that this study is being conducted under the supervision of a committee, which includes a medical practitioner of this community. I have been assured that there is very little risk involved in participating in the testing.

I have been informed by the Faculty of Human Kinetics, University of Windsor, that only my initials will appear in any presentation or publication of this research study. Also, I will have access to all data which pertain to my personal involvement in the project.

I hereby affirm that I have read and understood the contents of this form, and do agree to participate as a subject in the above described research study.

DATE: _____, 1980

(Please Print) (Signature)

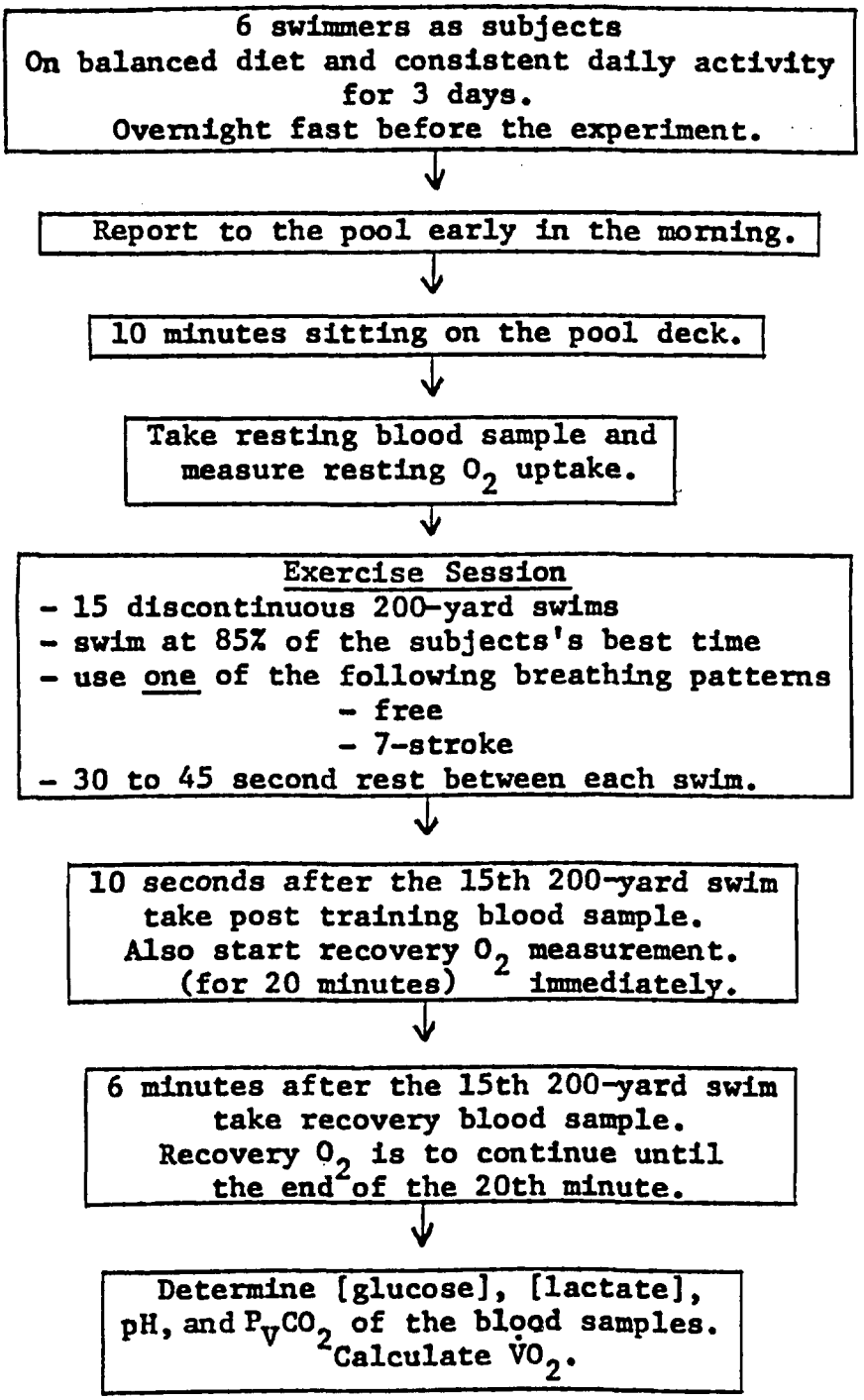
Subject: _____ _____

Parent or Guardian: _____ _____

Witness: _____ _____

# APPENDIX B

## EXPERIMENTAL PROTOCOL



APPENDIX C

Diet and Activity Record of _____

| | Food | Activity |
|---|---|---|
| Day 1<br>June ____<br>or<br>July ____ | Breakfast: | Morning: |
| | Lunch: | Afternoon: |
| | Supper: | Evening: |
| Day 2<br>June ____<br>or<br>July ____ | Breakfast: | Morning: |
| | Lunch: | Afternoon: |
| | Supper: | Evening: |

Continued ...

<table>
<tr><td rowspan="4">Day 3<br>June _____<br>or<br>July _____</td><td>Food</td><td>Activity</td></tr>
<tr><td>Breakfast:</td><td>Morning:</td></tr>
<tr><td>Lunch:</td><td>Afternoon:</td></tr>
<tr><td>Supper:</td><td>Evening:</td></tr>
<tr><td></td><td colspan="2">No food after 10:00 p.m.</td></tr>
<tr><td>Day 4<br>June _____<br>or<br>July _____</td><td colspan="2">Report to the Human Kinetics pool at 6:00 a.m. <u>without</u> having had any breakfast. Please bring this record sheet with you. Thank you.</td></tr>
</table>

# APPENDIX D

## LACTATE DETERMINATION AND CALIBRATION CURVE

The following material was modified in part according to Sigma Technical Bulletin, No. 826-UV, October 1978.

### Principle

The enzyme, lactate dehydrogenase (LDH) catalyzes the following reversible reaction:

$$\text{Pyruvic acid} + \underset{(\text{high } A_{340})}{\text{NADH}} \xrightleftharpoons{\text{LDH}} \text{Lactic acid} + \underset{(\text{low } A_{340})}{\text{NAD}}$$

To measure lactic acid, the reaction is carried from right to left with excess NAD. In order to force the reaction to completion in this direction, it is necessary to trap formed pyruvic acid with hydrazine. The increased absorbance at 340 nm due to NADH formation becomes a measure of lactic acid originally present.

### Reagents

A. Lactic dehydrogenase - contains LDH suspension isolated from beef heart in ammonium sulfate. Approximately 1,000 units/ml when prepared.

B. Glycine buffer - contains glycine and hydrazine, pH 9.2.

C. NAD preweighted vial - vial contains nicotinamide adenine dinucleotide, grade III, 10 mg.

D. Lactic acid standard solution - contains L(+) lactic acid, 0.40 mg/ml (4.44 m mol/l).

E. Lactic acid diluted standard - dilute 1.0 ml of reagent D to 5.0 ml with distilled water.

F. Perchloric acid, 8% (w/v) - dilute 7 ml of 70% (w/v) perchloric acid to 100 ml with distilled water.

Procedure (for multiple assays)

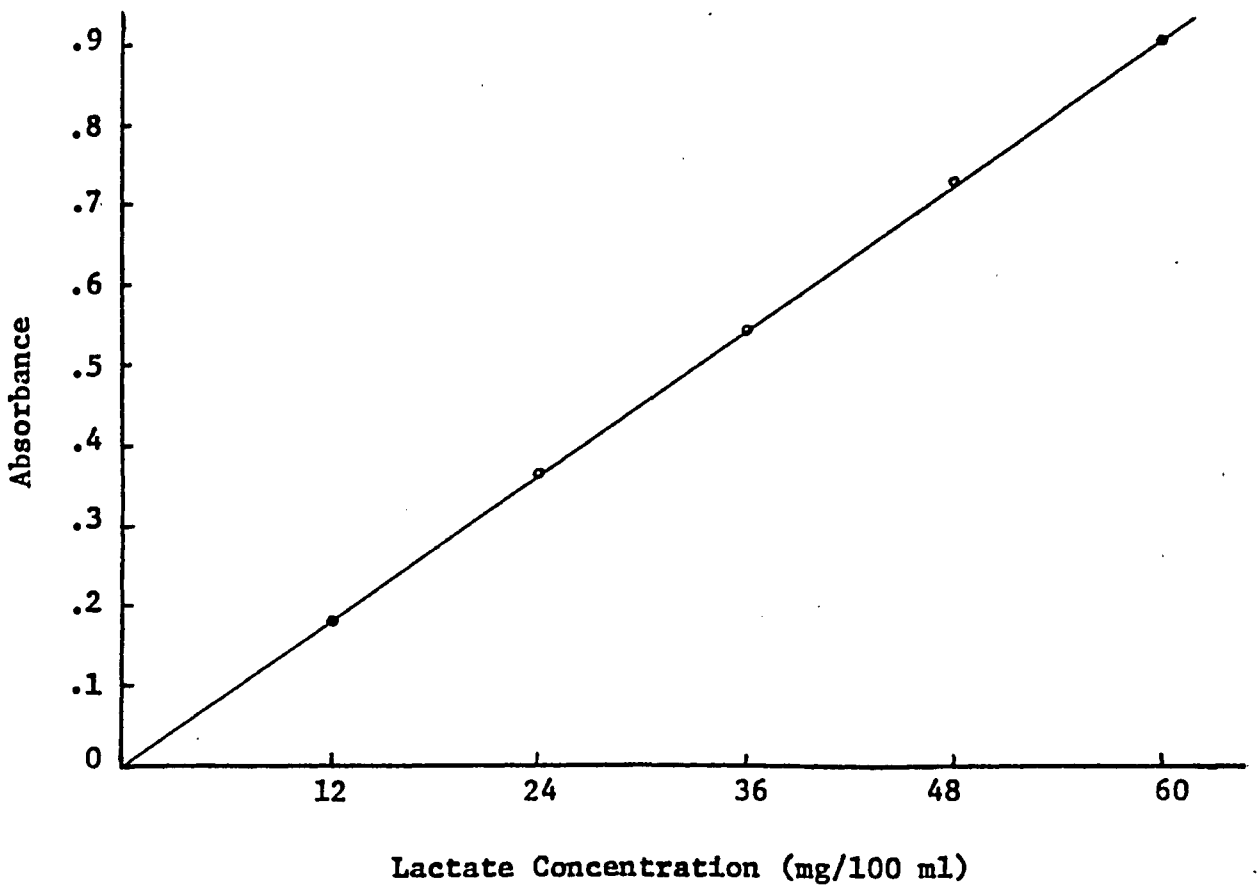
1. Determine the number of NAD vials with the following formula:
   Number of NAD vials required = $\frac{\text{number of samples to be assayed} + 1}{2}$
   If this is not a whole number, add 1/2 to it for actual number of vials required.
2. Pipet into each of the NAD vials with 2.0 ml glycine buffer, 4.0 ml water, and 0.1 ml of lactic dehydrogenase. Invert several times to dissolve NAD.
3. Combine the solutions into a flask. Mix well.
4. Label an appropriate number of tubes BLANK, TEST 1, TEST 2, etc. Pipet into each tube 2.8 ml of the mixture from step 3 above.
5. To the BLANK tube, add 0.2 ml of 8% perchloric acid, and to the TEST tubes add 0.2 ml of supernatant collected earlier for lactic acid determination. Gently shake both tubes to mix.
6. Incubate both tubes approximately 30 minutes at 37°C, or 45 minutes at 25°C.
7. Read and record absorbance of TEST at 340 nm (ultraviolet light) using BLANK as reference.
8. Calculations:
   Lactic acid concentration can be calculated directly from the absorbance readings if a narrow-bandwidth spectrophotometer is employed, such as the Beckman DB-G. Blood lactic acid (mg/100 ml) = Absorbance at 340 x 65.1.

## Lactic Acid Calibration Curve

**Absorbance points for lactic acid calibration curve (each point represents 3 determinations).**

| Lactate (mg/100 ml) | 12 | 24 | 36 | 48 | 60 |
|---|---|---|---|---|---|
| 1 | .184 | .361 | .563 | .740 | .912 |
| 2 | .182 | .375 | .545 | .721 | .897 |
| 3 | .179 | .359 | .524 | .726 | .915 |
| $\overline{X} \pm S\overline{X}$ | .182 ±.002 | .365 ±.005 | .544 ±.012 | .729 ±.006 | .908 ±.006 |

## Lactate Calibration Curve



# APPENDIX E

## GLUCOSE DETERMINATION AND CALIBRATION CURVE

The following material was modified in part according to Sigma Technical Bulletin, No. 635, November 1978.

### Principle

In the presence of heat and acid, o-toluidine reacts readily with glucose to form a colored complex.

$$\underset{\text{(colorless)}}{\text{o-toluidine}} + \text{glucose} \xrightarrow[\text{heat}]{\text{acid}} \underset{\text{(blue-green)}}{\text{colored complex}}$$

The intensity of the color found is measured with a photoelectric spectrophotometer at 620-650 nm, and is proportional to the glucose concentration.

### Reagents

A. o-toluidine reagent - contains o-toluidine, 6% (w/v), in glacial acetic acid with thiourea added as a stabilizer.

B. Glucose standard solutions -

(1) Standarized at 10.0 mg/ml (1000 mg/100 ml, or 55.5 m mol/l) with benzoic acid added as a preservative.

(2) Standarized at 1.0 mg/ml (100 mg/100 ml, or 5.5 m mol/l) with benzoic acid added as a preservative.

### Procedure (without deproteinization)

1. Label 3 or more test tubes BLANK, STANDARD, TEST 1, TEST 2, etc. To BLANK add 0.1 ml of distilled water, to STANDARD add 0.1 ml of glucose standard solution (Reagent B-2, 100 mg/100 ml) and to TEST add 0.1 ml of plasma prepared earlier.

2. To each tube, add 5.0 ml of o-toluidine reagent. Mix well by lateral shaking.
3. Put all tubes into a vigorously boiling water bath for exactly 10 minutes.
4. Quickly remove all tubes and cool to room temperature by placing in tap water for approximately 3 minutes.
5. Transfer contents of tubes to cuvets and read absorbance of STANDARD and TEST at 635 ± 15 nm (visual light), using BLANK as reference. Complete readings within 30 minutes.
6. Calculation:

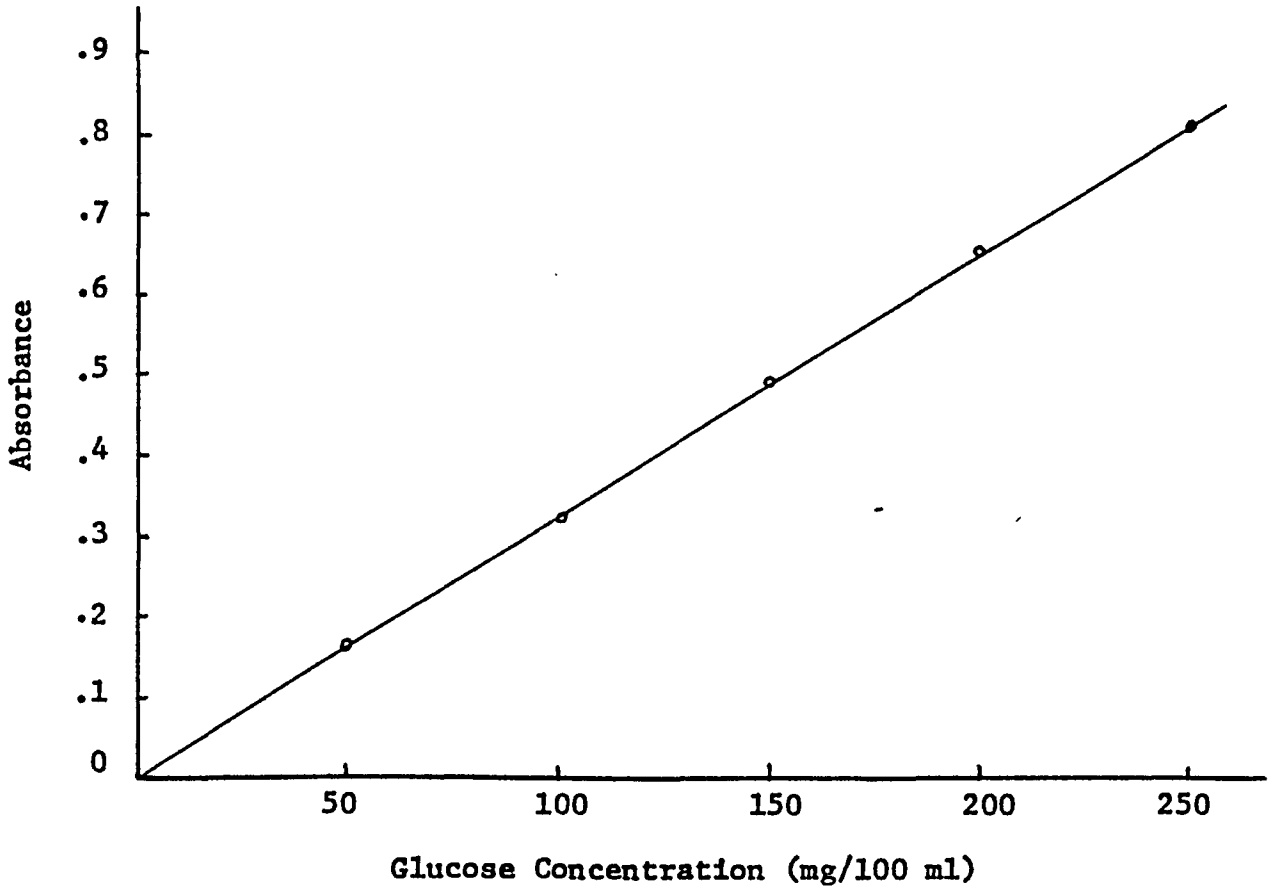
$$\text{glucose (mg/100 ml)} = \frac{\text{Absorbance of TEST}}{\text{Absorbance of STANDARD}} \times 100$$

Glucose Calibration Curve

Absorbance points for glucose calibration curve (each point represents 3 determinations)

| Glucose (mg/100 ml) | 50 | 100 | 150 | 200 | 250 |
|---|---|---|---|---|---|
| 1 | .157 | .315 | .489 | .642 | .813 |
| 2 | .165 | .317 | .483 | .631 | .792 |
| 3 | .161 | .328 | .480 | .668 | .801 |
| $\bar{X} \pm S\bar{X}$ | .161 ±.002 | .320 ±.004 | .484 ±.002 | .648 ±.011 | .802 ±.006 |

## Glucose Calibration Curve



# APPENDIX F

## OXYGEN UPTAKE ($\dot{V}O_2$) CALCULATION

**Formula**

$$\dot{V}O_2 = \dot{V}_E \text{ STPD } [(1 - F_EO_2 - F_ECO_2) \times .2648 - F_EO_2]$$

Because, $\dot{V}O_2 = \dot{V}_IF_IO_2 - \dot{V}_EF_EO_2$, $\dot{V}_IF_IN_2 = \dot{V}_EF_EN_2$

**Illustrated Example**

Subject: T.M.

Body Weight: 78.0 kg

Temperature of gas: 26.5°C

Barometric pressure: 748 mm Hg

STPD factor = .862

$\dot{V}_E$ = 20.65 l/min (at rest)

$\dot{V}_E$ STPD = 20.65 x .862 = 17.80 l/min

$F_EO_2$ = 18.50%

$F_ECO_2$ = 2.52%

$F_EN_2$ = 78.98%

Nitrogen factor = $\frac{F_IO_2}{F_IN_2} = \frac{20.93\%}{79.04\%} = .2648$

Actual $F_IO_2$ = 78.98 x .2648 = 20.91%

$O_2$ consumed = 20.91 - 18.50 = 2.41%

Absolute $O_2$ uptake = 17.80 x .0241 = .43 l/min

Relative $O_2$ uptake = $\frac{430 \text{ (ml/min)}}{78}$ = 5.51 ml/min/kg.

# APPENDIX G

## RAW DATA

Rest = Prior to the swim; IA = Immediately after the exercise session; 6-R = At the 6th minute of recovery.

(1) Glucose (mg/100 ml)

| Subject | Free Breathing | | | 7-Stroke Breathing | | |
|---|---|---|---|---|---|---|
| | Rest | IA | 6-R | Rest | IA | 6-R |
| J.S. | 79.46 | 91.66 | 87.79 | 83.00 | 96.72 | 82.73 |
| B.M. | 82.80 | 106.61 | 81.80 | 83.39 | 102.56 | 69.33 |
| E.J. | 78.94 | 81.16 | 80.01 | 89.40 | 104.39 | 92.29 |
| R.B. | 86.11 | 112.16 | 104.40 | 77.64 | 98.12 | 82.45 |
| R.H. | 86.46 | 100.71 | 85.25 | 86.71 | 97.40 | 90.75 |
| T.M. | 89.40 | 92.28 | 87.76 | 96.05 | 90.88 | 87.23 |
| $\bar{X} \pm S\bar{x}$ | 83.86 ±1.70 | 97.43 ±4.61 | 87.84 ±3.55 | 86.03 ±2.57 | 98.35 ±1.95 | 87.13 ±3.39 |

(2) Lactate (mg/100 ml)

| Subject | Free Breathing | | | 7-Stroke Breathing | | |
|---|---|---|---|---|---|---|
| | Rest | IA | 6-R | Rest | IA | 6-R |
| J.S. | 10.80 | 37.11 | 20.70 | 6.69 | 20.31 | 16.24 |
| B.M. | 6.16 | 57.11 | 35.00 | 5.53 | 47.13 | 25.30 |
| E.J. | 10.26 | 54.47 | 38.08 | 10.38 | 59.63 | 37.76 |
| R.B. | 9.78 | 49.04 | 31.64 | 6.90 | 46.14 | 32.86 |
| R.H. | 7.66 | 45.24 | 26.43 | 10.85 | 39.17 | 28.64 |
| T.M. | 8.53 | 17.14 | 11.20 | 10.61 | 19.20 | 10.55 |
| $\bar{X} \pm S\bar{x}$ | 8.87 ±0.72 | 43.35 ±5.99 | 27.18 ±4.07 | 8.99 ±0.91 | 38.60 ±6.54 | 25.22 ±4.18 |

(3) pH

| Subject | Free Breathing | | | 7-Stroke Breathing | | |
|---|---|---|---|---|---|---|
| | Rest | IA | 6-R | Rest | IA | 6-R |
| J.S. | 7.251 | 7.301 | 7.263 | 7.323 | 7.334 | 7.281 |
| B.M. | 7.318 | 7.310 | 7.284 | 7.329 | 7.153 | 7.283 |
| E.J. | 7.344 | 7.134 | 7.279 | 7.341 | 7.096 | 7.220 |
| R.B. | 7.342 | 7.313 | 7.326 | 7.368 | 7.330 | 7.317 |
| R.H. | 7.384 | 7.222 | 7.348 | 7.385 | 7.290 | 7.443 |
| T.M. | 7.366 | 7.353 | 7.349 | 7.377 | 7.323 | 7.313 |
| $\bar{X} \pm S\bar{x}$ | 7.334 ±0.019 | 7.272 ±0.033 | 7.308 ±0.015 | 7.354 ±0.011 | 7.254 ±0.042 | 7.310 ±0.030 |

(4) $P_vCO_2$ (mm Hg)

| Subject | Free Breathing | | | 7-Stroke Breathing | | |
|---|---|---|---|---|---|---|
| | Rest | IA | 6-R | Rest | IA | 6-R |
| J.S. | 66.7 | 48.8 | 61.5 | 53.9 | 48.5 | 60.9 |
| B.M. | 60.9 | 37.2 | 54.2 | 49.8 | 63.4 | 45.3 |
| E.J. | 52.9 | 73.6 | 50.1 | 51.1 | 79.4 | 59.7 |
| R.B. | 48.9 | 33.6 | 43.5 | 43.0 | 39.8 | 45.0 |
| R.H. | 44.0 | 57.7 | 43.9 | 45.8 | 46.5 | 31.9 |
| T.M. | 48.3 | 51.3 | 48.0 | 44.2 | 43.7 | 52.4 |
| $\bar{X} \pm S\bar{x}$ | 53.62 ±3.50 | 48.70 ±6.10 | 50.20 ±2.79 | 47.97 ±1.75 | 53.55 ±6.13 | 49.20 ±4.43 |

(5) $O_2$ Uptake (l/min)

| Subject | Free Breathing | | | 7-Stroke Breathing | | |
|---|---|---|---|---|---|---|
| | Rest | IA | NR* | Rest | IA | NR* |
| J.S. | 0.34 | 2.07 | 2.99 | 0.32 | 1.08 | 3.89 |
| B.M. | 0.39 | 3.00 | 6.27 | 0.41 | 4.04 | 6.60 |
| E.J. | 0.29 | 2.12 | 3.75 | 0.26 | 2.44 | 5.57 |
| R.B. | 0.31 | 2.86 | 4.97 | 0.28 | 2.26 | 4.79 |
| R.H. | 0.28 | 2.92 | 5.87 | 0.27 | 2.18 | 3.67 |
| T.M. | 0.40 | 3.36 | 4.43 | 0.43 | 4.10 | 4.50 |
| $\bar{X} \pm S\bar{x}$ | 0.33 ±0.02 | 2.72 ±0.21 | 4.71 ±0.51 | 0.33 ±0.03 | 2.68 ±0.48 | 4.84 ±0.45 |

*NR means net recovery $O_2$ uptake for 20 minutes.

# APPENDIX H (1)

GLUCOSE / CONTROLLED BREATHING 13:19 TUESDAY, SEPTEMBER 23, 1980 3

ANALYSIS OF VARIANCE PROCEDURE

DEPENDENT VARIABLE: Y

| SOURCE | DF | SUM OF SQUARES | MEAN SQUARE | F VALUE | PR > F | R-SQUARE | C.V. |
|---|---|---|---|---|---|---|---|
| MODEL | 35 | 3057.58846889 | 87.35967111 | 99999.99 | 0.0000 | 1.000000 | 0.0000 |
| ERROR | 0 | 0.00000000 | 0.00000000 | | STD DEV | | Y MEAN |
| CORRECTED TOTAL | 35 | 3057.58846889 | | | 0.00000000 | | 89.60565556 |

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| SUBJ | 5 | 198.16865556 | . | . |
| BREATH | 1 | 0.38440000 | . | . |
| SUBJ*BREATH | 5 | 730.35910000 | . | . |
| REST | 2 | 1241.06857222 | . | . |
| SUBJ*REST | 10 | 681.23616111 | . | . |
| BREATH*REST | 2 | 57.43505000 | . | . |
| SUBJ*BREATH*REST | 10 | 148.93655000 | . | . |

TESTS OF HYPOTHESES USING THE ANOVA MS FOR SUBJ*BREATH AS AN ERROR TERM

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| BREATH | 1 | 0.38440000 | 0.00 | 0.9611 |

TESTS OF HYPOTHESES USING THE ANOVA MS FOR SUBJ*REST AS AN ERROR TERM

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| REST | 2 | 1241.06857222 | 9.11 | 0.0056 |

TESTS OF HYPOTHESES USING THE ANOVA MS FOR SUBJ*BREATH*REST AS AN ERROR TERM

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| BREATH*REST | 2 | 57.43505000 | 1.93 | 0.1958 |

Continued ...

# APPENDIX H (1)

GLUCOSE / CONTROLLED BREATHING 13:10 TUESDAY, SEPTEMBER 23, 1980 4

ANALYSIS OF VARIANCE PROCEDURE

DUNCAN'S MULTIPLE RANGE TEST FOR VARIABLE Y

MEANS WITH THE SAME LETTER ARE NOT SIGNIFICANTLY DIFFERENT.

ALPHA LEVEL=.05 DF=10 MS=68.1236

| GROUPING | MEAN | N | REST |
|---|---|---|---|
| A | 97.887500 | 12 | 2 |
| B | 85.962500 | 12 | 3 |
| B | | | |
| B | 84.946667 | 12 | 1 |

# APPENDIX H (2)



ANALYSIS OF VARIANCE PROCEDURE

DEPENDENT VARIABLE: Y

| SOURCE | DF | SUM OF SQUARES | MEAN SQUARE | F VALUE | PR > F | R-SQUARE | C.V. |
|---|---|---|---|---|---|---|---|
| MODEL | 35 | 9672.16482222 | 276.34756635 | 99999.99 | 0.0000 | 1.000000 | 0.0000 |
| ERROR | 0 | 0.00000000 | 0.00000000 | | STD DEV | | Y MEAN |
| CORRECTED TOTAL | 35 | 9672.16482222 | | | 0.00000000 | | 25.36777778 |

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| SUBJ | 5 | 1936.40302222 | . | . |
| BREATH | 1 | 43.25254444 | . | . |
| SUBJ*BREATH | 5 | 116.67032222 | . | . |
| REST | 2 | 6173.75883889 | . | . |
| SUBJ*REST | 10 | 1260.36106111 | . | . |
| BREATH*REST | 2 | 36.03443889 | . | . |
| SUBJ*BREATH*REST | 10 | 103.78455444 | . | . |

TESTS OF HYPOTHESES USING THE ANOVA MS FOR SUBJ*BREATH AS AN ERROR TERM

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| BREATH | 1 | 43.25254444 | 1.82 | 0.2348 |

TESTS OF HYPOTHESES USING THE ANOVA MS FOR SUBJ*REST AS AN ERROR TERM

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| REST | 2 | 6173.75883889 | 24.49 | 0.0001 |

TESTS OF HYPOTHESES USING THE ANOVA MS FOR SUBJ*BREATH*REST AS AN ERROR TERM

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| BREATH*REST | 2 | 36.03443889 | 1.74 | 0.2253 |

Continued ...

# APPENDIX H (2)

LACTATE / CONTROLLED BREATHING 13:15 TUESDAY, SEPTEMBER 23, 1980 4

ANALYSIS OF VARIANCE PROCEDURE

DUNCAN'S MULTIPLE RANGE TEST FOR VARIABLE Y

MEANS WITH THE SAME LETTER ARE NOT SIGNIFICANTLY DIFFERENT.

ALPHA LEVEL=.05 DF=10 MS=126.036

| GROUPING | MEAN | N | REST |
|---|---|---|---|
| A | 40.974167 | 12 | 2 |
| B | 26.200000 | 12 | 3 |
| C | 8.929167 | 12 | 1 |

# APPENDIX H (3)



ANALYSIS OF VARIANCE PROCEDURE

DEPENDENT VARIABLE: Y

| SOURCE | DF | SUM OF SQUARES | MEAN SQUARE | F VALUE | PR > F | R-SQUARE | C.V. |
|---|---|---|---|---|---|---|---|
| MODEL | 36 | 0.17566431 | 0.00501898 | 99999.99 | 0.0000 | 1.000000 | 0.0000 |
| ERROR | 0 | 0.00000000 | 0.00000000 | | STD DEV | | Y MEAN |
| CORRECTED TOTAL | 35 | 0.17566431 | | | 0.00000000 | | 7.30536111 |

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| SUBJ | 5 | 0.05888114 | . | . |
| BREATH | 1 | 0.00001003 | . | . |
| SUBJ*BREATH | 5 | 0.01266914 | . | . |
| REST | 2 | 0.03934039 | . | . |
| SUBJ*REST | 10 | 0.05106528 | . | . |
| BREATH*REST | 2 | 0.00210972 | . | . |
| SUBJ*BREATH*REST | 10 | 0.01157861 | . | . |

TESTS OF HYPOTHESES USING THE ANOVA MS FOR SUBJ*BREATH AS AN ERROR TERM

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| BREATH | 1 | 0.00001003 | 0.00 | 0.9528 |

TESTS OF HYPOTHESES USING THE ANOVA MS FOR SUBJ*REST AS AN ERROR TERM

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| REST | 2 | 0.03934039 | 3.85 | 0.0575 |

TESTS OF HYPOTHESES USING THE ANOVA MS FOR SUBJ*BREATH*REST AS AN ERROR TERM

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| BREATH*REST | 2 | 0.00210972 | 0.91 | 0.4330 |



University of Windsor

# APPENDIX H (4)



ANALYSIS OF VARIANCE PROCEDURE

DEPENDENT VARIABLE: Y

| SOURCE | DF | SUM OF SQUARES | MEAN SQUARE | F VALUE | PR > F | R-SQUARE | C.V. |
|---|---|---|---|---|---|---|---|
| MODEL | 35 | 3707.54555556 | 105.92987302 | 99999.99 | 0.0000 | 1.000000 | 0.0000 |
| ERROR | 0 | 0.00000000 | 0.00000000 | | STD DEV | | Y MEAN |
| CORRECTED TOTAL | 35 | 3707.54555556 | | | 0.00000000 | | 50.53888889 |

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| SUBJ | 5 | 1612.62222222 | . | . |
| BREATH | 1 | 3.24000000 | . | . |
| SUBJ*BREATH | 5 | 143.35666667 | . | . |
| REST | 2 | 13.33388889 | . | . |
| SUBJ*REST | 10 | 1295.53444444 | . | . |
| BREATH*REST | 2 | 166.09500000 | . | . |
| SUBJ*BREATH*REST | 10 | 473.45833333 | . | . |

TESTS OF HYPOTHESES USING THE ANOVA MS FOR SUBJ*BREATH AS AN ERROR TERM

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| BREATH | 1 | 3.24000000 | 0.11 | 0.7504 |

TESTS OF HYPOTHESES USING THE ANOVA MS FOR SUBJ*REST AS AN ERROR TERM

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| REST | 2 | 13.33388889 | 0.05 | 0.9501 |

TESTS OF HYPOTHESES USING THE ANOVA MS FOR SUBJ*BREATH*REST AS AN ERROR TERM

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| BREATH*REST | 2 | 166.09500000 | 1.75 | 0.2223 |

# APPENDIX H (5)



ANALYSIS OF VARIANCE PROCEDURE

DEPENDENT VARIABLE: Y

| SOURCE | DF | SUM OF SQUARES | MEAN SQUARE | F VALUE | PR > F | R-SQUARE | C.V. |
|---|---|---|---|---|---|---|---|
| MODEL | 35 | 140.81132389 | 4.02318068 | 99999.99 | 0.0000 | 1.000000 | 0.0000 |
| ERROR | 0 | 0.00000000 | 0.00000000 | | STD DEV | | Y MEAN |
| CORRECTED TOTAL | 35 | 140.81132389 | | | 0.00000000 | | 2.60294444 |

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| SUBJ | 5 | 9.06628056 | . | . |
| BREATH | 1 | 0.00624100 | . | . |
| SUBJ*BREATH | 5 | 2.73609833 | . | . |
| REST | 2 | 118.65844422 | . | . |
| SUBJ*REST | 10 | 6.72396111 | . | . |
| BREATH*REST | 2 | 0.04390867 | . | . |
| SUBJ*BREATH*REST | 10 | 3.57939000 | . | . |

TESTS OF HYPOTHESES USING THE ANOVA MS FOR SUBJ*BREATH AS AN ERROR TERM

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| BREATH | 1 | 0.00624100 | 0.01 | 0.9191 |

TESTS OF HYPOTHESES USING THE ANOVA MS FOR SUBJ*REST AS AN ERROR TERM

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| REST | 2 | 118.65844422 | 88.23 | 0.0001 |

TESTS OF HYPOTHESES USING THE ANOVA MS FOR SUBJ*BREATH*REST AS AN ERROR TERM

| SOURCE | DF | ANOVA SS | F VALUE | PR > F |
|---|---|---|---|---|
| BREATH*REST | 2 | 0.04390867 | 0.06 | 0.9409 |

Continued ...

# APPENIDX H (5)

VOz / CONTROLLED BREATHING 13:15 TUESDAY, SEPTEMBER 23, 1980

ANALYSIS OF VARIANCE PROCEDURE

DUNCAN'S MULTIPLE RANGE TEST FOR VARIABLE Y

MEANS WITH THE SAME LETTER ARE NOT SIGNIFICANTLY DIFFERENT.

ALPHA LEVEL=.05 DF=10 MS=0.672396

| GROUPING | MEAN | N | REST |
|---|---|---|---|
| A | 4.775000 | 12 | 3 |
| B | 2.702500 | 12 | 2 |
| C | 0.331333 | 12 | 1 |

# VITA AUCTORIS



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| | | |
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