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THE UNIVERSITY OF ALBERTA

CARDIOVASCULAR AND PERCEIVED EXERTION RESPONSES TO CYCLING VELOCITIES

BY

ALLEN VICTOR ETTINGER

A THESIS
SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND
RESEARCH IN PARTIAL FULFILMENT OF THE REQUIREMENTS
FOR THE DEGREE OF
MASTER OF SCIENCE

DEPARTMENT OF PHYSICAL EDUCATION AND SPORT STUDIES

EDMONTON ALBERTA

SPRING 1989

ISBN 0-315-52822-2

THE UNIVERSITY OF ALBERTA

RELEASE FORM

NAME OF AUTHOR: ALLEN VICTOR ETTINGER

TITLE OF THESIS: CARDIOVASCULAR AND PERCEIVED EXERTION
RESPONSES TO CYCLING VELOCITIES.

DEGREE FOR WHICH THESIS WAS PRESENTED: Master of Science

YEAR THIS DEGREE GRANTED: 1989

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled CARDIOVASCULAR AND PERCEIVED EXERTION RESPONSES TO CYCLING VELOCITIES submitted by Allen Victor Ettinger in partial fulfilment of the requirements for the degree of Master of Science.

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Supervisor

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DATE: June 20/78

DEDICATION

This thesis is dedicated to:

Geordie & Bill Knull, my second parents, who provided love, understanding and direction at a time it was needed and have continued to do so in my adulthood;

Mrs. Daisy Teeter, my grandmother, who demonstrated by personal example love, a superior work ethic and personal sacrifice for the pleasure of others;

Mr. Charles Edgar Ettinger, my father, who demonstrated dedication to task, personal management and powerful interpersonal relationships; and

David, Denis and Robert, my three sons, who provided support, understanding, assistance and self-denial to make this experience possible.

ABSTRACT

The purpose of this study was to investigate cardiovascular and perceived exertion (RPE) responses to cycling velocities (CR) of 40, 60, 80 and 100 rpm at low, medium, and high power outputs (40, 55 and 70% of VO_2max). Nineteen young healthy physically active males participated in the study consisting of a VO_2max test and four cycling velocity treatment tests on a constant load cycle ergometer.

The cycling velocity tests, conducted 24 to 48 hours apart, required the subject to cycle at one velocity for 4 minutes at low power output (PO), 5 minutes at medium PO and 5 minutes at high PO. Oxygen uptake (VO_2), heart rate (HR), RPE, blood pressure, stroke volume (SV) (determined by impedance cardiography), and cardiac output (CO) were monitored. Blood samples for venous lactate concentrations (BdLa) were drawn immediately pre-exercise and 5 minutes post-exercise.

Data were analyzed using a two-way analysis of variance (ANOVA) with repeated measures. A Newman-Kuels' post hoc test was conducted to determine individual mean differences.

At low and medium PO, the cycling velocities that produced the lowest demands on the cardiovascular system (optimal cycling velocities) were 40 to 80 rpm. No difference existed (p>.05) in VO_2, HR, SV CO, MAP AND RPE. Significantly higher cardiovascular responses (p<.05) occurred at 100 rpm (VO_2, HR, CO, MAP). At high PO 60 and 80 rpm placed the lowest demands on the cardiovascular system and no significant difference existed (p>.05) in VO_2, HR, SV, CO, MAP, SVR and RPE. Both [illegible] and 100 rpm caused higher demands on the cardiovascular system. [illegible] BdLa and RPE were higher (p<.05) at 40 rpm than those at 60 and 80 rpm. At 100 rpm VO_2, HR, CO, Bdla and RPE were significantly higher (p<.05) than results at 60 and 80 rpm and all but RPE results were higher than those elicited at 40 rpm (p<.05).

The increased VO_2, CO, and Bdla at high cycling velocity (100 rpm) seemed to be a result increased static contraction of trunk and upper body muscles needed to provide stability. The possible cause of elevated VO_2 and BdLa for slow cycling velocity (40 rpm) at high PO was increased upper body and trunk muscle involvement and higher portions of anaerobic metabolism as a result of the larger force required for each pedal stroke.

The study demonstrated that for young healthy physically active males 60 and 80 rpm were the optimal

cycling velocities over a wide range of power outputs in terms of cardiovascular and metabolic responses. For low and medium PO, 40 to 80 rpm were found optimal.

The data suggest that 60 to 80 rpm can be used interchangably in submaximal testing over a wide range of power outputs in healthy active young males.

ACKNOWLEDGEMENTS

I would like to express my appreciation to a number of friends who have contributed to my graduate experience and in particular to the completion of this thesis:

The physical education and recreation graduate students who provided commradeship, support and an endless source of knowledge and assistance.

Marg Rees for her assistance and exceptional work ethics in data collection.

Chris Cummins for her tolerance, patients, friendship and assistance throughout the program.

Dr. Robert Haennel for his guidance, assistance and fruitful discussions.

Dr. Stu Petersen for his encouragement, invaluable advise, friendship and endless corrections.

Dr. Tissa Kappagoda for his encouragement, challenges, guidance, invaluable advise, direction and logistical support.

Dr. Art Quinney for ensuring an enriched graduate experience, direction, encouragement, support, challenge, invaluable advise and friendship.

The Department of National Defence who made this experience financially feasible.

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CHAPTER I

INTRODUCTION

Maximum oxygen uptake (VO_2max) has long been used by exercise physiologists as a measure of the ability to perform continuous physical work (Astrand and Rodahl, 1986; Glassford et al., 1965). Although some authors utilize the terms maximum aerobic power and aerobic capacity synonymously (Brooks and Fahey, 1984), others (Astrand and Rodahl, 1986; MacDougall et al., 1983) define 'aerobic' capacity as the total amount of adenosine triphosphate available for oxidation through aerobic processes while 'aerobic' capacity is defined as the maximum amount of oxygen that can be taken up per unit time, in spite of further increases in exercise intensity. The latter value is termed maximum oxygen uptake (VO_2max) and is measured in absolute terms in liters per minute or relative terms in milliliters per kilogram of body weight per minute.

Numerous methods of assessing maximal oxygen uptake have been developed (Astrand et al., 1959; Balke and Ware, 1956; Mitchell et al., 1958; Taylor, 1941). Maximum oxygen uptake is still considered one of the most objective measure in determining cardiovascular fitness (Shepherd and Shepherd, 1987).

If a precise measure of oxygen uptake is required, then direct assessment is necessary. If however, exact measure is not required or possible, then predictive VO_2max test may be used. Most predictive tests are based on the relationship that exists between oxygen uptake and one or more variables. The variable is monitored at submaximal workloads and then VO_2max is predicted from submaximal values (heart rate is frequently used). Submaximal assessment is appropriate if: precision is not essential; testing time is short; and/or subjects safety is a concern Direct determination of maximum oxygen uptake is expensive, time consuming and presents a potential risk to subjects. It requires expensive equipment, highly trained evaluators, a laboratory setting, and often restrictive selection of subjects.

As a result of these limitations, many investigators have attempted to develop submaximal tests which are safe, valid, reliable and less complex (Astrand, 1952; Astrand and Ryhming, 1954; Bruce et al., 1973; Margaria et al., 1954; Sjostrand, 1947).

Berggren and Christensen (1950) were amoung the first to find a linear relationship between heart rate (HR) and oxygen uptake (VO_2). They outlined the first conditions under which oxygen uptake could be predicted from heart rate emphasizing inter-individual differences due to age, sex, task and temperature. Although other physiological parameters have been investigated as indirect determinants of oxygen uptake (Astrand and Rodahl, 1986) they have been found to produce a larger margin of error than the use of heart rate. Consequently, heart rate has predominated as the best indirect predictor of oxygen uptake.

Numerous cycle ergometer tests have been developed to predict oxygen uptake (Andersen and Hermansen, 1965; Astrand, 1965; Sjostrand, 1947) from the measurement of heart rate and power output (PO). Formulae, nomograms, and graphing techniques which rely on the linear relationship between heart rate and power output (Astrand and Ryhming, 1954; Bruce et al., 1973) have been used.

Numerous investigators (Andersen and Hermansen, 1965; Astrand, 1952; Sjostrand, 1978) have compared submaximal estimation of maximal oxygen uptake with actual values and found the methods valid and reliable. Coefficients of variance of 5% to 15% for validity measures and 3% to 7% for reliability measures have been reported by these researchers.

However, most significant were the findings of Glassford et al. (1965) in the comparison of two maximal treadmill tests (Mitchell et al., 1958;

Taylor, 1941), one maximal cycle ergometer test (Astrand, 1952) and a submaximal cycle ergometer test (Astrand and Ryhming, 1954). They found the correlation of maximal oxygen uptake between values predicted by the submaximal test and any direct method was equivalent to that between any two of the direct methods. This and similar findings (Andersen and Hermansen, 1965; Astrand, 1965) resulted in acceptance of standards for cycle ergometry testing at the 16th World Congress on Sports Medicine in Hanover, Germany (June 1966) by the International Council of Sports Medicine and Physical Education (ICSPE) Research Committee for International Standardization in Ergometry (from Mellerowicz and Smodlaka, 1981). The primary recommendation was that cycle ergometer tests should be conducted at cycling velocities of 50 to 60 revolutions per minute (rpm) for International Standardization.

Since 1966 studies have been conducted by many researchers investigating the relationship between cycling frequency and oxygen uptake (Bannister and Jackson, 1967; McKay and Bannister, 1976), respiratory gases (Gueli and Shephard, 1976); heart rate (Eckermann and Millahn, 1967; Michielli and Stricevic, 1977); fiber recruitment patterns (Moffatt and Stamford, 1978; Seabury et al., 1977); cycling efficiency (Coast et al., 1986; Sjostrand, 1978; Takano, 1987); blood lactate (Burke et al., 1981; Buchanan and Weltman, 1985); efficiency (Gaesser and Brooks, 1975); perceived exertion (Pandolf and Noble, 1973; Cafarelli, 1978; Borg et al., 1985; Edwards et al., 1971; Ekblom and Goldbarg, 1971); or a combination of these variables (Lollgen et al., 1980; Gamberale, 1972; Hagberg et al., 1981; Boning et al., 1984).

These studies have included: investigation of few cardiovascular variables, small power output ranges, too few cycling velocities (CR), small samples and/or heterogenous samples. These authors have attempted to explain the differences in physiological responses to cycling velocities without investigating cardiac output (CO).

Since the primary cardiovascular adjustment results from the fact that oxygen uptake is the product of cardiac output and arteriovenous difference ($a-vO_2$ diff; the difference in oxygen content of the blood entering and leaving the pulmonary capillaries), it is imperative to evaluate cardiac output. The investigation of cardiac output should provide an in depth understanding of the cardiovascular response to cycling velocities.

A. PURPOSE

The purpose of this study was to investigate cardiovascular and perceived exertion responses to cycling velocities of 40, 60, 80 and 100 revolutions per minute (rpm) at power output equated to 40%, 55% and 70% of maximum oxygen uptake (VO_2max) in young healthy active males. Nineteen males participated in the study which consisted of a VO_2max test and four treatment tests (one for each cycling velocity) on an electric constant load cycle ergometer.

B. HYPOTHESIS

The research hypothesis for this study was that no difference existed in any of the physiological or perceived exertion responses between the 4 cycling velocities at equivalent power outputs.

Statistical Hypothesis: Ho: X-X = 0

Hi: X-X = 0

CHAPTER II

METHODS AND PROCEDURES

A. SUBJECTS

Twenty-four healthy, physically active, non-smoking, male volunteers were subjects in this study; however, only 19 completed all phases (4 were injured precluding further participation and one moved away from the city). All subjects were physically active (training approximately three hours aerobically each week), were familiar with cycling and maximum oxygen uptake tests on the cycle ergometer. Subjects were fully informed of the purpose of the experiment and written consent was obtained prior to commencement of the study (Appendix A). The study protocol was approved by the Institutional Ethics Review Committee.

B. ENVIRONMENTAL CONDITIONS

Temperature has been demonstrated to affect heart rate, oxygen uptake and other physiological parameters (Rowell, Taylor and Wang, 1964; Toner et al., 1982). In this study relative humidity was not controlled, and temperature was monitored and found to be consistent (22 to 24 degrees celcius) during all testing. In the laboratory, relative humidity varied only slightly. Laboratory traffic and other distractions were minimized during all data collection to reduce possible effects on physiological parameters at low power outputs (Astrand, 1950; Taylor et al., 1955).

C. TESTING STATE STANDARDIZATION

All subjects were post absorptive for a mimimum of three hours and did not take part in any strenuous exercise or physical activity for twelve hours prior to testing. These factors have been shown to affect heart rate and cardiac output (Astrand and Rodahl, 1977; Taylor et al., 1963). Subjects were scheduled at the same time of day for every treatment test to reduce biological variability (Brooks and Fahey, 1984).

D. STUDY OUTLINE

The experiment consisted of 6 separate sessions in the laboratory (lab): a familiarization session; a maximum oxygen uptake test, and four treatment tests.

The familiarization session was a one hour session to brief and allow the subjects to become familiar with the lab and testing conditions. During this session subjects were exposed to mini-trials which allowed familiarization with the [illegible]tus and experimental conditions (cycling frequency, impedance cardiography, respiratory gas analysis, and blood pressure [BP] determination).

The maximum oxygen uptake test (VO_2max) was conducted 24 hours after the familiarization sessions on the electrically braked constant load cycle ergometer.

The four treatment tests were conducted on 4 separate days, each at least 48 hours apart. The first treatment test for each subject was scheduled a minimum of 48 hours after the VO_2max test. In order to reduce intra-subject biological variability each subject was scheduled at the same time of day (Davies and Sargeant, 1975). During the treatment test, the subject cycled at one cycling rate (40, 60, 80 or 100 rpm). Subjects were randomly assigned to a specific treatment test order. With 24 possible permutations of the four cycling rates, each subject had a different exposure order to the treatment tests (see Appendix B). During the treatment test the subject cycled at the predetermined rate for 3 workloads; the first workload was cycling at a power output equated to 40% of VO_2max for 4 minutes; the second workload was cycling at [illegible]5% of VO_2max for 5 minutes, and the final workload was 5 minutes cycling at 70% of VO_2max.

E. TESTING APPARATUS

The equipment used in this study included : a constant load electric cycle ergometer (Model 740, Siemens-Elema, Siemens Electric Ltd., Mississuaga, Ontario); a magnetic cycling revolution counter (Cateye Micro Cyclocompter Model CC-6000, Tsuyama Co Ltd., Japan); Beckman Metabolic Measurement Cart (Model MMC-Cat554277, Advanced Technology Operations, Fullerton, CA); a Minnesota Impedance Cardiograph (Model 304B Surcom Inc., Minneapolis); 4 channel Gould ink recorder and amplifier (Model 8188-402, Ballainvillers, France), Gould Isolation Transformer (Model 882895-3, Gould inc., Cleveland, Ohio); Baumanometer Mercury Sphygmomanometer (Model Standby, WA Baum Co. Inc., Copiagne, New York) and stethescope ; cardiotachometer (Sport tester, Model 3000, Polar Electro, Kempele, Finland); a tape measure (mm), blood sampling equipment; Sybron Thermolyne Mixer (Model M-16715); Sovall General Lab Centrifuge (Model GLC-4, Dupont); and a UV/VIS Phillips Spectrophotometer (Model PU8800, Pye Unicam Ltd., Cambridge, England).

the same equipment was used for every subject during each test.

F. GENERAL METHODS

i) Stroke Volume and Cardiac Output Measurement

In this study stroke volume and cardiac output were measured using the Minnesota Impedance Cardiography Model 304B (Surcom Inc., Minneapolis). Impedance Cardiography is a non-invasive, atraumatic method of measuring heart rate and stroke volume from which cardiac output may be calculated. The technique involves the placement of self-adhesive disposible mylar-backed aluminum electrode bands around the neck and thorax. A weak frequency alternating current is passed through the outer two electrodes. The constant alternating current is undetectable by the subject and the frequency is so high [illegible] is incapable of stimulating the heart (Kubicek et al., 1966).

On each visit to the laboratory for testing, the subject reported at least three hours post-absorptive. The four bands of self-adhesive disposable mylar-backed aluminum electrode tape were placed around the neck and chest 30 minutes prior to any measurement. Two bands were placed around the neck, 3 to 5 cm apart the third around the trunk at the level of the xiphisternum and the fourth at a level just above the umbilicus When connected the two outer electrodes transmitted a constant, sinusoidal, alternating current (4 ma RMS and 100 KHz) through the thorax and the changes in transthoracic electrical impedance were detected by the inner two electrodes (Figure 2.1) Mean total transthoracic impedance between the inner electrodes (Zo) was computed and displayed by the Impedance Cardiograph Simultaneous recordings of the rate of change of impedance through each phase of the cardiac cycle (dZ/dt), were made through the electrocardiograph and phonocardiograph on a 4 channel ink recorder at a paper speed of 50 mm/s (Model 8188-402, Gould Inc., France). The heart rate, dZ/dt min, and left ventricular ejection time, were obtained from the recordings as shown in Figure 2.2. Calculation of stroke volume was made using the following equation:

$$\text{Stroke Volume} \quad \frac{P \times L^2 \times dZ/dt_{min} \times T}{Z_o^2}$$

where P = electrical resistivity of blood at body temperature ($P = 53.2e^{0.022H}$). H = hematocrit (%), L = average distance (cm) between the inner pair of electrodes measured at the anterior and posterior midline, Z_o = mean transthoracic impedance (ohms)

FIGURE 2.1

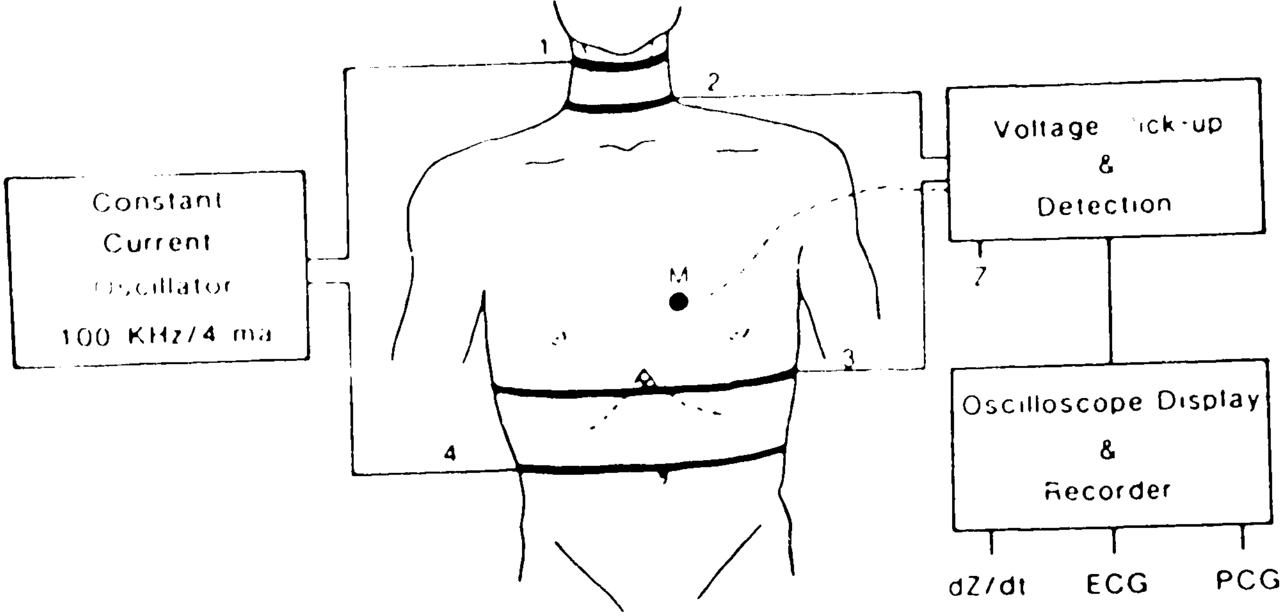
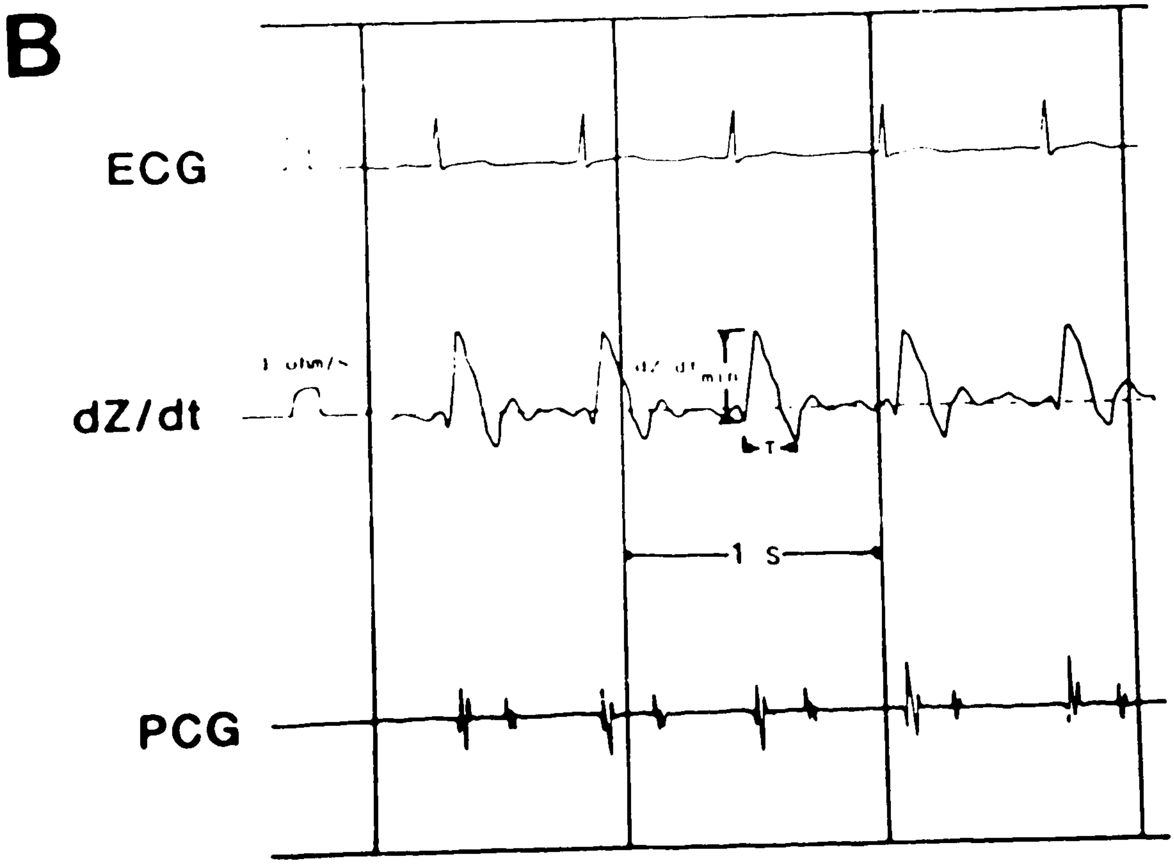


FIGURE 2.2



between the inner two electrodes, dZ/dt_{min} - minimum value for the rate of change of impedance (ohms) occuring during the cardiac cycle. T - left ventricular ejection time (s).

Recordings were made at rest and during exercise. It was found that movement caused by respiration and exercise introduced artifacts into the recordings. These artifacts were avoided by r[illegible]ring the subjects to stop all movement, remain [illegible]ess and hold their breath at normal end-expira[illegible] for approximately 5 seconds, while 5 to 10 cardiac cycles were recorded (Hetherington et al., 1985). Five cardiac cycles were used for the calculation of stroke volume. The hematocrit was measured from blood samples drawn from an antecubital vein immediately prior to exercise, and at 5 minutes post-exercise. The hematocrit value was interpolated between pre-test to post-test blood samples to correspond to the impedance cardiography recording.

ii) Measurement of Maximal Oxygen Uptake

Maximal aerobic power was determined by measuring oxygen uptake during a modified incremental exercise test (Thoden et al., 1983) on the cycle ergometer. The subject cycled at 60 rpm. The initial power output was 100 watts (W) with 50 watt increments added every two minutes.

The physiological measures obtained every 30 seconds were ventilation volume (Ve), volume of carbon dioxide produced (VCO_2), volume of oxygen consumed (VO_2), respiratory exchange ratio (R) and heart rate (HR). The peak oxygen uptake value obtained during the exercise test was recorded as VO_2max. Expired gases were collected continuously and analyzed every 30 seconds the Beckman Metabolic Measurement Cart. Standard gases of known concentration were used to calibrate the gas analyzers before and after each test. Heart rate was recorded every 30 seconds using the cardiotachometer and a simultaneous ECG strip was run on the impedance recorder (in the last fifteen seconds of each workload). Blood pressure was measured using a mercury sphygmomanometer at rest and during the last 30 seconds of each workload throughout the incremental test. Impedance cardiography recordings were taken at end of each workload using the Minnesota Impedance Cardiograph.

The exercise test continued until at least one of thefollowing end point criteria for VO_2max had been achieved: oxygen uptake increased less than 50 ml with increased workload; a heart rate of 90% of age predicted maximal heart rate was attained; or, the subject was unable to continue. Appendix C provides a detailed description of the maximum oxygen uptake test protocol.

iii) Workload Determination

In order to determine the power output necessary to equate to 40, 55 and 70% of VO_2max, the results of the VO_2max test were utilized. Oxygen uptake (l/min) was plotted against power output (watts) at each workload for every subject and the percentage of maximal oxygen uptake was determined (that is, the oxygen uptake equal to 40, 55 and 70% of VO_2max). The power outputs were interpolated from the graph at the required oxygen uptake for each subject. These were the power outputs, equated to 40, 55 and 70% of VO_2max, used for every treatment test.

iv) Treatment Protocol

Each subject attended four treatment tests, scheduled a minimum of 48 hours apart. Identical protocols were utilized for each treatment. The only difference between the treatments was the rate at which the subject cycled (40, 60, 80 or 100 rpm). The detailed treatment test protocol is contained in Appendix D.

In brief, the protocol consisted of the following: anthropometric measurement (height and weight); cardiac impedance measures; a 30 minute rest period, (seated); affixed the subject with headgear and mouthpiece for respiratory gas collection; collection of respiratory gas analysis for a 2 minutes rest period; resting physiological measures taken (heart rate, stroke volume, cardiac output and blood pressure); a blood sample drawn from an antecubital vein; an incremental cycle ergometer test; 1 and 3 minute seated recovery, physiological measures taken; and a 5 minute, post-exercise, blood sample drawn from the antecubital vein.

During the tests, subjects breathed through a low resistance respiratory valve and expired gases were collected and analyzed by a pre-calibrated Beckman Metabolic Measurement Cart (this occurred from 2 minutes pre-exercise to 3 minutes post-exercise). Oxygen uptake and heart rate were recorded every 30 seconds. The heart rate was recorded

every 30 seconds using a cardiotachometer simultaneous measurements of heart rate were recorded during rest, in the last 15 seconds of each workload, and after 1 and 3 minutes of recovery using the cardiotachometer and impedance cardiograph. Blood pressure was measured during rest, the last minute of each workload, and at 30 seconds and 2:30 minutes of recovery using a mercury sphygmomanometer. During the final minute of each workload, a rating of perceived exertion (RPE) was obtained using the Borg Scale (Borg, 1970). Blood samples were drawn after 32 minutes rest (immediately prior to exercise) and 5 minutes after cessation of the final workload.

v) Plasma Lactate Concentration

Whole venous blood was drawn from an antecubital vein after 32 minutes rest and at 5 minutes post-exercise (recovery). In each case 0.5 ml of blood was immediately pipetted into a cold vacutainer containing 2.0 ml of chilled perchloric acid. The perchloric and whole blood was mixed with a Sybron Thermolyne Mixer (Model M-16715) and placed on ice for 5 minutes to ensure protein precipitation was complete. The vacutainers were then centrifuged using a Sovall General Lab Centrifuge (Model GLC-4, Dupont) for 15 minutes. The de-proteinized supernatant (clear liquid) was pipetted into a pre-chilled sterile vacutainer which was

stoppered and frozen, at approximately -73 degrees

Celcius, until analysis was performed.

The assay used was based on the principle that lactate dehydrogenase (LDH) catalyzes the reverse reaction between pyruvic acid and nicotinamide adenine dinucleotide reduced (NADH) to produce lactic acid and nicotinamide adenine dinucleotide (NAD). In order to force the reaction to completion, formed pyruvate must be trapped with hydrazine. The increased absorbance due to the formation of NADH provides the measure of the original lactate concentration. The analysis was performed spectrophotometrically at 340 nm according to the Sigma Technical Bulletin No 825-UV (1981). All chemical reagents were obtained from the Sigma Chemical Company.

All the samples were analyzed in triplicate and the mean absorbance reading was used for calculation of lactate concentrations. One third of the lactates were analyzed per reagent mixture. A standard curve was constructed for each reagent mixture based on six samples of known concentration. Additionally, 12 samples were chosen at random and assayed with each normal assay sample. In total 36 samples were analyzed twice with different reagent mixtures (Appendix H) to ensure that the results were consistent across assay runs, reagent mixture and standard curves (r = 0.94, standard error of 5%).

G. STATISTICAL ANALYSIS

Statistical analysis of the effect of cycling velocities and relative power output was determined by a two-way analysis of variance (ANOVA) with repeated measures (Winer, 1971). The relative power output factor consisted of 3 levels: power output equated to 40, 55 and 70% of VO_2max. The factor of cycling velocities was repeated on four measures: 40, 60, 80 and 100 rpm. If a significant F value was attained for a variable at a power output and cycling velocity indicating a significant interaction between the two factors, a Newman-Keuls' multiple means comparison procedure was performed to assess the significance of specific difference between the mean values for each cycling velocity at each PO (Ferguson, 1981; Winer, 1971). Cycling velocity and blood lactate concentrations were analyzed using a one-way analysis of variance with repeated levels and a post hoc Newman-Keuls' procedure because only one level of the power output factor was evaluated [illegible] 5 minutes post-exercise). Linear regressions f[illegible] output on oxygen uptake were performed as rep[illegible] re to include all three power output leve[illegible] are represented as mean plus or minus t[illegible] error of the mean (mean ± SEM). Significance in this thesis is associated with a probability of less than 0.05 ($p<.05$) unless otherwise indicated.

CHAPTER III

RESULTS

A. SUBJECTS

Nineteen male subjects participated in the study. All subjects were physically active males (mean age of 27 ±1 years), ranging in age from 17 to 35. They all were participating in aerobic fitness activities of not less than 3 hours per week.

Subject characteristics are presented in Table 3.1 (detailed subject characteristic profiles are contained in Appendix E).

Table 3.1 - Subject Characteristics
(Mean ± SEM, Range)

CHARACTERISTICS	MEAN	SEM	RANGE
AGE (years)	27	±1	17 - 35
HEIGHT(cm)	180.0	±2.1	165.0 - 196.0
WEIGHT (kg)	76.0	±2.6	54.5 - 96.5
VO_2max (l/min)	3.93	±0.11	3.00 - 4.68
VO_2max (ml/kg/min)	52.1	±1.2	39.7 - 60.7

B. ANALYSIS OF RESULTS

The power outputs required to elicit 40, 55 and 70% of VO_2max for each subject were calculated. The mean power outputs (±SEM) were 111 (±3) watts at 40% of VO_2max, 167 (±5) watts at 55% of VO_2max and 223 (±7) watts at 70% of VO_2max. These power outputs (40, 55 and 70% VO_2max) are referred to as low, medium and high power outputs, respectively.

The variables investigated in this study are oxygen uptake, heart rate, stroke volume, cardiac output, mean arterial pressure, systemic vascular resistance, and rate of perceived exertion. Oxygen uptake, heart rate, stroke volume, systolic and diastolic blood pressures were measured. Cardiac output, mean arterial pressure and systemic vascular resistance were calculated. Cardiac output was calculated as the product of heart rate and stroke volume. Mean arterial pressure was calculated by adding diastolic blood pressure to one third of the difference between systolic and diastolic blood pressures. Systemic vascular resistance was calculated by dividing mean arterial pressure by cardiac output. Individual subject raw data are contained in Appendix F. The means (±SEM) are summarized in Tables 3.2, 3.3 and 3.4 for low, medium and high power outputs, respectively

In order to test for the significant difference between means for any variable and the four cycling velocities, a repeated measures analysis of variance (ANOVA) was used. For variables observed over 3 power outputs, a two-way ANOVA with repeated measures was conducted. For variables only observed at one level a one-way ANOVA with repeated measures was employed. A Student Newman Keuls' post hoc test was conducted on all significant interactions for any variable at a power output.

TABLE 3.2 - Physiological and RPE Responses at Power Output Equated to 40% VO_2max (Mean ± SEM)

Variables	40 rpm	60 rpm	80 rpm	100 rpm
VO_2 (l/min)	1.55 (±0.04)	1.55 (±.04)	1.63 (±0.04)	1.95 (±0.05)
HR (bpm)	111 (±3)	110 (±3)	115 (±3)	129 (±3)
SV (ml/beat)	116 (±6)	113 (±6)	112 (±6)	116 (±5)
CO (l/min)	11.51 (±0.29)	11.32 (±0.27)	11.63 (±0.31)	13.99 (±0.30)
MAP (mmHg)	96 (±2)	97 (±2)	94 (±2)	102 (±2)
SVR ($dyn \cdot s/cm^5$)	679 (±25)	697 (±28)	656 (±24)	590 (±17)
RPE (Borg)	8 (±0)	8 (±0)	8 (±0)	9 (±0)

3 Physiological and RPE Responses at Power Output Equated to 55% VO_2max
(Mean ± SEM)

Variables	40 rpm	60 rpm	80 rpm	100 rpm
VO_2 (l/min)	2.29 (±0.07)	2.20 (±0.06)	2.26 (±0.07)	2.61 (±0.08)
HR (bpm)	139 (±3)	134 (±3)	140 (±3)	153 (±3)
SV (ml/beat)	117 (±5)	118 (±5)	116 (±5)	115 (±5)
CO (l/min)	15.59 (±0.33)	15.15 (±0.31)	15.47 (±0.36)	17.14 (±0.38)
MAP (mmHg)	106 (±2)	102 (±2)	102 (±2)	103 (±2)
SVR (dyn·s/cm^5)	548 (±19)	540 (±16)	533 (±17)	487 (±13)
RPE (Borg)	12 (±0)	11 (±0)	11 (±0)	11 (±0)

TABLE 3.4 - Physiological and RPE Responses at Power Output Equated to 70% VO_2max (Mean ± SEM)

Variables	40 rpm	60 rpm	80 rpm	100 rpm
VO_2 (l/min)	3.10 (±0.08)	2.91 (±0.08)	2.95 (±0.08)	3.24 (±0.08)
HR (bpm	168 (±3)	161 (±3)	167 (±3)	173 (±3)
SV (ml/beat)	115 (±4)	114 (±4)	111 (±3)	114 (±4)
CO (l/min)	18.82 (±0.42)	18.01 (±0.44)	18.17 (±0.35)	19.69 (±0.46)
MAP (mmHg)	111 (±2)	110 (±2)	108 (±2)	107 (±2)
SVR dyn·s/cm^5)	476 (±13)	486 (±16)	474 (±14)	448 (±10)
RPE (Borg)	15 (±1)	13 (±0)	13 (±0)	14 (±1)

i) Oxygen Uptake and Cycling Velocity

Variations in oxygen uptake with cycling velocities were evident for the full range of power outputs. At all power outputs (low, medium and high), oxygen uptake for 60 rpm was lowest and was the highest at 100 rpm . Oxygen uptake at 60 and 80 rpm was not significantly different at any power output. For low and medium power outputs, oxygen uptake at 40 rpm was similar to 60 and 80 rpm; however, at high power outputs, oxygen uptake for 40 rpm was significantly higher than for 60 and 80 rpm, but significantly lower than oxygen uptake for 100 rpm. This is graphically demonstrated in Figure 3.1 and summarized in Table 3.5.

ii) Heart Rate and Cycling Velocity

Heart rates were significantly higher ($p<.05$) for 100 rpm than for any other cycling velocity over all power outputs. There was no significant difference between the heart rates elicited for 40, 60 and 80 rpm at all power outputs, although the heart rates produced at 60 rpm were the lowest over the range of power outputs. The means and SEM are displayed in Figure 3.2 and summarized in Table 3.6.

Figure 3.1 Oxygen Uptake Versus Cycling Velocity
(Missing Error Bars indicate SEM was too Small to Show)

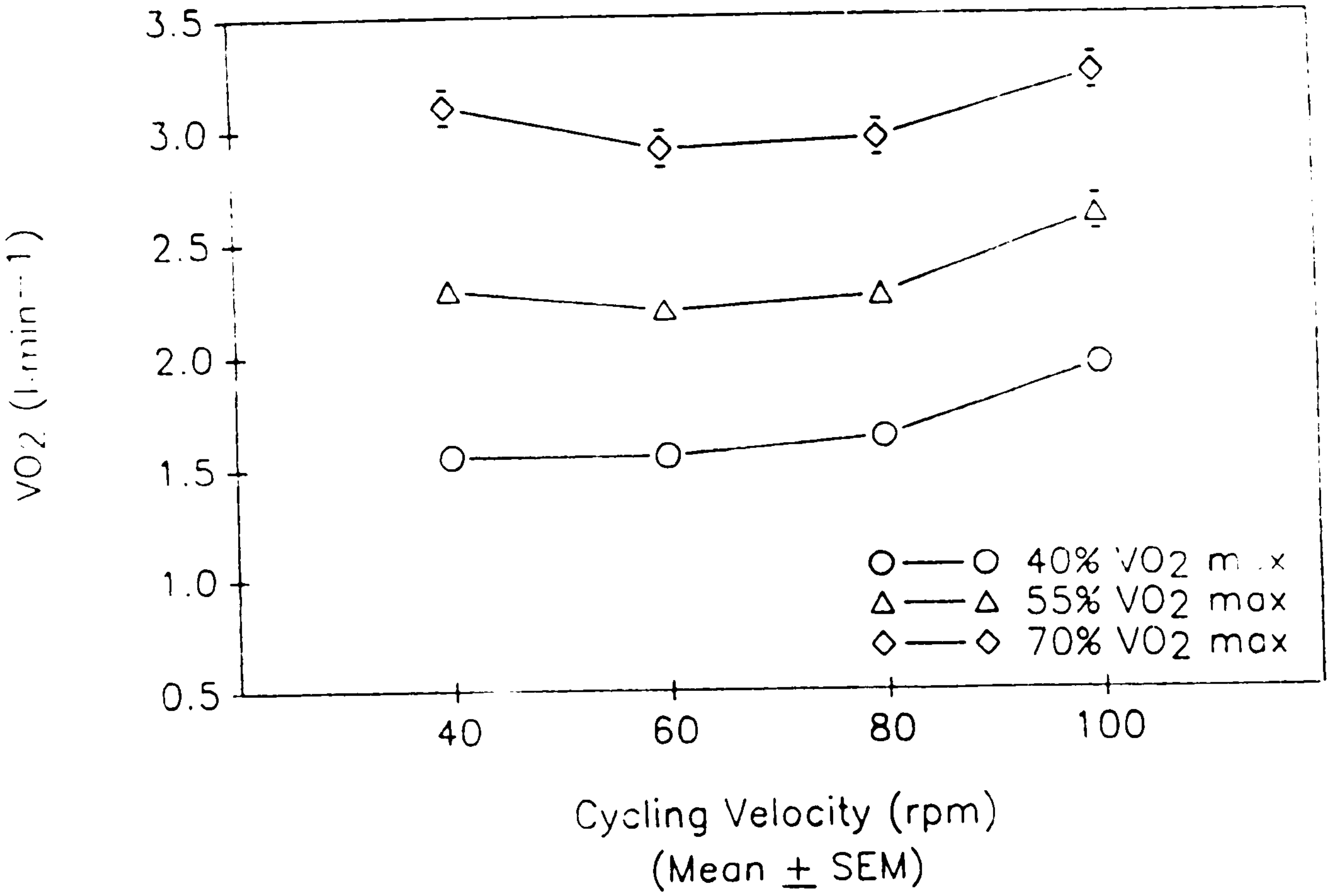


TABLE 3.5 - Significance and Means (±SEM) of Oxygen Uptake (l/min) and Cycling Velocities at Low, Medium and High Power Outputs

Power Output 40% (Low)	40 rpm	60 rpm	80 rpm	100 rpm
Mean (±SEM)	1.55 (±0.04)	1.55 (±0.04)	1.63 (±0.04)	1.95 (±0.05)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD **	
Power Output 55% (Medium)				
Mean (±SEM)	2.29 (±0.07)	2.20 (±0.06)	2.26 (±0.07)	2.61 (±0.08)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD **	
Power Output 70% (High)				
Mean (±SEM)	3.10 (±0.08)	2.91 (±0.08)	2.95 (±0.08)	3.24 (±0.08)
60 rpm	SD **			
80 rpm	SD *	NS		
100 rpm	SD **	SD **	SD **	

NS - NOT SIGNIFICANT AT 0.05 LEVEL
SD - SIGNIFICANT DIFFERENCE (** $P<0.01$; * $P<0.05$)

Figure 3.2 – Heart Rate Versus Cycling Velocity
(Missing Error Bars indicate SEM was too Small to Show)

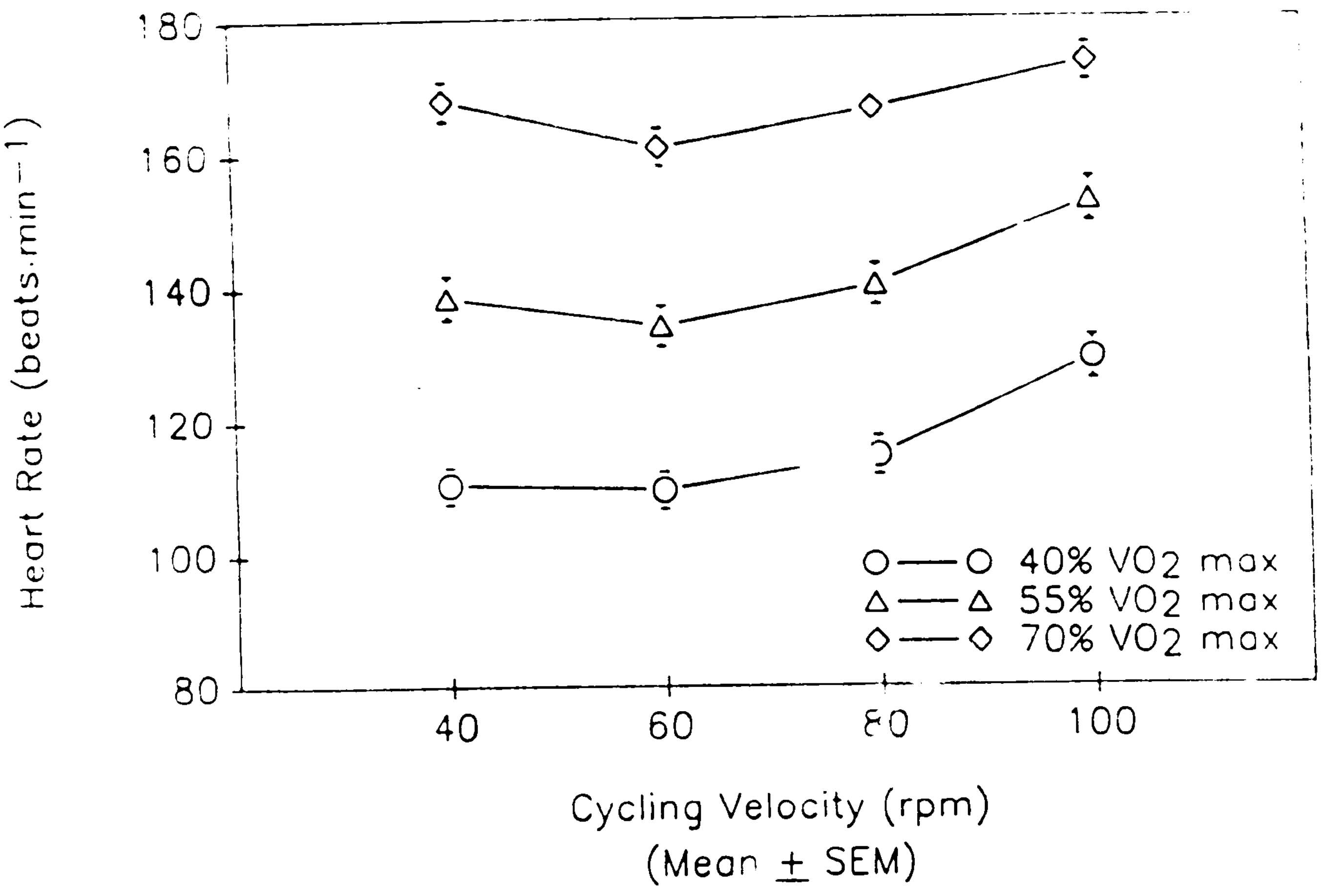


TABLE 3.6 - Significance and Means (±SEM) of Heart Rate (beat/min) and Cycling Velocities at Low, Medium and High Power Outputs

Power Output 40% (Low)	40 rpm	60 rpm	80 rpm	100 rpm
Mean (±SEM)	111 (±3)	110 (±3)	115 (±3)	129 (±3)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD **	
Power Output 55% (Medium)				
Mean (±SEM)	139 (±3)	134 (±3)	140 (±3)	153 (±3)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD **	
Power Output 70% (High)				
Mean (±SEM)	168 (±3)	161 (±3)	167 (±2)	173 (±3)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD *	SD **	SD *	

NS - NOT SIGNIFICANT AT 0.05 LEVEL
SD - SIGNIFICANT DIFFERENCE (** P<0.01; * P<0.05)

iii) Stroke Volume and Cycling Velocity

The stroke volumes elicited were not significantly different between cycling velocities at any power output. Nor were there any noticeable trends. Although the individual stroke volumes for medium power outputs were slightly higher than for low power outputs, the differences were only 2 ml/beat (not significant). A slight drop was evident in all stroke volumes at high power outputs in comparison to those elicted at medium power outputs with a difference of 3 ml/beat (not significant). These data of Table 3.7 are illustrated graphically in Figure 3.3.

iv) Cardiac Output and Cycling Velocity

At low, medium and high power outputs, 100 rpm resulted in higher cardiac outputs than any other cycling velocity (significantly different at .01 level in all cases). There was no significant difference between 40, 60 & 80 rpm at any power output. Although no significant difference existed, 60 rpm elicited the lowest cardiac output results at all power outputs, while 40 rpm required higher cardiac outputs than 60 & 80 rpm at medium and high power outputs. Means and

Figure 3.3 – Stroke Volume Versus Cycling Velocity
(Missing Error Bars indicate SEM was too Small to Show)

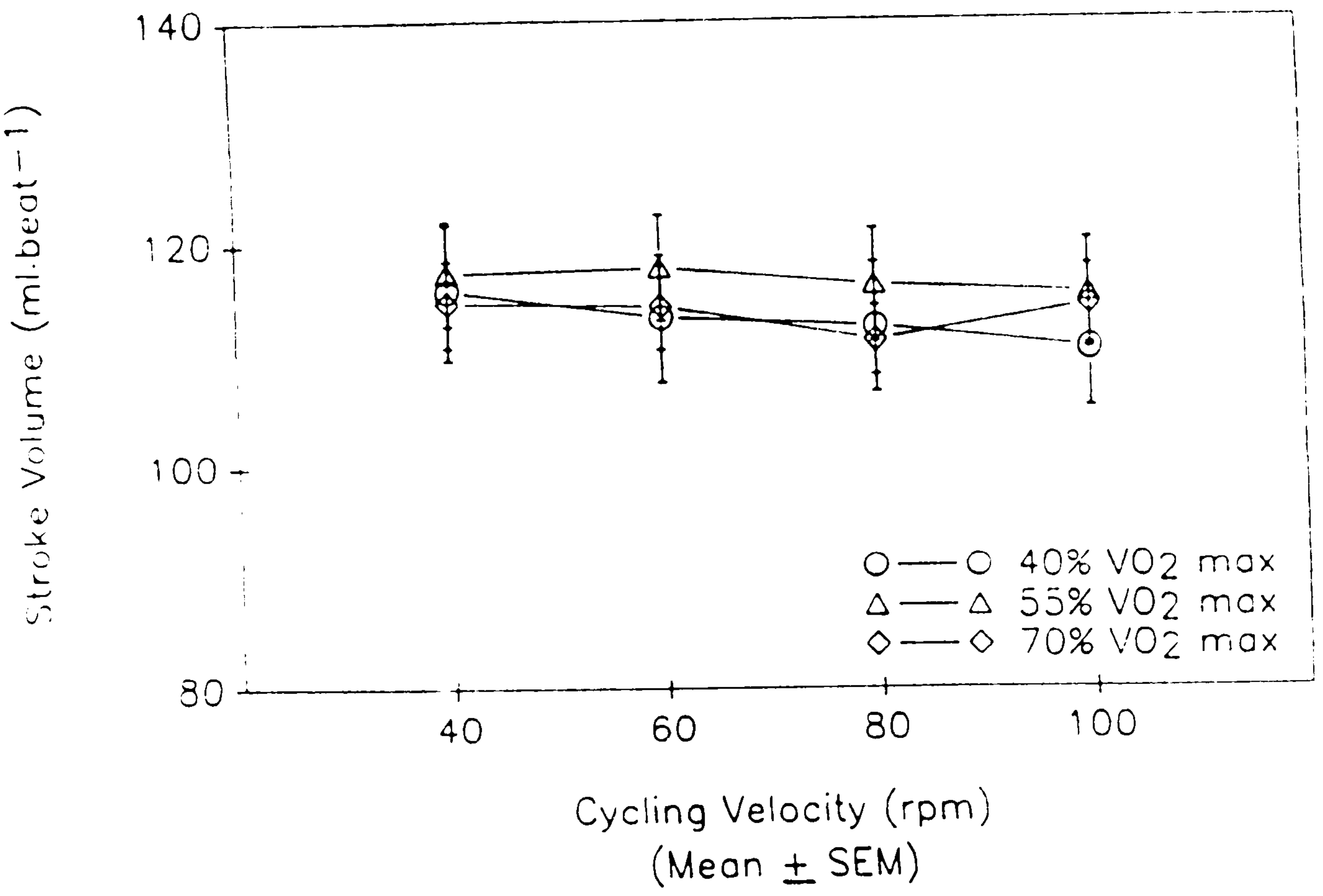


TABLE 3.7 - Significance and Means (±SEM) of Stroke Volume (ml/beat) and Cycling Velocities at Low, Medium and High Power Outputs

Power Output 40% (Low)	40 rpm	60 rpm	80 rpm	100 rpm
Mean (±SEM)	116 (±6)	113 (±6)	112 (±6)	116 (±5)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	NS	NS	NS	
Power Output 55% (Medium)				
Mean (±SEM)	117 (±5)	118 (±5)	116 (±5)	116 (±5)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	NS	NS	NS	
Power Output 70% (High)				
Mean (±SEM)	115 (±4)	114 (±4)	111 (±3)	114 (±4)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	NS	NS	NS	

NS - NOT SIGNIFICANT AT 0.05 LEVEL
SD - SIGNIFICANT DIFFERENCE (** $P<0.01$; * $P<0.05$)

significance differences are reported in Table 3.8 and illustrated in Figure 3.4.

v) Mean Arterial Pressure and Cycling Velocity

The only significant difference observed in mean arterial pressure was at low power output, where 100 rpm produced significantly higher pressures than other cycling velocities.

The only trend evident was that the mean arterial pressures at 40 rpm at medium and high power outputs were higher, although not significantly, than for any other cycling velocity. Mean arterial pressure increased with an increase in power output for all cycling velocities. Table 3.9 contains a summary of mean arterial pressure results and Figure 3.5 provides the means and SEM for these data.

vi) Systemic Vascular Resistance and Cycling Velocity

In all cases, systemic vascular resistance was lowest for 100 rpm; however, this was only significant for low and medium power outputs. At the low power outputs systemic vascular resistance was significantly lower for 80 rpm than for 60 rpm. At all power outputs, the trend was that 80 and 100 rpm produced

Figure 3.4 – Cardiac Output Versus Cycling Velocity
(Missing Error Bars indicate SEM was too Small to Show)

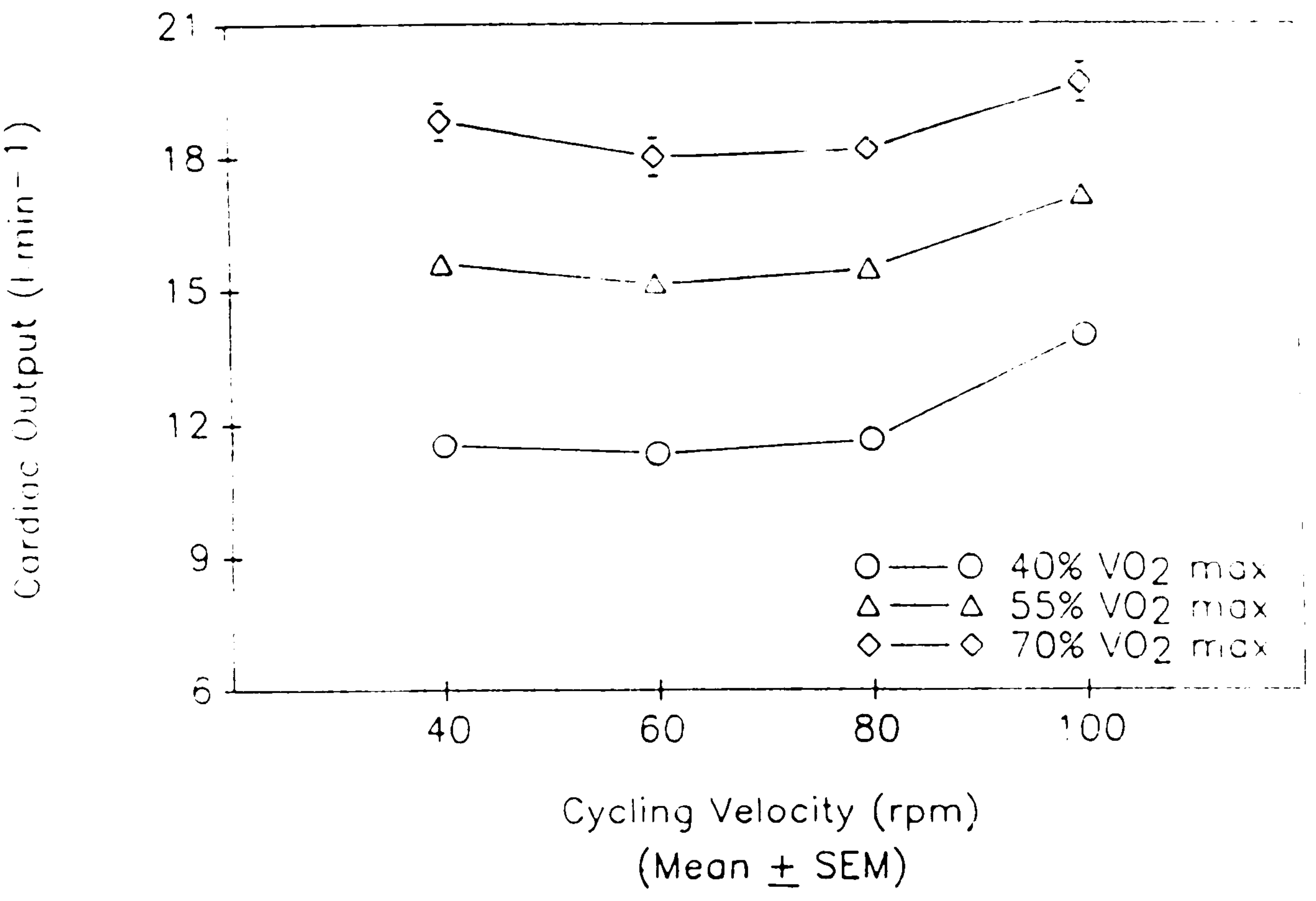


TABLE 8 - Significance and Means (±SEM) of Cardiac Output (l/min) and Cycling Velocities at Low, Medium and High Power Outputs

Power Output 40% (Low)	40 rpm	60 rpm	80 rpm	100 rpm
Mean (±SEM)	11.51 (±0.29)	11.32 (±0.27)	11.63 (±0.31)	13.99 (±0.30)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD **	
Power Output 55% (Medium)				
Mean (±SEM)	15.59 (±0.33)	15.15 (±0.31)	15.47 (±0.36)	17.14 (±0.38)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD **	
Power Output 70% (High)				
Mean ±SEM	18.82 (±0.42)	18.01 (±0.44)	18.17 (±0.35)	19.69 (±0.46)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD **	

NS - NOT SIGNIFICANT AT 0.05 LEVEL

SD - SIGNIFICANT DIFFERENCE (** P<0.01; * P<0.05)

Figure 3.5 - Mean Arterial Pressure Versus Cycling Velocity

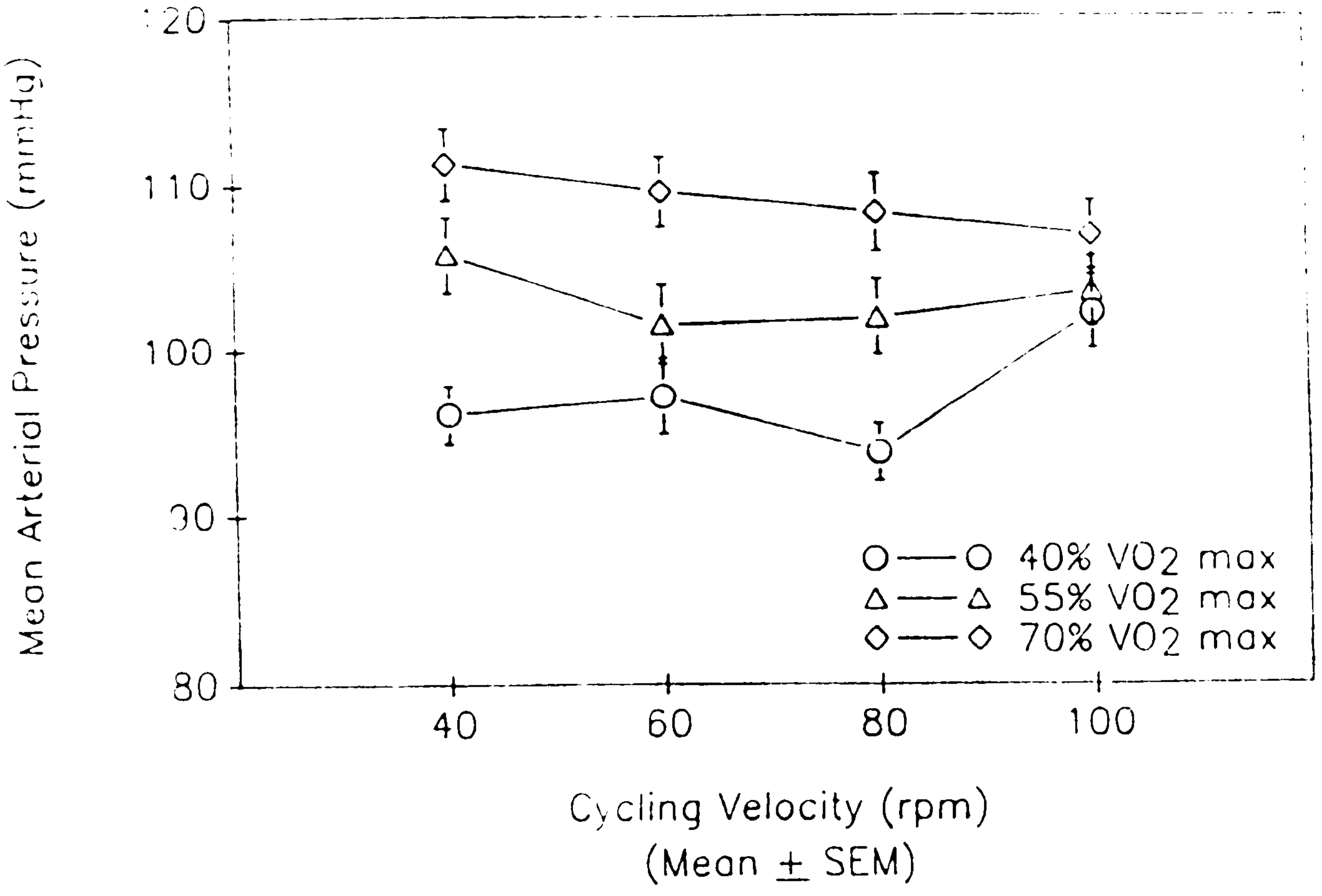


TABLE 3.9 - Significance and Means (±SEM) of Mean Arterial Pressures (mmHg) and Cycling Velocities at Low, Medium and High Power Outputs

Power Output 40% (Low)	40 rpm	60 rpm	80 rpm	100 rpm
Mean (±SEM)	96 (±2)	97 (±2)	94 (±2)	102 (±2)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD **	
Power Output 55% (Medium)				
Mean (±SEM)	106 (±2)	102 (±2)	102 (±2)	103 (±2)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	NS	NS	NS	
Power Output 70% (High)				
Mean (±SEM)	111 (±2)	110 (±2)	108 (±2)	107 (±2)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	NS	NS	NS	

NS - NOT SIGNIFICANT AT 0.05 LEVEL

SD - SIGNIFICANT DIFFERENCE (** P<0.01; * 0.05) lower (not significantly different) systemic vascular resistances than for 40 or 60 rpm. The summary of these results are contained in Table 3.10 and demonstrated graphically in Figure 3.6.

vii) Rate of Perceived Exertion and Cycling Velocity

The rate of perceived exertion for cycling velocities was only found to be significantly different at high power outputs. 40 rpm was perceived the most difficult and significantly higher than 60 or 80 rpm ($p<.01$), and 100 rpm was perceived more difficult than 60 or 80 ($p<.05$) but not different from 40 rpm. The trends that existed (not significantly different) were: at low power outputs perception of difficulty increased with cycling velocity, and at medium and high power outputs 80 rpm was perceived the least difficult. Table 3.11 contains the RPE results and Figure 3.7 graphically illustrates these data.

viii) Blood Lactate and Cycling Velocity

Venous lactate concentrations were only analyzed during pre-exercise and post-exercise. No significant differences existed during pre-exercise lactate results. The 5 minutes post-exercise lactates

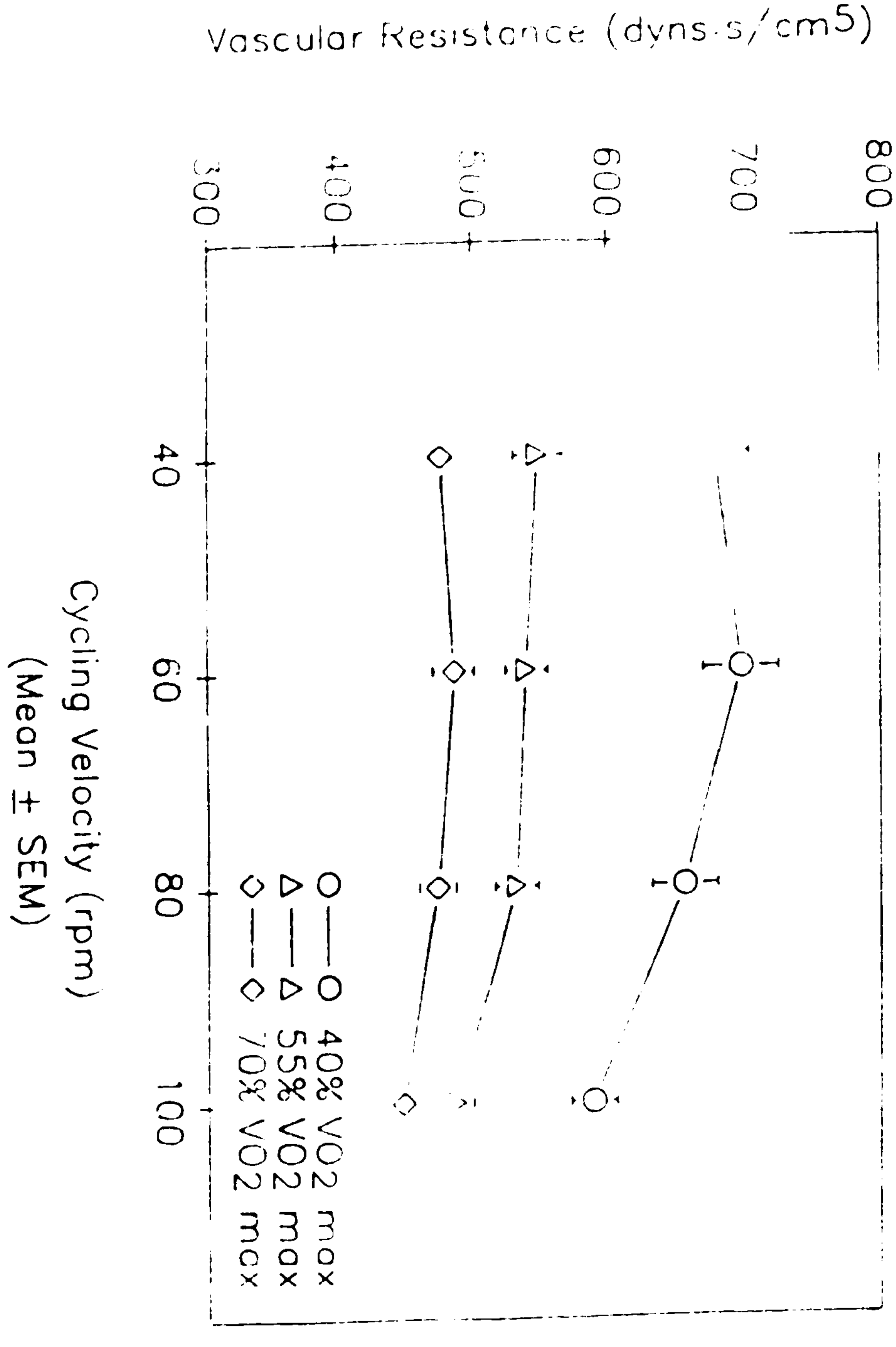


Figure 3.6 – Systemic Vascular Resistance Versus Cycling Velocity (Missing Error Bars indicate SEM was too Small to Show)

TABLE 3.10 - Significance and Means (±SEM) of Systemic Vascular Resistance (dyn·s/cm^5) and Cycling Velocities at Low, Medium and High Power Outputs

Power Output 40% (Low)	40 rpm	60 rpm	80 rpm	100 rpm
Mean ±SEM	679 (±25)	697 (±28)	656 (±24)	590 (±17)
60 rpm	NS			
80 rpm	NS	SD *		
100 rpm	SD **	SD **	SD **	
Power Output 55% (Medium)				
Mean (±SEM)	548 (±19)	540 (±16)	533 (±17)	487 (±13)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD *	
Power Output 70% (High)				
Mean (±SEM)	476 (±13)	486 (±16)	474 (±14)	448 (±10)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	NS	NS	NS	

NS - NOT SIGNIFICANT AT 0.05 LEVEL
SD - SIGNIFICANT DIFFERENCE (** P<0.01; * P<0.05)

Figure 3.7 – Rating of Perceived Exertion Versus Cycling Velocity (Missing Error Bars indicate SEM was too Small to Show)

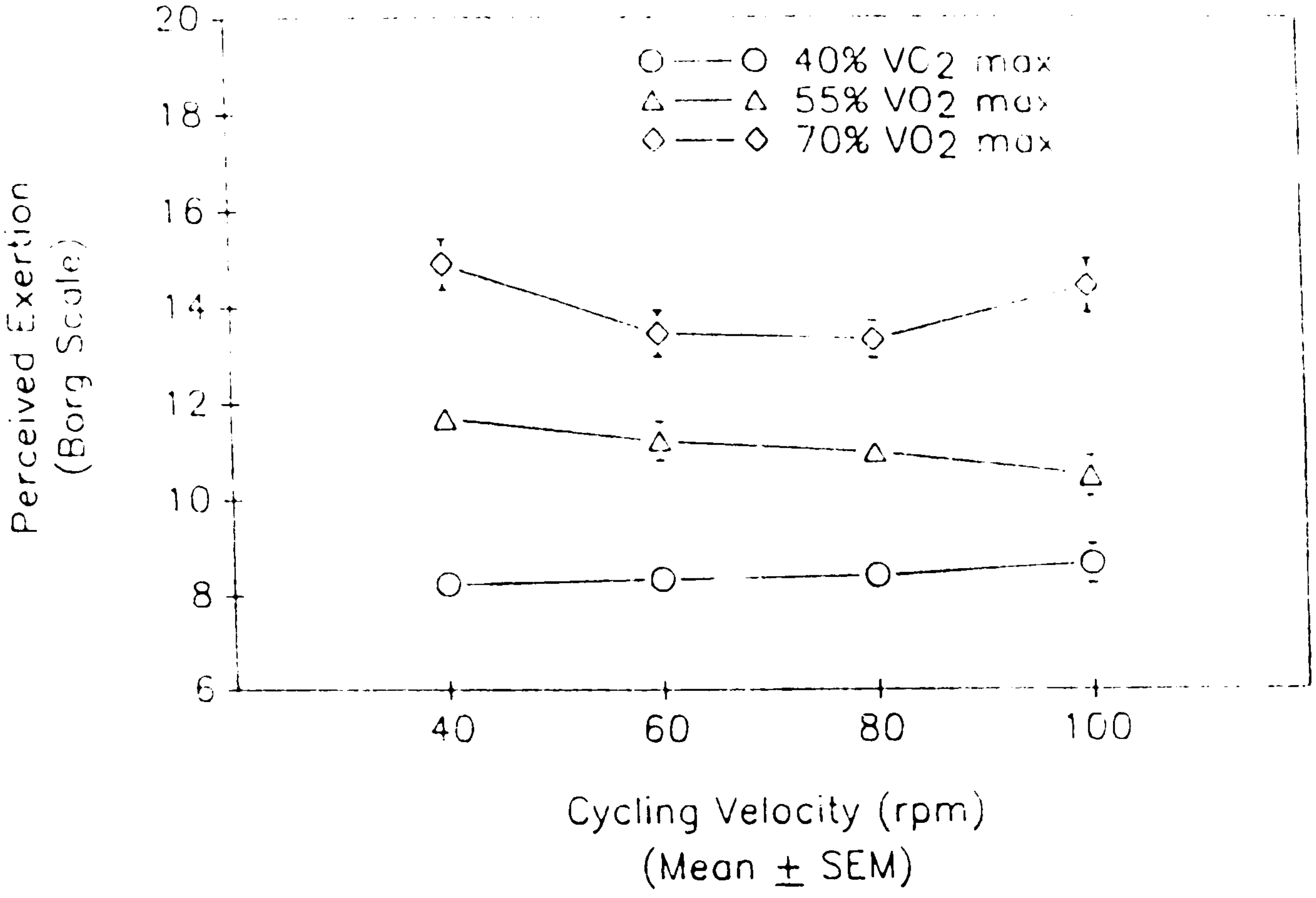


TABLE 3.11 - Significance and Means (±SEM) of Rate of Perceived Exertion (Borg Scale) and Cycling Velocities at Low, Medium and High Power Outputs

Power Output 40% (Low)	40 rpm	60 rpm	80 rpm	100 rpm
Mean ±SEM)	8 (±0)	8 (±0)	8 (±0)	9 (±0)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	NS	NS	NS	
Power Output 55% (Medium)				
Mean (±SEM)	12 (±0)	11 (±0)	(±0)	11 (±0)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	NS	NS	NS	
Power Output 70% (High)				
Mean (±SEM)	15 (±1)	13 (±0)	13 (±0)	14 (±1)
60 rpm	SD **			
80 rpm	SD **	NS		
100 rpm	NS	SD *	SD *	

NS - NOT SIGNIFICANT AT 0.05 LEVEL
SD - SIGNIFICANT DIFFERENCE (** $P<0.01$; * $P<0.05$)

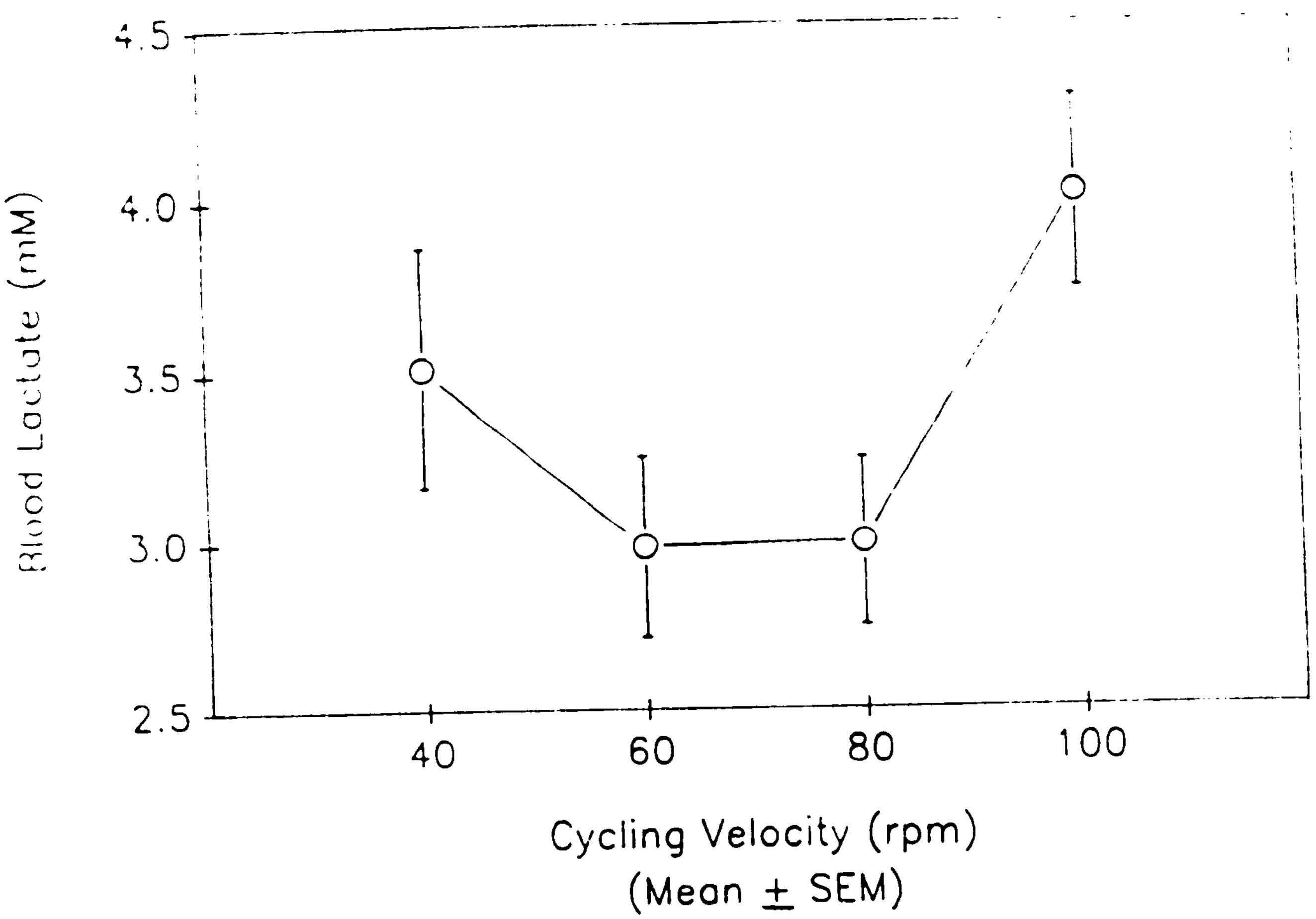
were analyzed to indicate the accumulative response over three workloads to each cycling velocity (that is after cycling 4 minutes at Low, 5 minutes at medium and 5 minutes at high power outputs for each cycling velocity). Lactate accumulation in the blood was significantly higher for 40 and 100 rpm ($p<0.01$) While no significance difference existed in blood lactate levels for cycling rates of 60 and 80 rpm; 100 rpm levels were elevated over those for 40, 60 and 80 rpm ($p<0.01$). The means (±SEM) and location of significant differences are provided at Table 3.12 and hown in Figure 3.8. No significant differences existed between pre-exercise blood lactate concentrations.

TABLE 3.12 - Significance and means (±SEM) of blood lactates (mM) and cycling velocities 5 minutes post-exercise

Power Output 5 min post Exercise	40 rpm	60 rpm	80 rpm	100 rpm
Mean (±SEM)	3.51 (±0.35)	2.98 (±0.27)	2.99 (±0.25)	4.01 (±0.28)
60 rpm	SD **			
80 rpm	SD **	NS		
100 rpm	SD **	SD **	SD **	

NS - NOT SIGNIFICANT AT 0.05 LEVEL
SD - SIGNIFICANT DIFFERENCE (** P<0.01; * P<0.05)

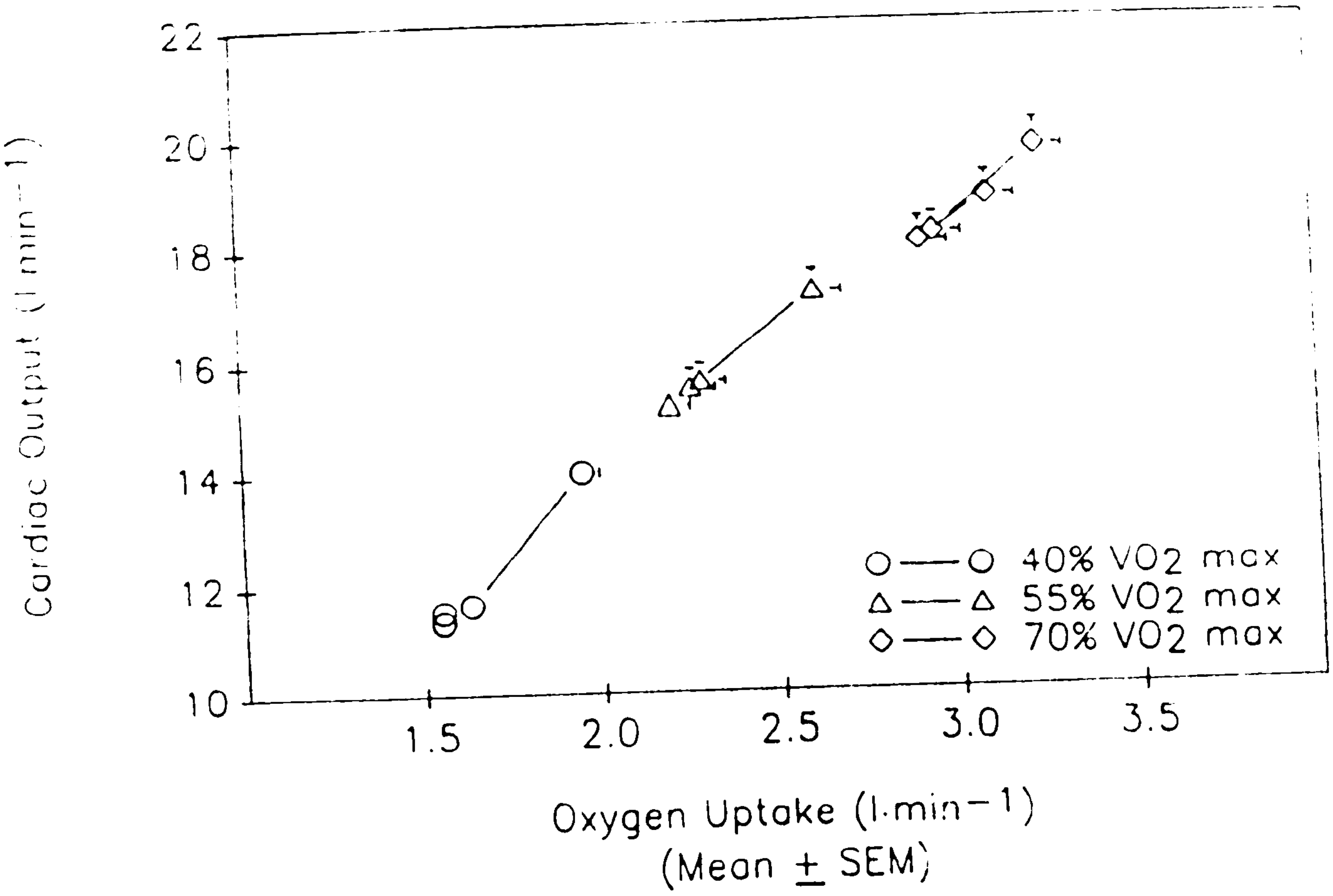
Figure 3.8 – Blood Lactate Versus Cycling Velocity
(5 minutes Post Exercise)



ix) Cardiac Output and Oxygen Consumption

The graph in Figure 3.9 compares oxygen consumption to cardiac output at every workload for each cycling velocity. The slope of the line of oxygen uptake to cardiac output is least at 100 rpm and greater at 40 rpm and greatest at 60 and 80 rpm. No significant differences existed between the regression lines.

Figure 3.9 – Cardiac Output Versus Oxygen Uptake
(Missing Error Bars indicate SEM was too Small to Show)



CHAPTER IV

DISCUSSION

There have been numerous studies investigating cycling velocities and various parameters such as heart rate, oxygen uptake, ventilation rate, blood lactate, perceived exertion, glycogen depletion and mechanical efficiency. Many studies (Bannister and Jackson, 1967; Buchanan and Weltman, 1985; Gaesser and Brooks, 1973; Hess and Seusing, 1962) have used small sample sizes. Others have studied only top caliber athletes (Bannister and Jackson, 1967; Buchanan and Weltman, 1985; Hagberg et al., 1981). Some investigators have studied a similar sample to that of the present study (healthy, active, young males) and found contradictory results. Gueli and Shephard (1976) found no significant difference in heart rate or oxygen uptake for cycling velocities between 60 and 85 rpm at a power output equated to 60% of VO_2max. Michielli and Stricevic (1977), on the other hand, found 80 rpm produced higher heart rates than 60 rpm (significant at 0.01 level).

Consequently, this study was undertaken to investigate cardiovascular and perceived exertion responses to 4 cycling velocities over a range of power outputs. The design permitted the evaluation of the separate effects at low, medium and high power outputs for cycling velocities of 40, 60, 80 and 100 rpm.

The original hypothesis was that cardiovascular function at submaximal loads would not be altered by varying the cycling velocity. It was found that not all velocities produced equivalent cardiovascular responses but rather that certain velocities produced results that were not significantly different (ie equivalent responses) and placed lower demands on the cardiovascular system (optimal cycling velocities). Both 60 and 80 rpm were not significantly different in any physiological or perceived exertion response (except for systemic vascular resistance at low power output) and placed lower demands on the cardiovascular system than any other investigated cycling velocity. No other two cycling velocities demonstrated this equivalency.

A. OXYGEN UPTAKE

Cycling velocities of 60 and 80 rpm at all power outputs were most efficient, that is, lower oxygen uptake values (significant at the 0.05 level) Oxygen uptake for low and medium power outputs were equivalent (no significant difference) at 40, 60 and

80 rpm, while 100 rpm required higher oxygen uptake ($p<0.01$) at all power outputs. Cycling velocities of 60 and 80 rpm resulted in lower oxygen uptake values at high power outputs. Other researchers support these findings; Eckermann and Millahn, 1967; Lollgen et al., 1980; Pandolf and Noble, 1973, all found that cycling velocities of 40 to 80 rpm at low and medium power outputs are optimal (that is, require lower oxygen uptake). When high power outputs are included the optimal cycling velocities are reduced to 60 to 80 rpm (Boning et al., 1984; Buchanan and Weltman, 1985; Coast et al., 1986).

Increases in oxygen uptake at high cycling velocities for all power outputs and at low velocities at high power outputs appeared to be caused by different mechanisms. A linear increase in oxygen uptake occurs with an increase in work intensity (Mitchell, 1985). Therefore, the difference in oxygen uptake over cycling velocities at equivalent power output must be a result of inequalities in external work (such as differences in fiber recruitment, body stabilizer utilization, muscle mass involvement and effective force).

The increased oxygen uptake at 100 rpm for all power outputs appeared to be a result of increased use of body stabilizing muscles of the trunk and upper body. Suzuki (1979) found that a predominance of fast twitch (FT) fibers are employed at high cycling rates. He suggests that slow twitch (ST) fibers were unable to cross-bridges (between actin and myosin) at faster cycling rates, and the result was higher oxygen uptake. Tankano (1987) and Hagberg et al. (1981) agreed that the static component (the increased use of body stabilizing muscles) required to cycle at higher cycling velocities required increased oxygen uptake.

At 40 rpm and high power output the higher oxygen uptake was possibly caused by increased upper body and trunk muscle involvement needed to maintain the cycling velocity and increased anaerobic metabolism resulting from a higher force for each pedal stroke (this is supported by increased blood lactate concentrations at this velocity). Suzuki (1979) found decreased efficiency of slow twitch fibers at high power output and increased anaerobic involvement. Kaneko et al. (1979) determined that low cycling rates at high power output resulted in increased external work.

B. HEART RATE

Heart rates were higher ($p<.01$) at 100 rpm for all power outputs. In terms of heart rate, cycling velocities of 40 to 80 rpm produced similarly lower heart rates at all power outputs. These heart rate results are well supported (Boning et al., 1984; Lollgen et al. 1980; Pandolf and Noble, 1973). This suggests that cycling velocities of 40 to 80 rpm are optimal in terms of heart rate.

The increase in heart rate associated with high cycling rates at all power outputs appeared to be the result of increased intensity of upper body and trunk stabilizing muscles (isometric contraction) and possibly the use of the less efficient fast twitch fibers (Suzuki, 1979; and Hagberg et al.,1981). Coast et al. (1986) found no difference in circulating catecholamines or oxygen uptake although heart rates were increased at higher cycling rates which suggests that the difference may be due to increased venous return from the maximized pumping action of the legs. Since the heart pumps all blood returned to it an increase in venous return results in an increased cardiac output. Since stroke volume has reached a maximum or peak plateau, the increase in cardiac output has to come from increased heart rate (no significant difference in stroke volumes over all power outputs). It appears at 100 rpm that the circulatory system is driven by venous return which results in creased cardiac output (since the healthy heart distributes all the blood returned to it) and thus heart rate is increased. This suggests that there is less oxygen extracted during each circulation of the blood.

C. CARDIAC OUTPUT

Stroke volumes were not significantly different between cycling velocities over the full range of power outputs. Stroke volume increased to peak values prior to 40% VO_2max and did not change with increased power outputs. Astrand and Rodahl (1986) suggest this occurs at or near 40% of VO_2max when heart rate has reached 110 to 120 beats per minute. Cardiac output, on the other hand, increased with power outputs and varied with cycling velocities. The cardiac outputs required to meet the demands of 100 rpm at all power outputs were higher ($p<.$ Consequently, the optimal cycling velocities in terms of minimizing cardiac output were 40 to 80 rpm for all power outputs. Although not significantly different, cardiac outputs at high power outputs for 40 rpm were elevated by more than 60 ml/minute in comparison to 60 and 80 rpm.

As suggested in heart rate response, the faster pumping action of the legs increased venous return which in term increased cardiac output. Added to this is the increased oxygen uptake required to supply the stabilizing muscle which also increased cardiac output.

D. BLOOD PRESSURE

Systolic blood pressure increased as power outputs increased. At low power outputs systolic blood pressure was much higher for 100 rpm ($p<.01$) than for other cycling velocities. For low and medium power outputs, the lowest pressures (not significantly different) were produced at 40 rpm.

No trend and major differences were observed in diatolic blood pressures over the cycling velocities and power outputs.

Significantly higher ($p<.01$) mean arterial pressures occurred at low power outputs by 100 rpm. No other significant differences existed in mean arterial pressures, but 40 rpm at medium and high power outputs caused the highest pressures. The fact that both mean arterial and systolic blood pressures were elevated at low power output for 100 rpm, but not at medium and high power outputs, suggest that the reflex neural system was not active. And that the circulatory system is being driven primarily by venous return and central command (Mitchell, 1985). It appears that blood pressures are elevated for 100 rpm at medium and high power outputs as a possible result of increased vasodilation (likely due to increased involvement of the upper body and trunk muscles). Astrand (1960) found cardiac output related to external workload but exercise vasodilation dependent on the muscle mass involved (amount and intensity).

Major differences in systemic vascular resistances occurred at low and medium power outputs. The lowest systemic vascular resistances were caused at 100 rpm (p<.01). Systemic vascular resistances at 80 rpm were elevated (p<.05) over all others. No differences in systemic vascular resistance occurred at high power output; however, the trend over all power outputs was that systemic vascular resistances for 100 rpm were lower.

In exercise that employs a large muscle mass, more extensive vasodilation tends to reduce systemic vascular resistance while large increases in cardiac output tend to increase systemic vascular resistance (Lewis et al., 1983). It would appear that larger increases in systemic vascular resistance should occur at 100 rpm at all power outputs and at 40 rpm at high power outputs because oxygen uptake was increased. This is not the case, in fact, at these cycling velocities while oxygen uptake increased significantly and cardiac outputs were elevated, systemic vascular resistance was reduced. This would suggest that in part, a larger muscle mass is being employed. Observation of the subjects tends to support this. At 100 rpm, the subjects moved more and used more stabilizing muscles. At 40 rpm and high power outputs, the subjects appeared to use more upper body and trunk movement. Consequently, reduced systemic vascular resistance at 40 rpm supports the proposition that extensive vasodilation occurred and could be a result of increased muscle mas involvement.

E. BLOOD LACTATES

Blood lactate concentrations were obtained 5 minutes after the subjects had performed at high power outputs (that is after 4 minutes at low, 5 minutes at medium, 5 minutes at high power outputs and 5 minutes of recovery). Blood lactate levels were elevated (p<.01) for 40 and 100 rpm over those for 60 and 80 rpm. Lactate levels for 100 rpm were elevated over those at 40 rpm (p<.01). The lactate results were identical to the oxygen uptake results at high power outputs. These results are similar to those of Hagberg et al. (1981), in that the cycling velocities that are most economical (lowest oxygen uptake) produced the lowest blood lactate levels. Buchanan and Weltman (1985) using 60, 90 and 120 rpm found significantly lower ($p < .05$) blood lactate concentrations at 60 and 90 rpm in comparison to 120 rpm.

The elevation of blood lactates at high power output for both fast and slow velocites is more complex. At high power output and low cycling velocities the increase appears to be a result of the high anaerobic component (Hagberg et al., 1981) and due to restricted blood flow caused by the high force required for each pedal stroke (Suzuki (1979). At high velocities the increased use of body stabilizing muscles appears to be sufficient to increase blood lactate levels which supports increased anaerobic involvement. It appears that 60 and 80 rpm are more economical cycling velocities (lower cardiovascular demand) and energy requirements can be met by the aerobic metabolism possibly because of reduced blood occlusion, hence increased oxygen exchange.

F. RATE OF PERCEIVED EXERTION

Cycling velocities were not perceived different at low and medium power output. These finding are not consistent with physiological responses. In addition, there were no similarities in the trends. At high power outputs, 60 and 80 rpm were perceived the easiest ($p<0.05$) and 40 and 100 rpm were perceived the most difficult. At high power outputs, perceived exertion varied with two physiological responses to cycling velocities (blood lactate and oxygen uptake). This would indicate that for physically active athletic males, perceived exertion responses at high power outputs are more indicative of blood lactate levels and oxygen uptake than any other measured physiological variable.

G. GENERAL DISCUSSION

Although there is much disagreement as to which cycling velocities place minimal demands on the cardiovascular system (optimal velocity), many authors suggest that an optimal range exists. This study clearly demonstrated that 60 to 80 rpm are within the optimal range and that 40 and 100 rpm are outside the optimal range. While there is support for the present findings (Boning et al., 1984; Buchanan and Weltman, 1985; Coast et al., 1986; Gueli and Shephard, 1976), other cycling velocities have received support as being the least demanding on the cardiovascular system. Some investigators have found 40 to 80 rpm to be most efficient cycling velocities, that is reduced oxygen uptake (Eckermann and Millahn, 1966; Lollgen et al., 1980; Pandolf and Noble, 1973). Hagberg et al. (1981) found higher velocities (72-100 rpm) more efficient as did Moffatt and Stamford (1978; 80-97 rpm). Still other investigators have found lower velocities to be more efficient (Hess and Seusing, 1962: 40 to 60 rpm and Michielli and Stricevic, 1977: 40 to 60 rpm). Closer scrutiny is required of those studies which used a wider range of cycling velocities. Eckermann and Millahn (1967) used power output of low and medium range and found results that are consistent with the present findings, that is at low and medium power outputs no signficant differences existed between physiological responses to cycling velocities of 40 to 80 rpm. Pandolf and Noble (1973), using extremely fit subjects and a small sample, found no significant difference between oxygen uptake between 40, 60 70 and 80 rpm; however, at high power output 40 rpm resulted in increased oxygen uptake (the difference was not significant but may have been if a large sample had been investigated).

Therefore, the findings that at low and medium power outputs 40 to 80 rpm were similar and the least stressful in terms of physiological responses is well supported (Boning et al., 1984; Buchanan and Weltman, 1985; Eckermann and Millahn, 1967; Gueli and Shephard, 1976). The support for 60 to 80 rpm producing similar physiological responses, is also quite strong (Boning et al., 1984; Bannister and Jackson, 1976; Buchanan and Weltman, 1985; Gueli and Shephard, 1976).

The value of the present study is the observation of more cardiovascular variables. The addition of cardiac output, mean arterial pressure and systemic vascular resistance measures to the complex interaction, provided additional insight. While oxygen uptake and blood lactate at 40 rpm and higher power outputs were significantly higher than 60 or 80 rpm, no other physiological response demonstrated this. In fact, for cardiac output, stroke volume, heart rate and mean arterial pressure, no significant differences existed between cycling velocities of 40 to 80 rpm. This is not consistent with the suggestion by Lewis et al. (1983) that a 1:1 relationship exists between systemic oxygen transport and utilization. These findings would suggest that the rate of muscle contraction (due to cycling velocity) might alter this proposed relationship. east et al. (1986) ruled out increasing resistance per pedal revolution, or the difference in fiber type as probable causes of increased blood lactate concentration and oxygen uptake at lower rates. They suggest the difference is due to increased metabolic costs of moving the muscles at greater or lesser than optimal speed. The increased metabolic cost in turn resulted in increased oxygen uptake and blood lactate levels at a given power output. Hagberg et al. (1981) suggest the possible cause of increased in oxygen uptake at cycling velocities which are not optimal might be a result of the employment of additional muscles to complete the task (that is of muscles to stabilize the body). Observation of subjects in the present study tends to confirm Hagberg et al.'s (1981) suggestion. At high cycling rates body stabilizers appear to play a greater role, with added upper body rigidity (isometric contraction of upper body muscles). At high power outputs and low velocity (40 rpm), more gross movement of trunk and the inclusion of the arm and shoulder muscles appeared necessary to maintain the cycling rate. In both situations, a higher intensity of work for body stabilizing muscles was employed and thus a probable increase in oxygen uptake was required. Increased work was accompanied by elevated blood lactate levels and likely increased vasodilation. In this study, it would appear that there was not an equivalent increase in cardiac output to meet the increased oxygen uptake. Coast et al.

(1986) found that although oxygen uptake varied with cycling velocities, circulatory catecholamines did not. They suggested that the difference in cycling velocities may not been sufficient to elicit a differential in norepinephrine response in spite of small oxygen uptake differences. The present data tends to support the findings of Coast et al. (1986), in that, cardiac outputs were not significantly larger with small increases in oxygen uptake (40 rpm compared to 60 and 0 rpm), but in larger oxygen uptake differences (100 rpm compared to 40 to 80 rpm) cardiovascular output was significantly different.

While cyclists tend to suggest that they feel most efficient at high cycling rates, most researchers have found lower rates (60 to 80 rpm) more efficient. As Hagberg et al. (1981) and Coast et al. (1986), suggest, preferred cycling rate may coincide with the predominance of muscle fiber type and vary from individual to individual. Previous research tends to suggest that a true variance exists in the most economical rate for each individual. It is further suggested that in normal individuals not trained or genetically equipped for higher muscular contraction efficiency, slower cycling velocities may be more efficient.

G. SUMMARY

This study clearly demonstrates that an equivalence in cycling velocities for most cardiovascular responses does exist over a wide range of power outputs for young physically active males. Additionally, there are cycling velocities which are beyond this range. That is, 60 and 80 rpm produce very similar cardiovascular responses which are the most economical (lower cardiovascular demands) on the system. In contrast, 40 and 100 rpm are beyond the range of optimal cycling velocities.

At high power outputs (70% VO_2max), perceived exertion appears to be a good indicator of differences in both oxygen uptake and blood lactate concentrations.

From a practical standpoint, these data suggest that in submaximal testing (for example oxygen uptake prediction) on a cycle ergometer 60 and 80 rpm can be used interchangably for young, physically active males, and that beyond these velocities significant physiological variations may occur.

These data would suggest the need for further investigation in three areas. First, to determine if these finding are appropriate for untrained as well as trained. Work by Boning et al., (1984), suggests this is true for young untrained males. Secondly, to determine if gender is a factor. And thirdly, to investigate these physiological variables using numerous cycling velocities greater than 40 rpm and less than 100 rpm to identify the exact range of optimal cycling velocities.

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APPENDIX A

UNIVERSITY OF ALBERTA CONSENT FORM

I acknowledge that the research procedures described on the attached form and of which I have a copy, have been explained to me and that any questions that I have asked have been answered to my satisfaction. I have been informed of the alternatives to participation in this study. I also understand the benefits (if any) of joining the research study. The possible risk and discomforts have been explained to me. I know that I may ask now, or in the future, any questions I have about the study or the research procedures. I have been assured that personal records relating to these experimental protocols will be kept confidential and that no information will be released or printed that would disclose personal identity without my permission.

I understand that I am free to withdraw from the study at any time. I further understand that if the study is not joined, or if there is withdrawal from it at any time, the quality of medical care will not be affected.

The person who may be contacted about the research

is: ______________________________
NAME of PERSON to CONTACT

____
NAME of SUBJECT (print)

____
SIGNATURE of SUBJECT

____
Telephone # subject

____
Signature of Witness

____
Date

INFORMATION SHEET

TITLE OF PROJECT: Cardiovascular response to 4
different cycling ve[illegible]ities on
the bicycle ergometer

INVESTIGATORS: Dr. H.A. Quinney; Dr C.T.
Kappagoda; Dr. R. Macnab
Dr. S. [illegible]terson; Dr. R.G. Haennel
and Mr. [illegible].V. Et[illegible]nger

This project is designed to [illegible]ine the cardiovascular response to four di[illegible] cycling velocities and to provide a [illegible]line for cardiovascular response in normals to [illegible]imum stress test in normals.

The study consists of th[illegible] phases; a familiarization phase, a maximum [illegible]s test and a treatment phase. The familiarizat[illegible] phase is a one hour session during which you will [illegible] oriented to the testing equipment and procedures of the study. The stress test phase is a one hour session in which you will be required to pedal to exhaustion on the bicycle ergometer (VO_2max test). For this test you will rest for 30 minutes and then begin pedaling at 60 rpm and a low resistance. The resistance will be increased every 2 minutes until you are unable to continue or maximum consumption has been reached. During this test respiratory gases and heart rate will be monitored continually; blood samples will be drawn from the anticupital vein prior to exercise and 5 min post-exercise. The third phase consists of 4 separate tests to be conducted 24 to 48 hours apart. On each occasion, you will cycle at a different cycling velocity (randomly assigned from 40, 60, 80 and 100 rpm). Each test will consist of 14 minutes of cycling with heart rate and respiratory gases being monitored continually; impedance cardiography measures will be taken six times during the test, and blood lactate and hematocrit samples taken up to five times from the anticupital vein. You will cycle for 4 minutes at a resistance equal to 40% VO_2max, 5 minutes at 55% and 5 minutes at 70%.

Any information obtained from this study will be kept confidential and will be released only to those conducting the study. This information will be made available to others only with your approval. The group results will be used in publication.

You will be required to make six visits to the

U of A Hospital (Cardiology stress lab) over approximately a 2 week period. Each visit will take approximately an hour.

Please be assured that your participation in this study is entirely voluntary and refusal or withdrawal from it will not jeopardize you in any way. The investigators would be pleased to clarify any concerns you may have prior to or during the study.

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THE UNIVERSITY OF ALBERTA

CARDIOVASCULAR AND PERCEIVED EXERTION RESPONSES TO CYCLING VELOCITIES

BY

ALLEN VICTOR ETTINGER

A THESIS
SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND
RESEARCH IN PARTIAL FULFILMENT OF THE REQUIREMENTS
FOR THE DEGREE OF
MASTER OF SCIENCE

DEPARTMENT OF PHYSICAL EDUCATION AND SPORT STUDIES

EDMONTON ALBERTA

SPRING 1989

ISBN 0-315-52822-2

THE UNIVERSITY OF ALBERTA

RELEASE FORM

NAME OF AUTHOR: ALLEN VICTOR ETTINGER

TITLE OF THESIS: CARDIOVASCULAR AND PERCEIVED EXERTION RESPONSES TO CYCLING VELOCITIES.

DEGREE FOR WHICH THESIS WAS PRESENTED: Master of Science

YEAR THIS DEGREE GRANTED: 1989

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FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled CARDIOVASCULAR AND PERCEIVED EXERTION RESPONSES TO CYCLING VELOCITIES submitted by Allen Victor Ettinger in partial fulfilment of the requirements for the degree of Master of Science.

....

Supervisor

....

....

....

DATE: Jun 20/78

DEDICATION

This thesis is dedicated to:

Geordie & Bill Knull, my second parents, who provided love, understanding and direction at a time it was needed and have continued to do so in my adulthood;

Mrs. Daisy Teeter, my grandmother, who demonstrated by personal example love, a superior work ethic and personal sacrifice for the pleasure of others;

Mr. Charles Edgar Ettinger, my father, who demonstrated dedication to task, personal management and powerful interpersonal relationships; and

David, Denis and Robert, my three sons, who provided support, understanding, assistance and self-denial to make this experience possible.

ABSTRACT

The purpose of this study was to investigate cardiovascular and perceived exertion (RPE) responses to cycling velocities (CR) of 40, 60, 80 and 100 rpm at low, medium, and high power outputs (40, 55 and 70% of VO_2max). Nineteen young healthy physically active males participated in the study consisting of a VO_2max test and four cycling velocity treatment tests on a constant load cycle ergometer.

The cycling velocity tests, conducted 24 to 48 hours apart, required the subject to cycle at one velocity for 4 minutes at low power output (PO), 5 minutes at medium PO and 5 minutes at high PO. Oxygen uptake (VO_2), heart rate (HR), RPE, blood pressure, stroke volume (SV) (determined by impedance cardiography), and cardiac output (CO) were monitored. Blood samples for venous lactate concentrations (BdLa) were drawn immediately pre-exercise and 5 minutes post-exercise.

Data were analyzed using a two-way analysis of variance (ANOVA) with repeated measures. A Newman-Kuels' post hoc test was conducted to determine individual mean differences.

At low and medium PO, the cycling velocities that produced the lowest demands on the cardiovascular system (optimal cycling velocities) were 40 to 80 rpm. No difference existed (p>.05) in VO_2, HR, SV CO, MAP AND RPE. Significantly higher cardiovascular responses (p<.05) occurred at 100 rpm (VO_2, HR, CO, MAP). At high PO 60 and 80 rpm placed the lowest demands on the cardiovascular system and no significant difference existed [illegible]05) in VO_2, HR, SV, CO, MAP, SVR and RPE. Both [illegible] and 100 rpm caused higher demands on the cardiovascular system. [illegible] BdLa and RPE were higher (p<.05) at 40 rpm than those at 60 and 80 rpm. At 100 rpm VO_2, HR, CO, Bdla and RPE were significantly higher (p<.05) than results at 60 and 80 rpm and all but RPE results were higher than those elicited at 40 rpm (p<.05).

The increased VO_2, CO, and Bdla at high cycling velocity (100 rpm) seemed to be a result increased static contraction of trunk and upper body muscles needed to provide stability. The possible cause of elevated VO_2 and BdLa for slow cycling velocity (40 rpm) at high PO was increased upper body and trunk muscle involvement and higher portions of anaerobic metabolism as a result of the larger force required for each pedal stroke.

The study demonstrated that for young healthy physically active males 60 and 80 rpm were the optimal

cycling velocities over a wide range of power outputs in terms of cardiovascular and metabolic responses. For low and medium PO, 40 to 80 rpm were found optimal.

The data suggest that 60 to 80 rpm can be used interchangably in submaximal testing over a wide range of power outputs in healthy active young males.

ACKNOWLEDGEMENTS

I would like to express my appreciation to a number of friends who have contributed to my graduate experience and in particular to the completion of this thesis:

The physical education and recreation graduate students who provided commradeship, support and an endless source of knowledge and assistance.

Marg Rees for her assistance and exceptional work ethics in data collection.

Chris Cummins for her tolerance, patients, friendship and assistance throughout the program.

Dr. Robert Haennel for his guidance, assistance and fruitful discussions.

Dr. Stu Petersen for his encouragement, invaluable advise, friendship and endless corrections.

Dr. Tissa Kappagoda for his encouragement, challenges, guidance, invaluable advise, direction and logistical support.

Dr. Art Quinney for ensuring an enriched graduate experience, direction, encouragement, support, challenge, invaluable advise and friendship.

The Department of National Defence who made this experience financially feasible.

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CHAPTER I

INTRODUCTION

Maximum oxygen uptake (VO_2max) has long been used by exercise physiologists as a measure of the ability to perform continuous physical work (Astrand and Rodahl, 1986; Glassford et al., 1965). Although some authors utilize the terms maximum aerobic power and aerobic capacity synonymously (Brooks and Fahey, 1984), others (Astrand and Rodahl, 1986; MacDougall et al., 1983) define 'aerobic' capacity as the total amount of adenosine triphosphate available for oxidation through aerobic processes while 'aerobic' capacity is defined as the maximum amount of oxygen that can be taken up per unit time, in spite of further increases in exercise intensity. The latter value is termed maximum oxygen uptake (VO_2max) and is measured in absolute terms in liters per minute or relative terms in milliliters per kilogram of body weight per minute.

Numerous methods of assessing maximal oxygen uptake have been developed (Astrand et al., 1959; Balke and Ware, 1956; Mitchell et al., 1958; Taylor, 1941). Maximum oxygen uptake is still considered one of the most objective measure in determining cardiovascular fitness (Shepherd and Shepherd, 1987).

If a precise measure of oxygen uptake is required, then direct assessment is necessary. If however, exact measure is not required or possible, then predictive VO_2max test may be used. Most predictive tests are based on the relationship that exists between oxygen uptake and one or more variables. The variable is monitored at submaximal workloads and then VO_2max is predicted from submaximal values (heart rate is frequently used). Submaximal assessment is appropriate if: precision is not essential; testing time is short; and/or subjects safety is a concern Direct determination of maximum oxygen uptake is expensive, time consuming and presents a potential risk to subjects. It requires expensive equipment, highly trained evaluators, a laboratory setting, and often restrictive selection of subjects.

As a result of these limitations, many investigators have attempted to develop submaximal tests which are safe, valid, reliable and less complex (Astrand, 1952; Astrand and Ryhming, 1954; Bruce et al., 1973; Margaria et al., 1954; Sjostrand, 1947).

Berggren and Christensen (1950) were amoung the first to find a linear relationship between heart rate (HR) and oxygen uptake (VO_2). They outlined the first conditions under which oxygen uptake could be predicted from heart rate emphasizing inter-individual differences due to age, sex, task and temperature. Although other physiological parameters have been investigated as indirect determinants of oxygen uptake (Astrand and Rodahl, 1986) they have been found to produce a larger margin of error than the use of heart rate. Consequently, heart rate has predominated as the best indirect predictor of oxygen uptake.

Numerous cycle ergometer tests have been developed to predict oxygen uptake (Andersen and Hermansen, 1965; Astrand, 1965; Sjostrand, 1947) from the measurement of heart rate and power output (PO). Formulae, nomograms, and graphing techniques which rely on the linear relationship between heart rate and power output (Astrand and Ryhming, 1954; Bruce et al., 1973) have been used.

Numerous investigators (Andersen and Hermansen, 1965; Astrand, 1952; Sjostrand, 1978) have compared submaximal estimation of maximal oxygen uptake with actual values and found the methods valid and reliable. Coefficients of variance of 5% to 15% for validity measures and 3% to 7% for reliability measures have been reported by these researchers.

However, most significant were the findings of Glassford et al. (1965) in the comparison of two maximal treadmill tests (Mitchell et al., 1958;

Taylor, 1941), one maximal cycle ergometer test (Astrand, 1952) and a submaximal cycle ergometer test (Astrand and Ryhming, 1954). They found the correlation of maximal oxygen uptake between values predicted by the submaximal test and any direct method was equivalent to that between any two of the direct methods. This and similar findings (Andersen and Hermansen, 1965; Astrand, 1965) resulted in acceptance of standards for cycle ergometry testing at the 16th World Congress on Sports Medicine in Hanover, Germany (June 1966) by the International Council of Sports Medicine and Physical Education (ICSPE) Research Committee for International Standardization in Ergometry (from Mellerowicz and Smodlaka, 1981). The primary recommendation was that cycle ergometer tests should be conducted at cycling velocities of 50 to 60 revolutions per minute (rpm) for International Standardization.

Since 1966 studies have been conducted by many researchers investigating the relationship between cycling frequency and oxygen uptake (Bannister and Jackson, 1967; McKay and Bannister, 1976), respiratory gases (Gueli and Shephard, 1976); heart rate (Eckermann and Millahn, 1967; Michielli and Stricevic, 1977); fiber recruitment patterns (Moffatt and Stamford, 1978; Seabury et al., 1977); cycling efficiency (Coast et al., 1986; Sjostrand, 1978; Takano, 1987); blood lactate (Burke et al., 1981; Buchanan and Weltman, 1985); efficiency (Gaesser and Brooks, 1975); perceived exertion (Pandolf and Noble, 1973; Cafarelli, 1978; Borg et al., 1985; Edwards et al., 1971; Ekblom and Goldbarg, 1971); or a combination of these variables (Lollgen et al., 1980; Gamberale, 1972; Hagberg et al., 1981; Boning et al., 1984).

These studies have included: investigation of few cardiovascular variables, small power output ranges, too few cycling velocities (CR), small samples and/or heterogenous samples. These authors have attempted to explain the differences in physiological responses to cycling velocities without investigating cardiac output (CO).

Since the primary cardiovascular adjustment results from the fact that oxygen uptake is the product of cardiac output and arteriovenous difference ($(a-vO_2$ diff; the difference in oxygen content of the blood entering and leaving the pulmonary capillaries), it is imperative to evaluate cardiac output. The investigation of cardiac output should provide an in depth understanding of the cardiovascular response to cycling velocities.

A. PURPOSE

The purpose of this study was to investigate cardiovascular and perceived exertion responses to cycling velocities of 40, 60, 80 and 100 revolutions per minute (rpm) at power output equated to 40%, 55% and 70% of maximum oxygen uptake (VO_2max) in young healthy active males. Nineteen males participated in the study which consisted of a VO_2max test and four treatment tests (one for each cycling velocity) on an electric constant load cycle ergometer.

B. HYPOTHESIS

The research hypothesis for this study was that no difference existed in any of the physiological or perceived exertion responses between the 4 cycling velocities at equivalent power outputs.

Statistical Hypothesis: Ho: $X - X = 0$

Hi: $X - X = 0$

CHAPTER II

METHODS AND PROCEDURES

A. SUBJECTS

Twenty-four healthy, physically active, non-smoking, male volunteers were subjects in this study; however, only 19 completed all phases (4 were injured precluding further participation and one moved away from the city). All subjects were physically active (training approximately three hours aerobically each week), were familiar with cycling and maximum oxygen uptake tests on the cycle ergometer. Subjects were fully informed of the purpose of the experiment and written consent was obtained prior to commencement of the study (Appendix A). The study protocol was approved by the Institutional Ethics Review Committee.

B. ENVIRONMENTAL CONDITIONS

Temperature has been demonstrated to affect heart rate, oxygen uptake and other physiological parameters (Rowell, Taylor and Wang, 1964; Toner et al., 1982). In this study relative humidity was not controlled, and temperature was monitored and found to be consistent (22 to 24 degrees celcius) during all testing. In the laboratory, relative humidity varied only slightly. Laboratory traffic and other distractions were minimized during all data collection to reduce possible effects on physiological parameters at low power outputs (Astrand, 1960; Taylor et al., 1955).

C. TESTING STATE STANDARDIZATION

All subjects were post absorptive for a mimimum of three hours and did not take part in any strenuous exercise or physical activity for twelve hours prior to testing. These factors have been shown to affect heart rate and cardiac output (Astrand and Rodahl, 1977; Taylor et al., 1963). Subjects were scheduled at the same time of day for every treatment test to reduce biological variability (Brooks and Fahey, 1984).

D. STUDY OUTLINE

The experiment consisted of 6 separate sessions in the laboratory (lab): a familiarization session; a maximum oxygen uptake test, and four treatment tests.

The familiarization session was a one hour session to brief and allow the subjects to become familiar with the lab and testing conditions. During this session subjects were exposed to mini-trials which allowed familiarization with the tus and experimental conditions (cycling frequency, impedance cardiography, respiratory gas analysis, and blood pressure [BP] determination).

The maximum oxygen uptake test (VO_2max) was conducted 24 hours after the familiarization sessions on the electrically braked constant load cycle ergometer.

The four treatment tests were conducted on 4 separate days, each at least 48 hours apart. The first treatment test for each subject was scheduled a minimum of 48 hours after the VO_2max test. In order to reduce intra-subject biological variability each subject was scheduled at the same time of day (Davies and Sargeant, 1975). During the treatment test, the subject cycled at one cycling rate (40, 60, 80 or 100 rpm). Subjects were randomly assigned to a specific treatment test order. With 24 possible permutations of the four cycling rates, each subject had a different exposure order to the treatment tests (see Appendix B). During the treatment test the subject cycled at the predetermined rate for 3 workloads; the first workload was cycling at a power output equated to 40% of VO_2max for 4 minutes; the second workload was cycling at 65% of VO_2max for 5 minutes, and the final workload was 5 minutes cycling at 70% of VO_2max.

E. TESTING APPARATUS

The equipment used in this study included : a constant load electric cycle ergometer (Model 740, Siemens-Elema, Siemens Electric Ltd., Mississuaga, Ontario); a magnetic cycling revolution counter (Cateve Micro Cyclocompter Model CC-6000, Tsuyama Co Ltd., Japan); Beckman Metabolic Measurement Cart (Model MMC-Cat554277, Advanced Technology Operations, Fullerton, CA); a Minnesota Impedance Cardiograph (Model 304B Surcom Inc., Minneapolis); 4 channel Gould ink recorder and amplifier (Model 8188-402, Ballainvillers, France), Gould Isolation Transformer (Model 882895-3, Gould inc., Cleveland, Ohio); Baumanometer Mercury Sphygmomanometer (Model Standby, WA Baum Co. Inc., Copiagne, New York) and stethescope ; cardiotachometer (Sport tester, Model 3000, Polar Electro, Kempele, Finland); a tape measure (mm), blood sampling equipment; Sybron Thermolyne Mixer (Model M-16715); Sovall General Lab Centrifuge (Model GLC-4, Dupont); and a UV/VIS Phillips Spectrophotometer (Model PU8800, Pye Unicam Ltd., Cambridge, England).

the same equipment was used for every subject during each test.

F. GENERAL METHODS

i) Stroke Volume and Cardiac Output Measurement

In this study stroke volume and cardiac output were measured using the Minnesota Impedance Cardiography Model 304B (Surcom Inc., Minneapolis). Impedance Cardiography is a non-invasive, atraumatic method of measuring heart rate and stroke volume from which cardiac output may be calculated. The technique involves the placement of self-adhesive disposible mylar-backed aluminum electrode bands around the neck and thorax. A weak frequency alternating current is passed through the outer two electrodes. The constant alternating current is undetectable by the subject and the frequency is so high it is incapable of stimulating the heart (Kubicek et al., 1966).

On each visit to the laboratory for testing, the subject reported at least three hours post-absorptive. The four bands of self-adhesive disposable mylar-backed aluminum electrode tape were placed around the neck and chest 30 minutes prior to any measurement. Two bands were placed around the neck, 3 to 5 cm apart the third around the trunk at the level of the xiphisternum and the fourth at a level just above the umbilicus. When connected the two outer electrodes transmitted a constant, sinusoidal, alternating current (4 ma RMS and 100 KHz) through the thorax and the changes in transthoracic electrical impedance were detected by the inner two electrodes (Figure 2.1). Mean total transthoracic impedance between the inner electrodes (Zo) was computed and displayed by the Impedance Cardiograph. Simultaneous recordings of the rate of change of impedance through each phase of the cardiac cycle (dZ/dt), were made through the electrocardiograph and phonocardiograph on a 4 channel ink recorder at a paper speed of 50 mm/s (Model 8188-402, Gould Inc., France). The heart rate, dZ/dt min, and left ventricular ejection time, were obtained from the recordings as shown in Figure 2.2. Calculation of stroke volume was made using the following equation:

$$\text{Stroke Volume} \quad \frac{P \times L^2 \times dZ/dt_{min} \times T}{Z_o^2}$$

where P = electrical resistivity of blood at body temperature ($P = 53.2e^{0.022H}$), H = hematocrit (%), L = average distance (cm) between the inner pair of electrodes measured at the anterior and posterior midline, Z_o = mean transthoracic impedance (ohms)

FIGURE 2.1

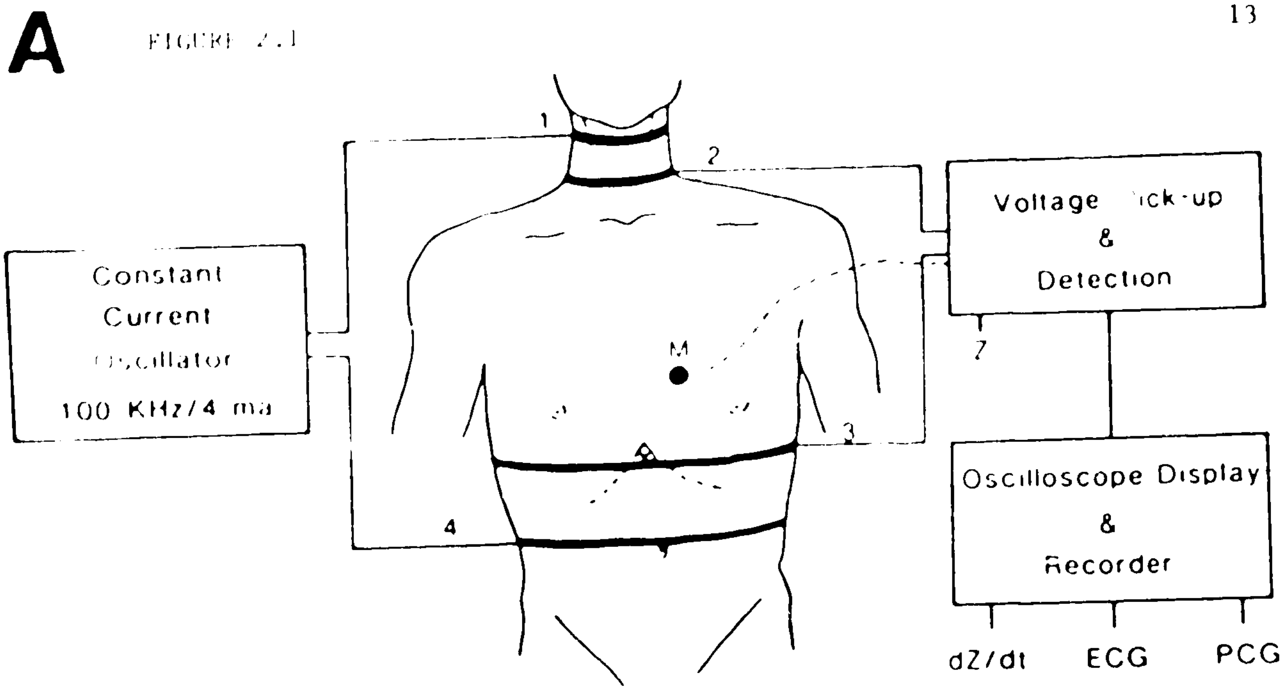
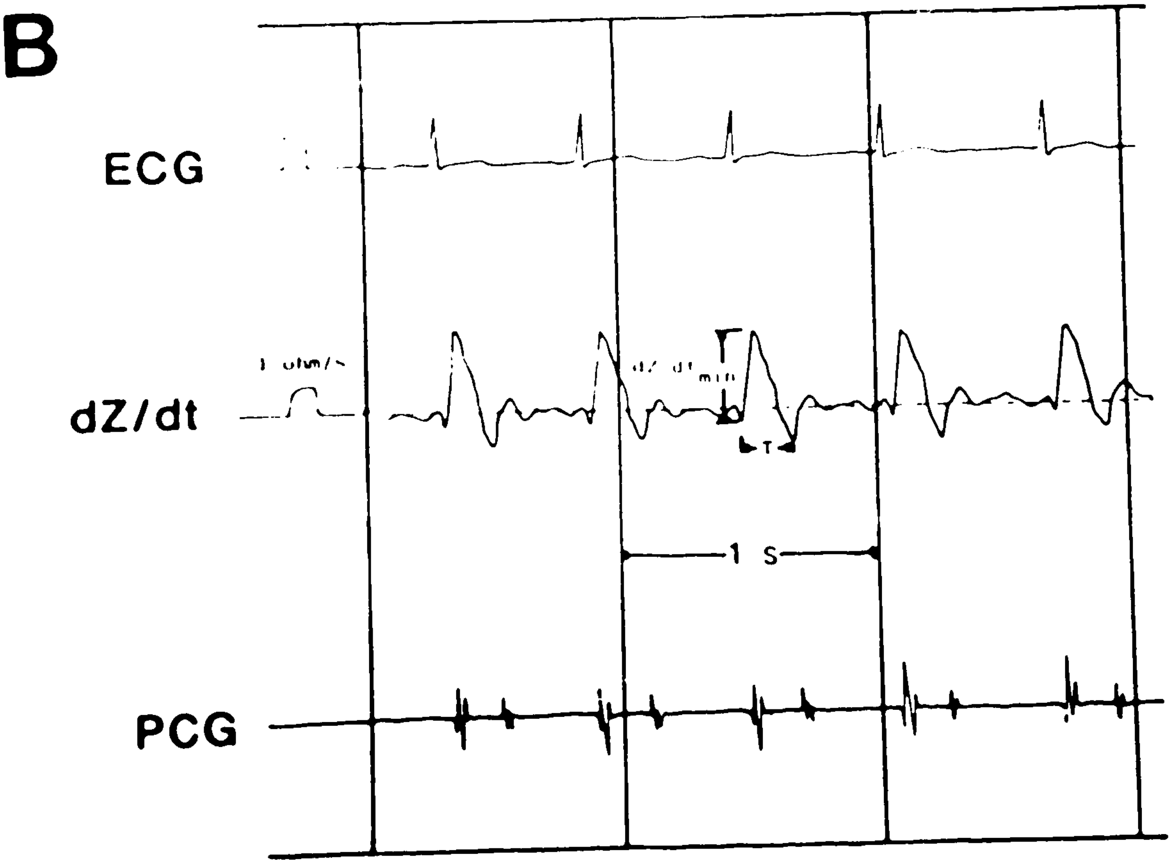


FIGURE 2.2



between the inner two electrodes, dZ/dt_{min} - minimum value for the rate of change of impedance (ohms) occuring during the cardiac cycle, T - left ventricular ejection time (s).

Recordings were made at rest and during exercise. It was found that movement caused by respiration and exercise introduced artifacts into the recordings. These artifacts were avoided by r[illegible]ring the subjects to stop all movement, remain [illegible]ess and hold their breath at normal end-expira[illegible] for approximately 5 seconds, while 5 to 10 cardiac cycles were recorded (Hetherington et al., 1985). Five cardiac cycles were used for the calculation of stroke volume. The hematocrit was measured from blood samples drawn from an antecubital vein immediately prior to exercise, and at 5 minutes post-exercise. The hematocrit value was interpolated between pre-test to post-test blood samples to correspond to the impedance cardiography recording.

ii) Measurement of Maximal Oxygen Uptake

Maximal aerobic power was determined by measuring oxygen uptake during a modified incremental exercise test (Thoden et al., 1983) on the cycle ergometer. The subject cycled at 60 rpm. The initial power output was 100 watts (W) with 50 watt increments added every two minutes.

The physiological measures obtained every 30 seconds were ventilation volume (Ve), volume of carbon dioxide produced (VCO_2), volume of oxygen consumed (VO_2), respiratory exchange ratio (R) and heart rate (HR). The peak oxygen uptake value obtained during the exercise test was recorded as VO_2max. Expired gases were collected continuously and analyzed every 30 seconds the Beckman Metabolic Measurement Cart. Standard gases of known concentration were used to calibrate the gas analyzers before and after each test. Heart rate was recorded every 30 seconds using the cardiotachometer and a simultaneous ECG strip was run on the impedance recorder (in the last fifteen seconds of each workload). Blood pressure was measured using a mercury sphygmomanometer at rest and during the last 30 seconds of each workload throughout the incremental test. Impedance cardiography recordings were taken at end of each workload using the Minnesota Impedance Cardiograph.

The exercise test continued until at least one of thefollowing end point criteria for VO_2max had been achieved: oxygen uptake increased less than 50 ml with increased workload; a heart rate of 90% of age predicted maximal heart rate was attained; or, the subject was unable to continue. Appendix C provides a detailed description of the maximum oxygen uptake test protocol.

iii) Workload Determination

In order to determine the power output necessary to equate to 40, 55 and 70% of VO_2max, the results of the VO_2max test were utilized. Oxygen uptake (l/min) was plotted against power output (watts) at each workload for every subject and the percentage of maximal oxygen uptake was determined (that is, the oxygen uptake equal to 40, 55 and 70% of VO_2max). The power outputs were interpolated from the graph at the required oxygen uptake for each subject. These were the power outputs, equated to 40, 55 and 70% of VO_2max, used for every treatment test.

iv) Treatment Protocol

Each subject attended four treatment tests, scheduled a minimum of 48 hours apart. Identical protocols were utilized for each treatment. The only difference between the treatments was the rate at which the subject cycled (40, 60, 80 or 100 rpm). The detailed treatment test protocol is contained in Appendix D.

In brief, the protocol consisted of the following: anthropometric measurement (height and weight); cardiac impedance measures; a 30 minute rest period, (seated); affixed the subject with headgear and mouthpiece for respiratory gas collection; collection of respiratory gas analysis for a 2 minutes rest period; resting physiological measures taken (heart rate, stroke volume, cardiac output and blood pressure); a blood sample drawn from an antecubital vein; an incremental cycle ergometer test; 1 and 3 minute seated recovery, physiological measures taken; and a 5 minute, post-exercise, blood sample drawn from the antecubital vein.

During the tests, subjects breathed through a low resistance respiratory valve and expired gases were collected and analyzed by a pre-calibrated Beckman Metabolic Measurement Cart (this occurred from 2 minutes pre-exercise to 3 minutes post-exercise). Oxygen uptake and heart rate were recorded every 30 seconds. The heart rate was recorded

every 30 seconds using a cardiotachometer simultaneous measurements of heart rate were recorded during rest, in the last 15 seconds of each workload, and after 1 and 3 minutes of recovery using the cardiotachometer and impedance cardiograph. Blood pressure was measured during rest, the last minute of each workload, and at 30 seconds and 2:30 minutes of recovery using a mercury sphygmomanometer. During the final minute of each workload, a rating of perceived exertion (RPE) was obtained using the Borg Scale (Borg, 1970). Blood samples were drawn after 32 minutes rest (immediately prior to exercise) and 5 minutes after cessation of the final workload.

v) Plasma Lactate Concentration

Whole venous blood was drawn from an antecubital vein after 32 minutes rest and at 5 minutes post-exercise (recovery). In each case 0.5 ml of blood was immediately pipetted into a cold vacutainer containing 2.0 ml of chilled perchloric acid. The perchloric and whole blood was mixed with a Sybron Thermolyne Mixer (Model M-16715) and placed on ice for 5 minutes to ensure protein precipitation was complete. The vacutainers were then centrifuged using a Sovall General Lab Centrifuge (Model GLC-4, Dupont) for 15 minutes. The de-proteinized supernatant (clear liquid) was pipetted into a pre-chilled sterile vacutainer which was
stoppered and frozen, at approximately -73 degrees

Celcius, until analysis was performed.

The assay used was based on the principle that lactate dehydrogenase (LDH) catalyzes the reverse reaction between pyruvic acid and nicotinamide adenine dinucleotide reduced (NADH) to produce lactic acid and nicotinamide adenine dinucleotide (NAD). In order to force the reaction to completion, formed pyruvate must be trapped with hydrazine. The increased absorbance due to the formation of NADH provides the measure of the original lactate concentration. The analysis was performed spectrophotometrically at 340 nm according to the Sigma Technical Bulletin No 825-UV (1981). All chemical reagents were obtained from the Sigma Chemical Company.

All the samples were analyzed in triplicate and the mean absorbance reading was used for calculation of lactate concentrations. One third of the lactates were analyzed per reagent mixture. A standard curve was constructed for each reagent mixture based on six samples of known concentration. Additionally, 12 samples were chosen at random and assayed with each normal assay sample. In total 36 samples were analyzed twice with different reagent mixtures (Appendix H) to ensure that the results were consistent across assay runs, reagent mixture and standard curves (r = 0.94, standard error of 5%).

G. STATISTICAL ANALYSIS

Statistical analysis of the effect of cycling velocities and relative power output was determined by a two-way analysis of variance (ANOVA) with repeated measures (Winer, 1971). The relative power output factor consisted of 3 levels: power output equated to 40, 55 and 70% of VO_2max. The factor of cycling velocities was repeated on four measures: 40, 60, 80 and 100 rpm. If a significant F value was attained for a variable at a power output and cycling velocity indicating a significant interaction between the two factors, a Newman-Keuls' multiple means comparison procedure was performed to assess the significance of specific difference between the mean values for each cycling velocity at each PO (Ferguson, 1981; Winer, 1971). Cycling velocity and blood lactate concentrations were analyzed using a one-way analysis of variance with repeated levels and a post hoc Newman-Keuls' procedure because only one level of the power output factor was evaluat[illegible] minutes post-exercise). Linear regressions f[illegible] output on oxygen uptake were performed as rep[illegible] re to include all three power output leve[illegible] are represented as mean plus or minus t[illegible] error of the mean (mean ± SEM). Significance in this thesis is associated with a probability of less than 0.05 ($p<.05$) unless otherwise indicated.

CHAPTER III

RESULTS

A. SUBJECTS

Nineteen male subjects participated in the study. All subjects were physically active males (mean age of 27 ±1 years), ranging in age from 17 to 35. They all were participating in aerobic fitness activities of not less than 3 hours per week.

Subject characteristics are presented in Table 3.1 (detailed subject characteristic profiles are contained in Appendix E).

Table 3.1 - Subject Characteristics
(Mean ± SEM, Range)

CHARACTERISTICS	MEAN	SEM	RANGE
AGE (years)	27	±1	17 - 35
HEIGHT(cm)	180.0	±2.1	165.0 - 196.0
WEIGHT (kg)	76.0	±2.6	54.5 - 96.5
VO_2max (l/min)	3.93	±0.11	3.00 - 4.68
VO_2max (ml/kg/min)	52.1	±1.2	39.7 - 60.7

B. ANALYSIS OF RESULTS

The power outputs required to elicit 40, 55 and 70% of VO_2max for each subject were calculated. The mean power outputs (±SEM) were 111 (±3) watts at 40% of VO_2max, 167 (±5) watts at 55% of VO_2max and 223 (±7) watts at 70% of VO_2max. These power outputs (40, 55 and 70% VO_2max) are referred to as low, medium and high power outputs, respectively.

The variables investigated in this study are oxygen uptake, heart rate, stroke volume, cardiac output, mean arterial pressure, systemic vascular resistance, and rate of perceived exertion. Oxygen uptake, heart rate, stroke volume, systolic and diastolic blood pressures were measured. Cardiac output, mean arterial pressure and systemic vascular resistance were calculated. Cardiac output was calculated as the product of heart rate and stroke volume. Mean arterial pressure was calculated by adding diastolic blood pressure to one third of the difference between systolic and diastolic blood pressures. Systemic vascular resistance was calculated by dividing mean arterial pressure by cardiac output. Individual subject raw data are contained in Appendix F. The means (±SEM) are summarized in Tables 3.2, 3.3 and 3.4 for low, medium and high power outputs, respectively.

In order to test for the significant difference between means for any variable and the four cycling velocities, a repeated measures analysis of variance (ANOVA) was used. For variables observed over 3 power outputs, a two-way ANOVA with repeated measures was conducted. For variables only observed at one level a one-way ANOVA with repeated measures was employed. A Student Newman Keuls' post hoc test was conducted on all significant interactions for any variable at a power output.

TABLE 3.2 - Physiological and RPE Responses at Power Output Equated to 40% VO_2max (Mean ± SEM)

Variables	40 rpm	60 rpm	80 rpm	100 rpm
VO_2 (l/min)	1.55 (±0.04)	1.55 (±.04)	1.63 (±0.04)	1.95 (±0.05)
HR (bpm)	111 (±3)	110 (±3)	115 (±3)	129 (±3)
SV (ml/beat)	116 (±6)	113 (±6)	112 (±6)	116 (±5)
CO (l/min)	11.51 (±0.29)	11.32 (±0.27)	11.63 (±0.31)	13.99 (±0.30)
MAP (mmHg)	96 (±2)	97 (±2)	94 (±2)	102 (±2)
SVR (dyn·s/cm^5)	679 (±25)	697 (±28)	656 (±24)	590 (±17)
RPE (Borg)	8 (±0)	8 (±0)	8 (±0)	9 (±0)

3 Physiological and RPE Responses at Power Output Equated to 55% VO_2max (Mean ± SEM)

Variables	40 rpm	60 rpm	80 rpm	100 rpm
VO_2 (l/min)	2.29 (±0.07)	2.20 (±0.06)	2.26 (±0.07)	2.61 (±0.08)
HR (bpm)	139 (±3)	134 (±3)	140 (±3)	153 (±3)
SV (ml/beat)	117 (±5)	118 (±5)	116 (±5)	115 (±5)
CO (l/min)	15.59 (±0.33)	15.15 (±0.31)	15.47 (±0.36)	17.14 (±0.38)
MAP (mmHg)	106 (±2)	102 (±2)	102 (±2)	103 (±2)
SVR (dyn·s/cm^5)	548 (±19)	540 (±16)	533 (±17)	487 (±13)
RPE (Borg)	12 (±0)	11 (±0)	11 (±0)	11 (±0)

TABLE 3.4 - Physiological and RPE Responses at Power Output Equated to 70% VO_2max (Mean ± SEM)

Variables	40 rpm	60 rpm	80 rpm	100 rpm
VO_2 (l/min)	3.10 (±0.08)	2.91 (±0.08)	2.95 (±0.08)	3.24 (±0.08)
HR (bpm	168 (±3)	161 (±3)	167 (±3)	173 (±3)
SV (ml/beat)	115 (±4)	114 (±4)	111 (±3)	114 (±4)
CO (l/min)	18.82 (±0.42)	18.01 (±0.44)	18.17 (±0.35)	19.69 (±0.46)
MAP (mmHg)	111 (±2)	110 (±2)	108 (±2)	107 (±2)
SVR dyn·s/cm^5)	476 (±13)	486 (±16)	474 (±14)	448 (±10)
RPE (Borg)	15 (±1)	13 (±0)	13 (±0)	14 (±1)

i) Oxygen Uptake and Cycling Velocity

Variations in oxygen uptake with cycling velocities were evident for the full range of power outputs. At all power outputs (low, medium and high), oxygen uptake for 60 rpm was lowest and was the highest at 100 rpm . Oxygen uptake at 60 and 80 rpm was not significantly different at any power output. For low and medium power outputs, oxygen uptake at 40 rpm was similar to 60 and 80 rpm; however, at high power outputs, oxygen uptake for 40 rpm was significantly higher than for 60 and 80 rpm, but significantly lower than oxygen uptake for 100 rpm. This is graphically demonstrated in Figure 3.1 and summarized in Table 3.5.

ii) Heart Rate and Cycling Velocity

Heart rates were significantly higher ($p<.05$) for 100 rpm than for any other cycling velocity over all power outputs. There was no significant difference between the heart rates elicited for 40, 60 and 80 rpm at all power outputs, although the heart rates produced at 60 rpm were the lowest over the range of power outputs. The means and SEM are displayed in Figure 3.2 and summarized in Table 3.6.

Figure 3.1 Oxygen Uptake Versus Cycling Velocity
(Missing Error Bars indicate SEM was too Small to Show)

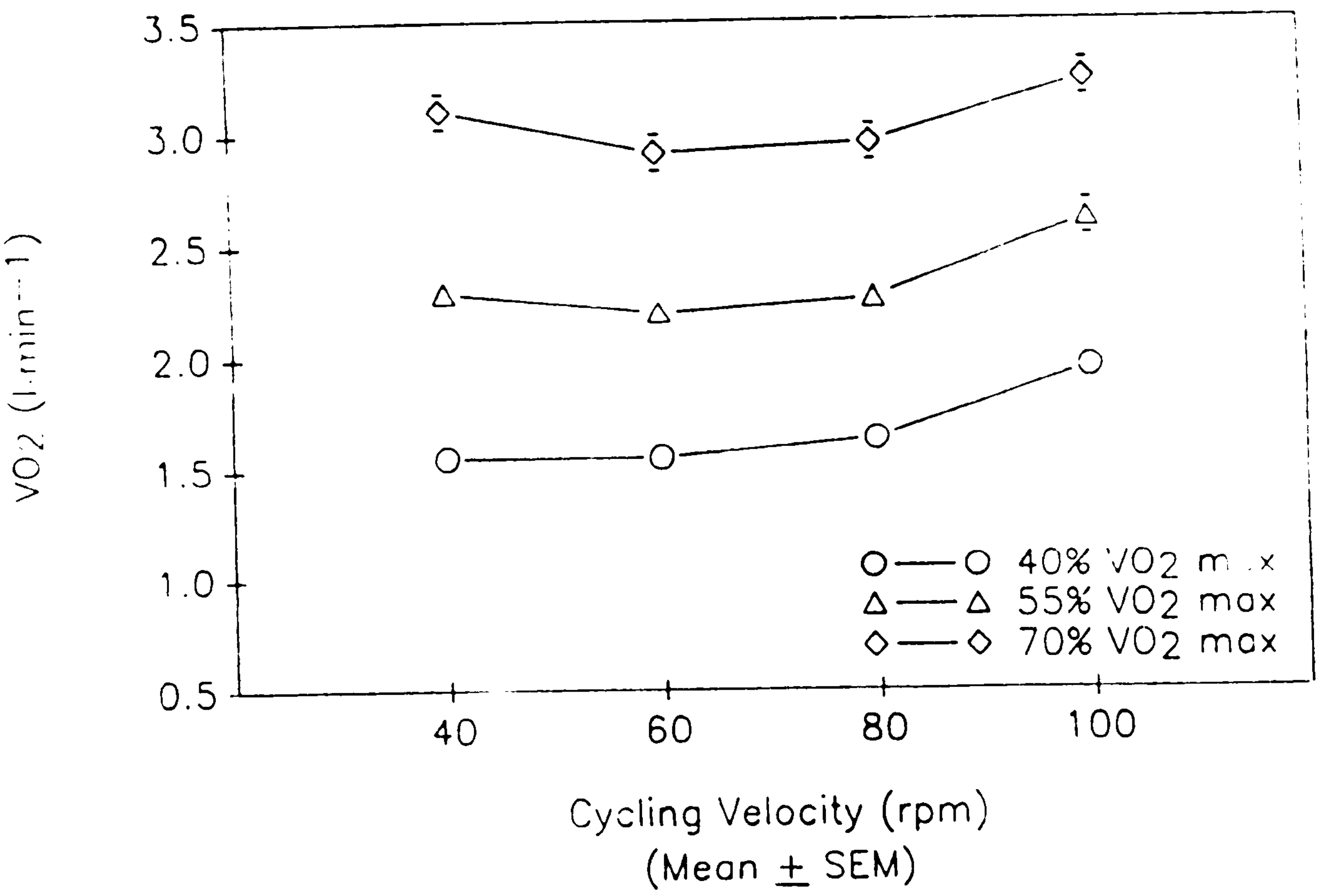


TABLE 3.5 - Significance and Means (±SEM) of Oxygen Uptake (l/min) and Cycling Velocities at Low, Medium and High Power Outputs

Power Output 40% (Low)	40 rpm	60 rpm	80 rpm	100 rpm
Mean (±SEM)	1.55 (±0.04)	1.55 (±0.04)	1.63 (±0.04)	1.95 (±0.05)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD **	
Power Output 55% (Medium)				
Mean (±SEM)	2.29 (±0.07)	2.20 (±0.06)	2.26 (±0.07)	2.61 (±0.08)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD **	
Power Output 70% (High)				
Mean (±SEM)	3.10 (±0.08)	2.91 (±0.08)	2.95 (±0.08)	3.24 (±0.08)
60 rpm	SD **			
80 rpm	SD *	NS		
100 rpm	SD **	SD **	SD **	

NS - NOT SIGNIFICANT AT 0.05 LEVEL
SD - SIGNIFICANT DIFFERENCE (** $P<0.01$; * $P<0.05$)

Figure 3.2 – Heart Rate Versus Cycling Velocity
(Missing Error Bars indicate SEM was too Small to Show)

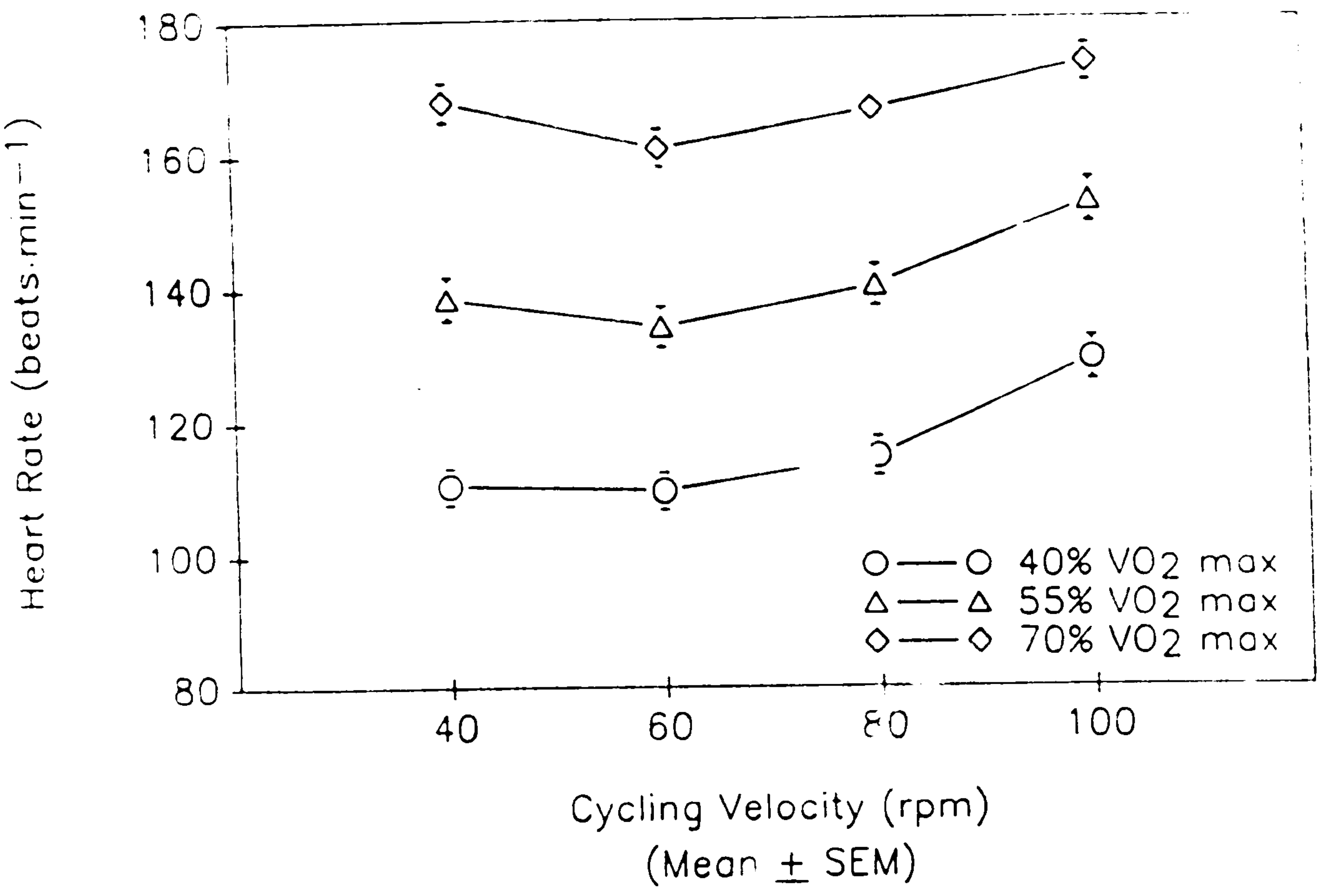


TABLE 3.6 - Significance and Means (±SEM) of Heart Rate (beat/min) and Cycling Velocities at Low, Medium and High Power Outputs

Power Output 40% (Low)	40 rpm	60 rpm	80 rpm	100 rpm
Mean (±SEM)	111 (±3)	110 (±3)	115 (±3)	129 (±3)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD **	
Power Output 55% (Medium)				
Mean (±SEM)	139 (±3)	134 (±3)	140 (±3)	153 (±3)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD **	
Power Output 70% (High)				
Mean (±SEM)	168 (±3)	161 (±3)	167 (±2)	173 (±3)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD *	SD **	SD *	

NS - NOT SIGNIFICANT AT 0.05 LEVEL

SD - SIGNIFICANT DIFFERENCE (** $P<0.01$; * $P<0.05$)

iii) Stroke Volume and Cycling Velocity

The stroke volumes elicited were not significantly different between cycling velocities at any power output. Nor were there any noticeable trends. Although the individual stroke volumes for medium power outputs were slightly higher than for low power outputs, the differences were only 2 ml/beat (not significant). A slight drop was evident in all stroke volumes at high power outputs in comparison to those elicted at medium power outputs with a difference of 3 ml/beat (not significant). These data of Table 3.7 are illustrated graphically in Figure 3.3.

iv) Cardiac Output and Cycling Velocity

At low, medium and high power outputs, 100 rpm resulted in higher cardiac outputs than any other cycling velocity (significantly different at .01 level in all cases). There was no significant difference between 40, 60 & 80 rpm at any power output. Although no significant difference existed, 60 rpm elicited the lowest cardiac output results at all power outputs, while 40 rpm required higher cardiac outputs than 60 & 80 rpm at medium and high power outputs. Means and

Figure 3.3 – Stroke Volume Versus Cycling Velocity
(Missing Error Bars indicate SEM was too Small to Show)

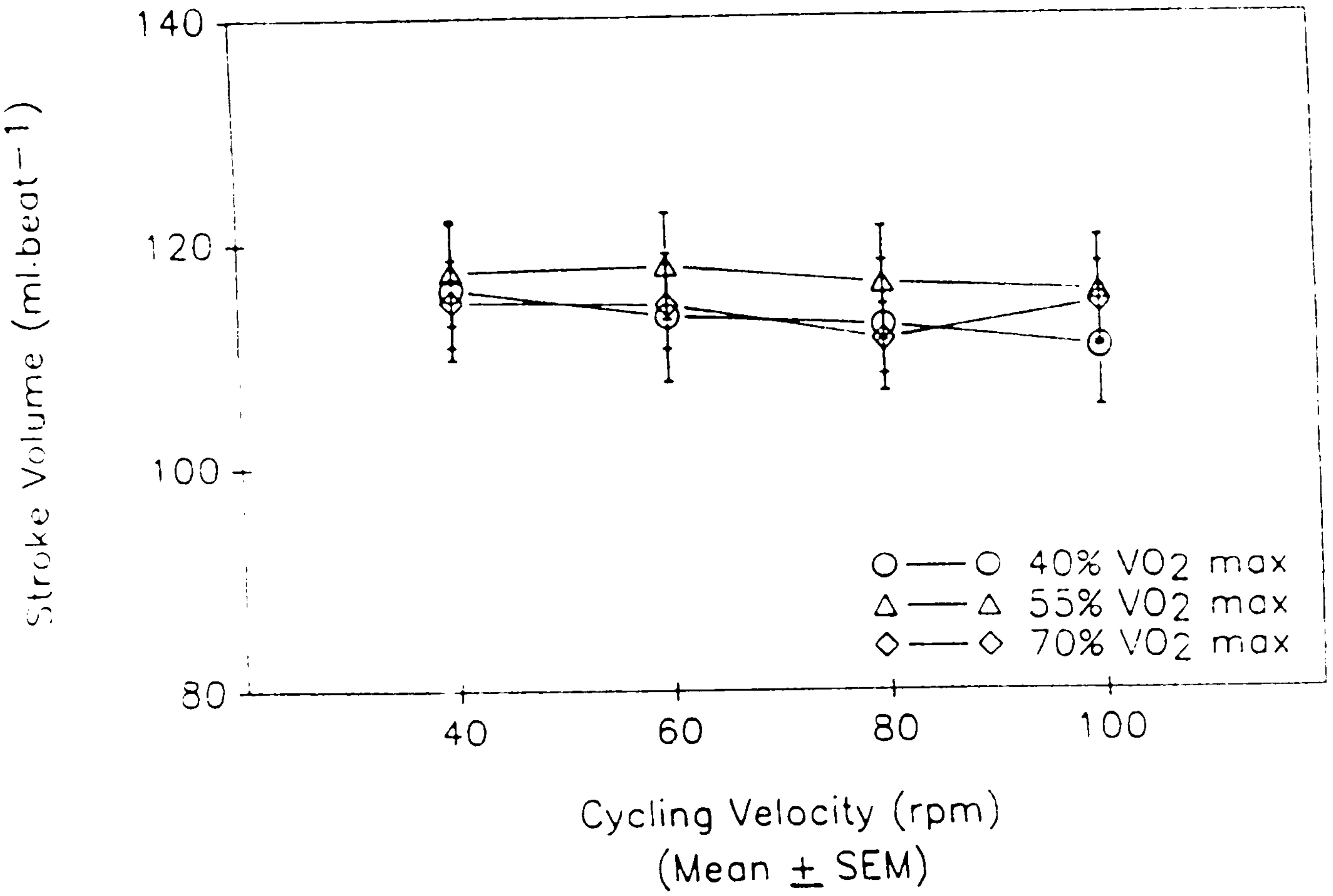


TABLE 3.7 - Significance and Means (±SEM) of Stroke Volume (ml/beat) and Cycling Velocities at Low, Medium and High Power Outputs

Power Output 40% (Low)	40 rpm	60 rpm	80 rpm	100 rpm
Mean (±SEM)	116 (±6)	113 (±6)	112 (±6)	116 (±5)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	NS	NS	NS	
Power Output 55% (Medium)				
Mean (±SEM)	117 (±5)	118 (±5)	116 (±5)	116 (±5)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	NS	NS	NS	
Power Output 70% (High)				
Mean (±SEM)	115 (±4)	114 (±4)	111 (±3)	114 (±4)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	NS	NS	NS	

NS - NOT SIGNIFICANT AT 0.05 LEVEL
SD - SIGNIFICANT DIFFERENCE (** $P<0.01$; * $P<0.05$)

significance differences are reported in Table 3.8 and illustrated in Figure 3.4.

v) Mean Arterial Pressure and Cycling Velocity

The only significant difference observed in mean arterial pressure was at low power output, where 100 rpm produced significantly higher pressures than other cycling velocities.

The only trend evident was that the mean arterial pressures at 40 rpm at medium and high power outputs were higher, although not significantly, than for any other cycling velocity. Mean arterial pressure increased with an increase in power output for all cycling velocities. Table 3.9 contains a summary of mean arterial pressure results and Figure 3.5 provides the means and SEM for these data.

vi) Systemic Vascular Resistance and Cycling Velocity

In all cases, systemic vascular resistance was lowest for 100 rpm; however, this was only significant for low and medium power outputs. At the low power outputs systemic vascular resistance was significantly lower for 80 rpm than for 60 rpm. At all power outputs, the trend was that 80 and 100 rpm produced

Figure 3.4 – Cardiac Output Versus Cycling Velocity
(Missing Error Bars indicate SEM was too Small to Show)

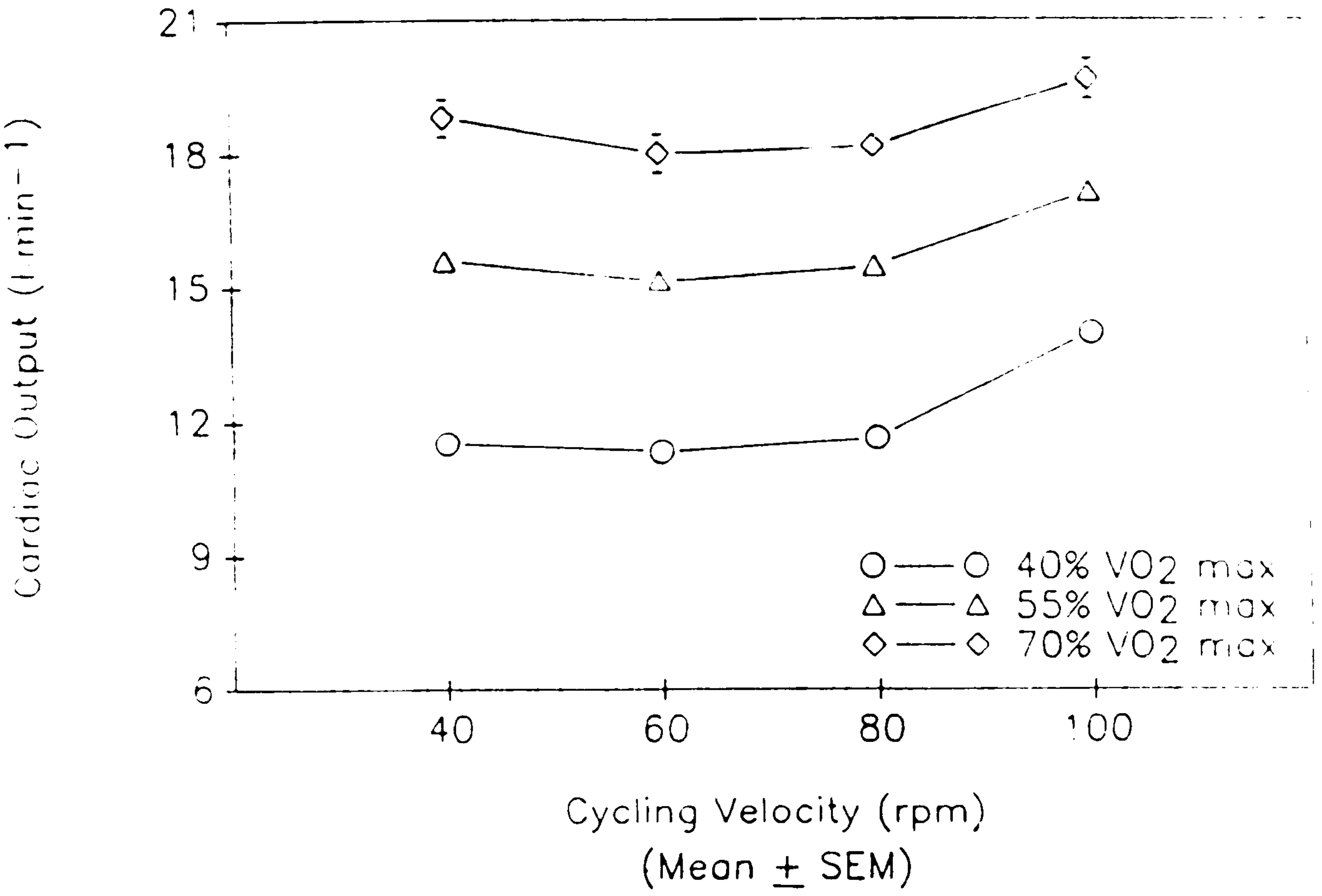


TABLE 8 - Significance and Means (±SEM) of Cardiac Output (l/min) and Cycling Velocities at Low, Medium and High Power Outputs

Power Output 40% (Low)	40 rpm	60 rpm	80 rpm	100 rpm
Mean (±SEM)	11.51 (±0.29)	11.32 (±0.27)	11.63 (±0.31)	13.99 (±0.30)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD **	
Power Output 55% (Medium)				
Mean (±SEM)	15.59 (±0.33)	15.15 (±0.31)	15.47 (±0.36)	17.14 (±0.38)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD **	
Power Output 70% (High)				
Mean ±SEM	18.82 (±0.42)	18.01 (±0.44)	18.17 (±0.35)	19.69 (±0.46)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD **	

NS - NOT SIGNIFICANT AT 0.05 LEVEL

SD - SIGNIFICANT DIFFERENCE (** $P<0.01$; * $P<0.05$)

Figure 3.5 - Mean Arterial Pressure Versus Cycling Velocity

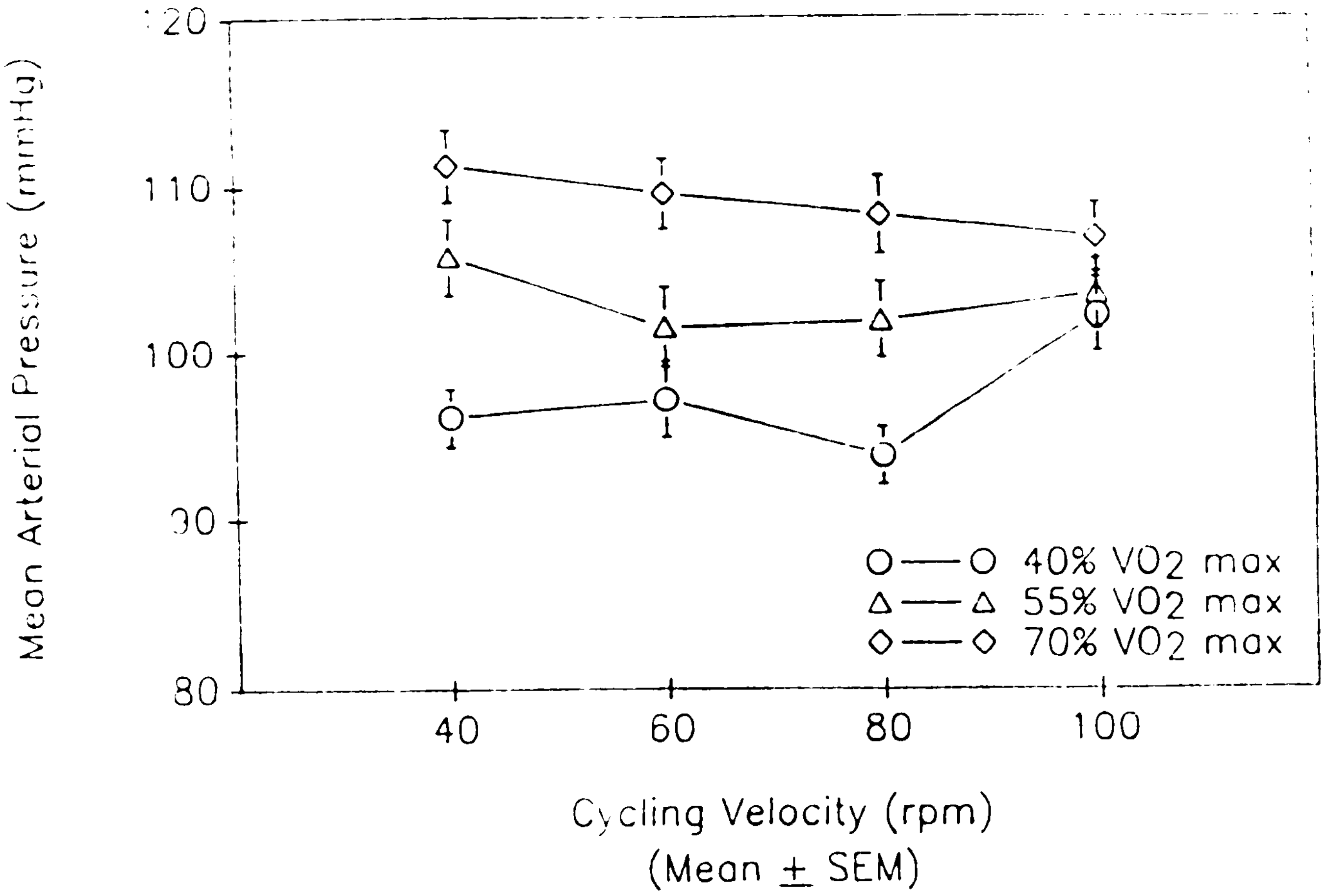


TABLE 3.9 - Significance and Means (±SEM) of Mean Arterial Pressures (mmHg) and Cycling Velocities at Low, Medium and High Power Outputs

Power Output 40% (Low)	40 rpm	60 rpm	80 rpm	100 rpm
Mean (±SEM)	96 (±2)	97 (±2)	94 (±2)	102 (±2)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD **	
Power Output 55% (Medium)				
Mean (±SEM)	106 (±2)	102 (±2)	102 (±2)	103 (±2)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	NS	NS	NS	
Power Output 70% (High)				
Mean (±SEM)	111 (±2)	110 (±2)	108 (±2)	107 (±2)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	NS	NS	NS	

NS - NOT SIGNIFICANT AT 0.05 LEVEL
SD - SIGNIFICANT DIFFERENCE (** P<0.01; * 0.05)

lower (not significantly different) systemic vascular resistances than for 40 or 60 rpm. The summary of these results are contained in Table 3.10 and demonstrated graphically in Figure 3.6.

vii) Rate of Perceived Exertion and Cycling Velocity

The rate of perceived exertion for cycling velocities was only found to be significantly different at high power outputs. 40 rpm was perceived the most difficult and significantly higher than 60 or 80 rpm (p<.01), and 100 rpm was perceived more difficult than 60 or 80 (p<.05) but not different from 40 rp. The trends that existed (not significantly diffe) were: at low power outputs perception of diffi increased with cycling velocity, and at medium and high power outputs 80 rpm was perceived the least difficult. Table 3.11 contains the RPE results and Figure 3.7 graphically illustrates these data.

viii) Blood Lactate and Cycling Velocity

Venous lactate concentrations were only analyzed during pre-exercise and post-exercise. No significant differences existed during pre-exercise lactate results. The 5 minutes post-exercise lactates

Figure 3.6 – Systemic Vascular Resistance Versus Cycling Velocity
(Missing Error Bars indicate SEM was too Small to Show)

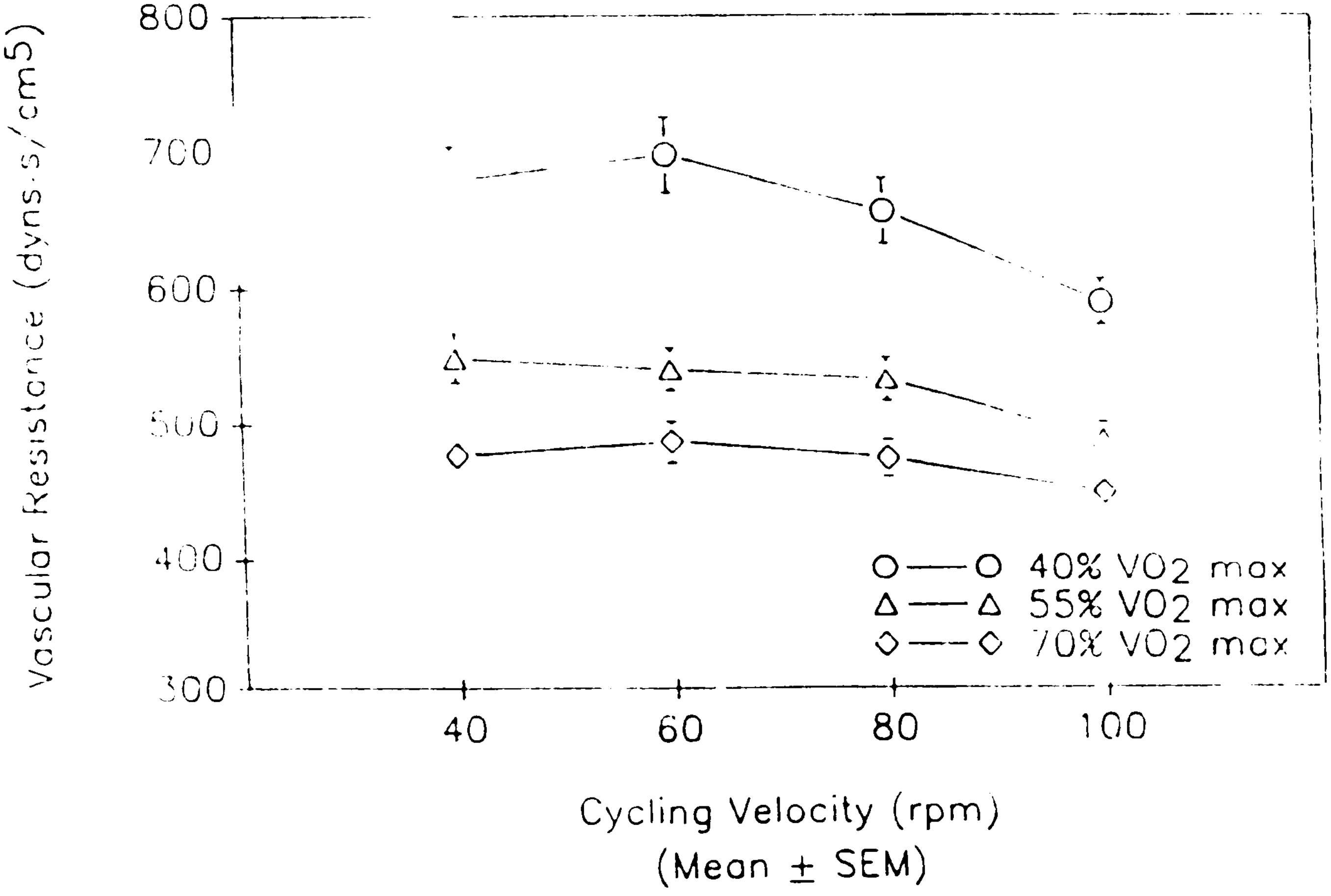


TABLE 3.10 - Significance and Means (±SEM) of Systemic Vascular Resistance (dyn·s/cm^5) and Cycling Velocities at Low, Medium and High Power Outputs

Power Output 40% (Low)	40 rpm	60 rpm	80 rpm	100 rpm
Mean ±SEM	679 (±25)	697 (±28)	656 (±24)	590 (±17)
60 rpm	NS			
80 rpm	NS	SD *		
100 rpm	SD **	SD **	SD **	
Power Output 55% (Medium)				
Mean (±SEM)	548 (±19)	540 (±16)	533 (±17)	487 (±13)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	SD **	SD **	SD *	
Power Output 70% (High)				
Mean (±SEM)	476 (±13)	486 (±16)	474 (±14)	448 (±19)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	NS	NS	NS	

NS - NOT SIGNIFICANT AT 0.05 LEVEL
SD - SIGNIFICANT DIFFERENCE (** p<0.01; * p<0.05)

Figure 3.7 – Rating of Perceived Exertion Versus Cycling Velocity
(Missing Error Bars indicate SEM was too Small to Show)

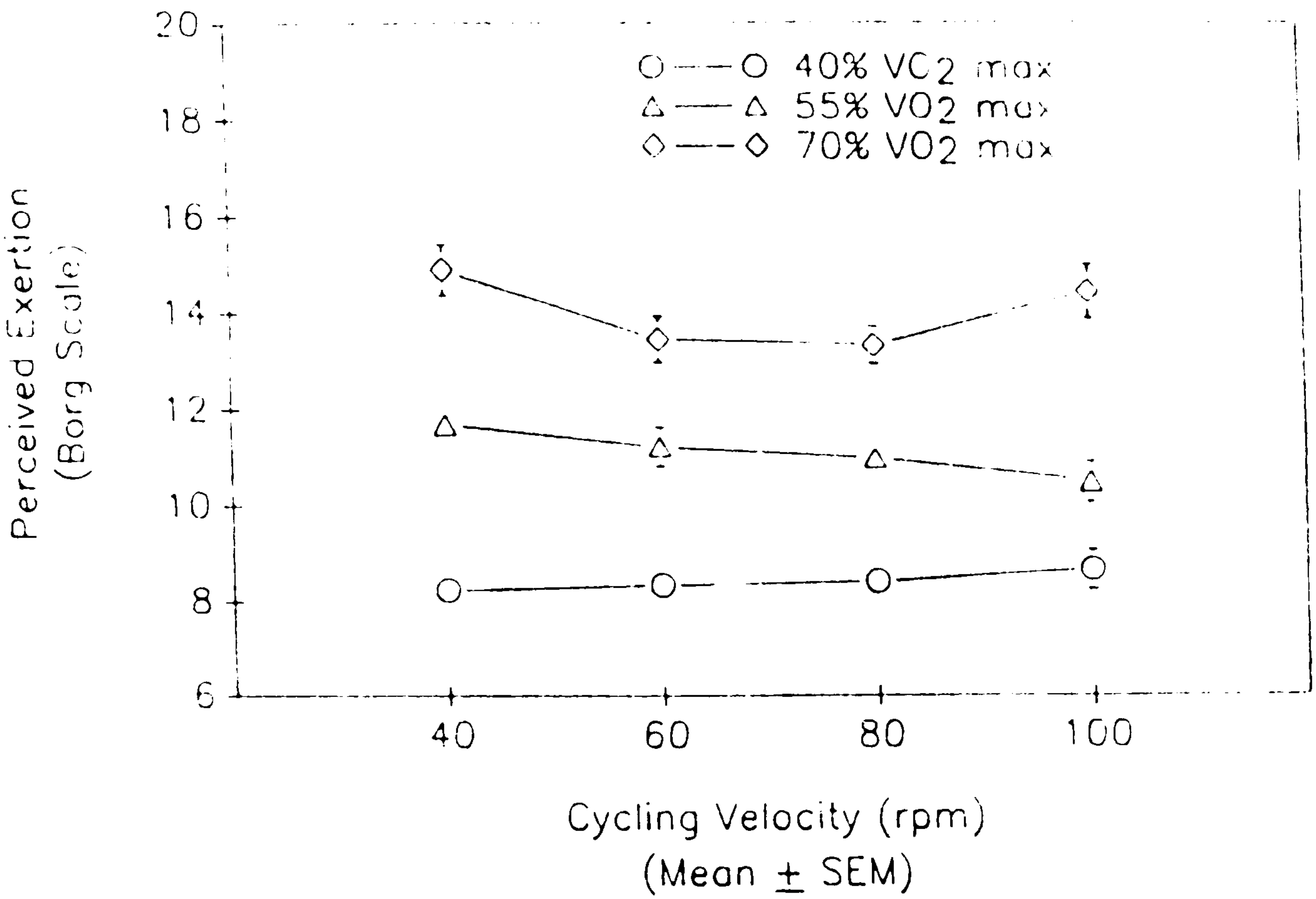


TABLE 3.11 - Significance and Means (±SEM) of Rate of Perceived Exertion (Borg Scale) and Cycling Velocities at Low, Medium and High Power Outputs

Power Output 40% (Low)	40 rpm	60 rpm	80 rpm	100 rpm
Mean (±SEM)	8 (±0)	8 (±0)	8 (±0)	9 (±0)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	NS	NS	NS	
Power Output 55% (Medium)				
Mean (±SEM)	12 (±0)	11 (±0)	(±0)	11 (±0)
60 rpm	NS			
80 rpm	NS	NS		
100 rpm	NS	NS	NS	
Power Output 70% (High)				
Mean (±SEM)	15 (±1)	13 (±0)	13 (±0)	14 (±1)
60 rpm	SD **			
80 rpm	SD **	NS		
100 rpm	NS	SD *	SD *	

NS - NOT SIGNIFICANT AT 0.05 LEVEL
SD - SIGNIFICANT DIFFERENCE (** $p<0.01$; * $p<0.05$)

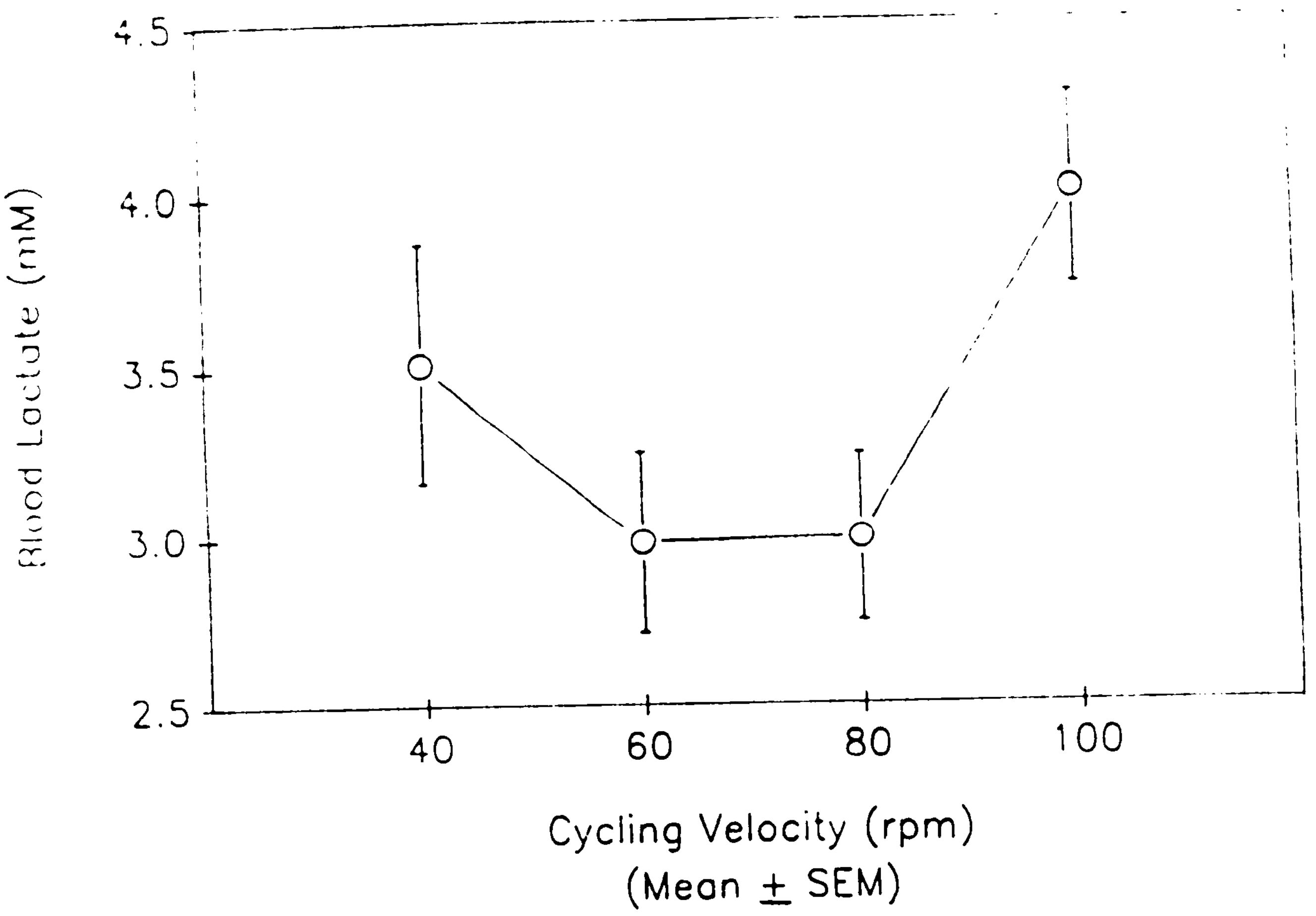
were analyzed to indicate the accumulative response over three workloads to each cycling velocity (that is after cycling 4 minutes at Low, 5 minutes at medium and 5 minutes at high power outputs for each cycling velocity). Lactate accumulation in the blood was significantly higher for 40 and 100 rpm (p<0.01) While no significance difference existed in blood lactate levels for cycling rates of 60 and 80 rpm; 100 rpm levels were elevated over those for 40, 60 and 80 rpm (p<0.01). The means (±SEM) and location of significant differences are provided at Table 3.12 and hown in Figure 3.8. No significant differences existed between pre-exercise blood lactate concentrations.

TABLE 3.12 - Significance and means (±SEM) of blood lactates (mM) and cycling velocities 5 minutes post-exercise

Power Output 5 min post Exercise	40 rpm	60 rpm	80 rpm	100 rpm
Mean (±SEM)	3.51 (±0.35)	2.98 (±0.27)	2.99 (±0.25)	4.01 (±0.28)
60 rpm	SD **			
80 rpm	SD **	NS		
100 rpm	SD **	SD **	SD **	

NS - NOT SIGNIFICANT AT 0.05 LEVEL
SD - SIGNIFICANT DIFFERENCE (** P<0.01; * P<0.05)

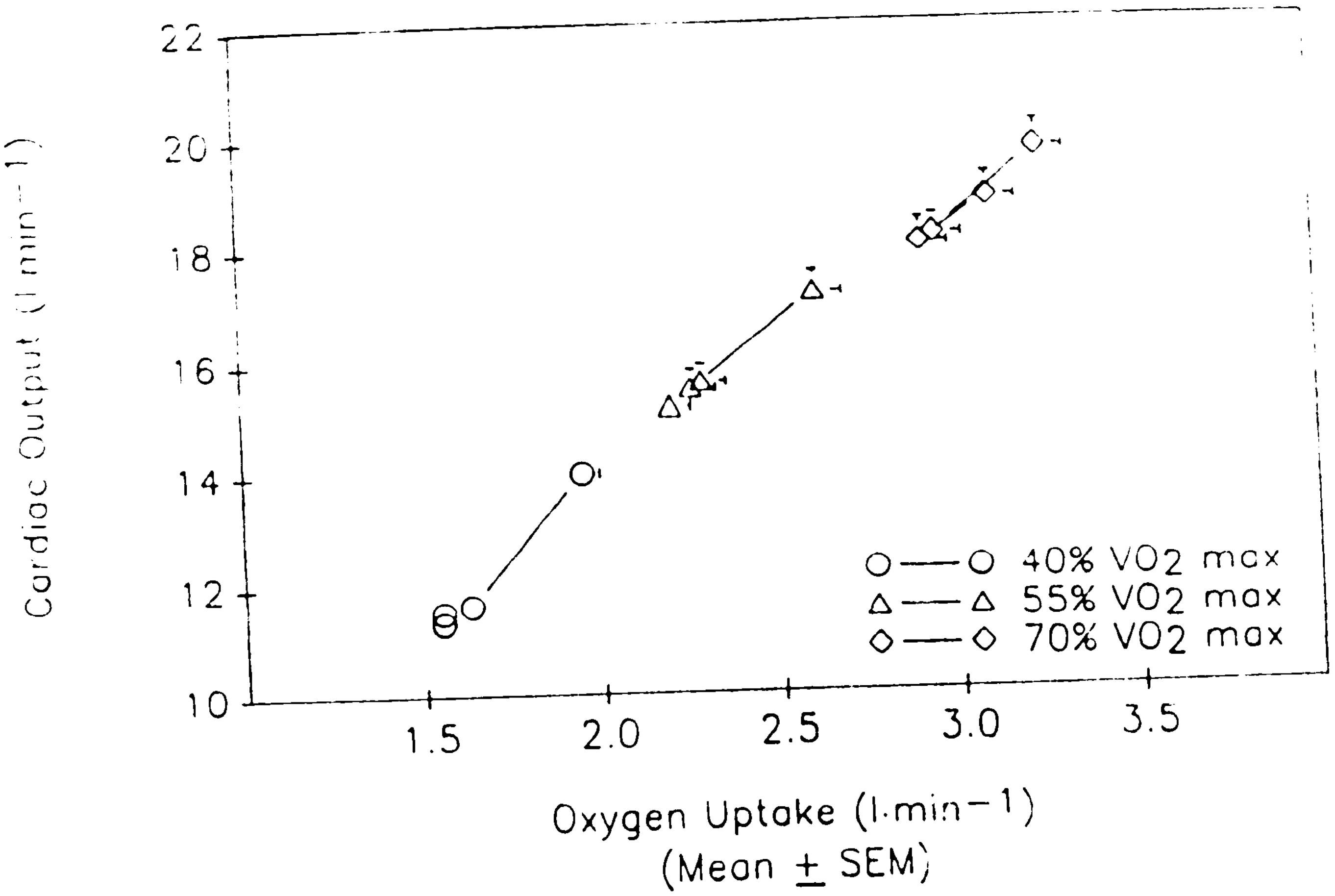
Figure 3.8 – Blood Lactate Versus Cycling Velocity
(5 minutes Post Exercise)



ix) Cardiac Output and Oxygen Consumption

The graph in Figure 3.9 compares oxygen consumption to cardiac output at every workload for each cycling velocity. The slope of the line of oxygen uptake to cardiac output is least at 100 rpm and greater at 40 rpm and greatest at 60 and 80 rpm. No significant differences existed between the regression lines.

Figure 3.9 – Cardiac Output Versus Oxygen Uptake
(Missing Error Bars indicate SEM was too Small to Show)



CHAPTER IV

DISCUSSION

There have been numerous studies investigating cycling velocities and various parameters such as heart rate, oxygen uptake, ventilation rate, blood lactate, perceived exertion, glycogen depletion and mechanical efficiency. Many studies (Bannister and Jackson, 1967; Buchanan and Weltman, 1985; Gaesser and Brooks, 1973; Hess and Seusing, 1962) have used small sample sizes. Others have studied only top caliber athletes (Bannister and Jackson, 1967; Buchanan and Weltman, 1985; Hagberg et al., 1981). Some investigators have studied a similar sample to that of the present study (healthy, active, young males) and found contradictory results. Gueli and Shephard (1976) found no significant difference in heart rate or oxygen uptake for cycling velocities between 60 and 85 rpm at a power output equated to 60% of VO_2max. Michielli and Stricevic (1977), on the other hand, found 80 rpm produced higher heart rates than 60 rpm (significant at 0.01 level).

Consequently, this study was undertaken to investigate cardiovascular and perceived exertion responses to 4 cycling velocities over a range of power outputs. The design permitted the evaluation of the separate effects at low, medium and high power outputs for cycling velocities of 40, 60, 80 and 100 rpm.

The original hypothesis was that cardiovascular function at submaximal loads would not be altered by varying the cycling velocity. It was found that not all velocities produced equivalent cardiovascular responses but rather that certain velocities produced results that were not significantly different (ie equivalent responses) and placed lower demands on the cardiovascular system (optimal cycling velocities). Both 60 and 80 rpm were not significantly different in any physiological or perceived exertion response (except for systemic vascular resistance at low power output) and placed lower demands on the cardiovascular system than any other investigated cycling velocity. No other two cycling velocities demonstrated this equivalency.

A. OXYGEN UPTAKE

Cycling velocities of 60 and 80 rpm at all power outputs were most efficient, that is, lower oxygen uptake values (significant at the 0.05 level) Oxygen uptake for low and medium power outputs were equivalent (no significant difference) at 40, 60 and

80 rpm, while 100 rpm required higher oxygen uptake ($p<0.01$) at all power outputs. Cycling velocities of 60 and 80 rpm resulted in lower oxygen uptake values at high power outputs. Other researchers support these findings; Eckermann and Millahn, 1967; Lollgen et al., 1980; Pandolf and Noble, 1973, all found that cycling velocities of 40 to 80 rpm at low and medium power outputs are optimal (that is, require lower oxygen uptake). When high power outputs are included the optimal cycling velocities are reduced to 60 to 80 rpm (Boning et al., 1984; Buchanan and Weltman, 1985; Coast et al., 1986).

Increases in oxygen uptake at high cycling velocities for all power outputs and at low velocities at high power outputs appeared to be caused by different mechanisms. A linear increase in oxygen uptake occurs with an increase in work intensity (Mitchell, 1985). Therefore, the difference in oxygen uptake over cycling velocities at equivalent power output must be a result of inequalities in external work (such as differences in fiber recruitment, body stabilizer utilization, muscle mass involvement and effective force).

The increased oxygen uptake at 100 rpm for all power outputs appeared to be a result of increased use of body stabilizing muscles of the trunk and upper body. Suzuki (1979) found that a predominance of fast twitch (FT) fibers are employed at high cycling rates. He suggests that slow twitch (ST) fibers were unable to cross-bridges (between actin and myosin) at faster cycling rates, and the result was higher oxygen uptake. Tankano (1987) and Hagberg et al. (1981) agreed that the static component (the increased use of body stabilizing muscles) required to cycle at higher cycling velocities required increased oxygen uptake.

At 40 rpm and high power output the higher oxygen uptake was possibly caused by increased upper body and trunk muscle involvement needed to maintain the cycling velocity and increased anaerobic metabolism resulting from a higher force for each pedal stroke (this is supported by increased blood lactate concentrations at this velocity). Suzuki (1979) found decreased efficiency of slow twitch fibers at high power output and increased anaerobic involvement. Kaneko et al. (1979) determined that low cycling rates at high power output resulted in increased external work.

B. HEART RATE

Heart rates were higher ($p<.01$) at 100 rpm for all power outputs. In terms of heart rate, cycling velocities of 40 to 80 rpm produced similarly lower heart rates at all power outputs. These heart rate results are well supported (Boning et al., 1984; Lollgen et al. 1980; Pandolf and Noble, 1973). This suggests that cycling velocities of 40 to 80 rpm are optimal in terms of heart rate.

The increase in heart rate associated with high cycling rates at all power outputs appeared to be the result of increased intensity of upper body and trunk stabilizing muscles (isometric contraction) and possibly the use of the less efficient fast twitch fibers (Suzuki, 1979; and Hagberg et al.,1981). Coast et al. (1986) found no difference in circulating catecholamines or oxygen uptake although heart rates were increased at higher cycling rates which suggests that the difference may be due to increased venous return from the maximized pumping action of the legs. Since the heart pumps all blood returned to it an increase in venous return results in an increased cardiac output. Since stroke volume has reached a maximum or peak plateau, the increase in cardiac output has to come from increased heart rate (no significant difference in stroke volumes over all power outputs). It appears at 100 rpm that the circulatory system is driven by venous return which results in creased cardiac output (since the healthy heart distributes all the blood returned to it) and thus heart rate is increased. This suggests that there is less oxygen extracted during each circulation of the blood.

C. CARDIAC OUTPUT

Stroke volumes were not significantly different between cycling velocities over the full range of power outputs. Stroke volume increased to peak values prior to 40% VO_2max and did not change with increased power outputs. Astrand and Rodahl (1986) suggest this occurs at or near 40% of VO_2max when heart rate has reached 110 to 120 beats per minute. Cardiac output, on the other hand, increased with power outputs and varied with cycling velocities. The cardiac outputs required to meet the demands of 100 rpm at all power outputs were higher (p<. Consequently, the optimal cycling velocities in terms of minimizing cardiac output were 40 to 80 rpm for all power outputs. Although not significantly different, cardiac outputs at high power outputs for 40 rpm were elevated by more than 60 ml/minute in comparison to 60 and 80 rpm.

As suggested in heart rate response, the faster pumping action of the legs increased venous return which in term increased cardiac output. Added to this is the increased oxygen uptake required to supply the stabilizing muscle which also increased cardiac output.

D. BLOOD PRESSURE

Systolic blood pressure increased as power outputs increased. At low power outputs systolic blood pressure was much higher for 100 rpm ($p<.01$) than for other cycling velocities. For low and medium power outputs, the lowest pressures (not significantly different) were produced at 40 rpm.

No trend and major differences were observed in diatolic blood pressures over the cycling velocities and power outputs.

Significantly higher ($p<.01$) mean arterial pressures occurred at low power outputs by 100 rpm. No other significant differences existed in mean arterial pressures, but 40 rpm at medium and high power outputs caused the highest pressures. The fact that both mean arterial and systolic blood pressures were elevated at low power output for 100 rpm, but not at medium and high power outputs, suggest that the reflex neural system was not active. And that the circulatory system is being driven primarily by venous return and central command (Mitchell, 1985). It appears that blood pressures are elevated for 100 rpm at medium and high power outputs as a possible result of increased vasodilation (likely due to increased involvement of the upper body and trunk muscles). Astrand (1960) found cardiac output related to external workload but exercise vasodilation dependent on the muscle mass involved (amount and intensity).

Major differences in systemic vascular resistances occurred at low and medium power outputs. The lowest systemic vascular resistances were caused at 100 rpm (p<.01). Systemic vascular resistances at 80 rpm were elevated (p<.05) over all others. No differences in systemic vascular resistance occurred at high power output; however, the trend over all power outputs was that systemic vascular resistances for 100 rpm were lower.

In exercise that employs a large muscle mass, more extensive vasodilation tends to reduce systemic vascular resistance while large increases in cardiac output tend to increase systemic vascular resistance (Lewis et al., 1983). It would appear that larger increases in systemic vascular resistance should occur at 100 rpm at all power outputs and at 40 rpm at high power outputs because oxygen uptake was increased. This is not the case, in fact, at these cycling velocities while oxygen uptake increased significantly and cardiac outputs were elevated, systemic vascular resistance was reduced. This would suggest that in part, a larger muscle mass is being employed. Observation of the subjects tends to support this. At 100 rpm, the subjects moved more and used more stabilizing muscles. At 40 rpm and high power outputs, the subjects appeared to use more upper body and trunk movement. Consequently, reduced systemic vascular resistance at 40 rpm supports the proposition that extensive vasodilation occurred and could be a result of increased muscle mas involvement.

E. BLOOD LACTATES

Blood lactate concentrations were obtained 5 minutes after the subjects had performed at high power outputs (that is after 4 minutes at low, 5 minutes at medium, 5 minutes at high power outputs and 5 minutes of recovery). Blood lactate levels were elevated ($p<.01$) for 40 and 100 rpm over those for 60 and 80 rpm. Lactate levels for 100 rpm were elevated over those at 40 rpm ($p<.01$). The lactate results were identical to the oxygen uptake results at high power outputs. These results are similar to those of Hagberg et al. (1981), in that the cycling velocities that are most economical (lowest oxygen uptake) produced the lowest blood lactate levels. Buchanan and Weltman (1985) using 60, 90 and 120 rpm found significantly lower ($p < .05$) blood lactate concentrations at 60 and 90 rpm in comparison to 120 rpm.

The elevation of blood lactates at high power output for both fast and slow velocites is more complex. At high power output and low cycling velocities the increase appears to be a result of the high anaerobic component (Hagberg et al., 1981) and due to restricted blood flow caused by the high force required for each pedal stroke (Suzuki (1979). At high velocities the increased use of body stabilizing muscles appears to be sufficient to increase blood lactate levels which supports increased anaerobic involvement. It appears that 60 and 80 rpm are more economical cycling velocities (lower cardiovascular demand) and energy requirements can be met by the aerobic metabolism possibly because of reduced blood occlusion, hence increased oxygen exchange.

F. RATE OF PERCEIVED EXERTION

Cycling velocities were not perceived different at low and medium power output. These finding are not consistent with physiological responses. In addition, there were no similarities in the trends. At high power outputs, 60 and 80 rpm were perceived the easiest ($p<0.05$) and 40 and 100 rpm were perceived the most difficult. At high power outputs, perceived exertion varied with two physiological responses to cycling velocities (blood lactate and oxygen uptake). This would indicate that for physically active athletic males, perceived exertion responses at high power outputs are more indicative of blood lactate levels and oxygen uptake than any other measured physiological variable.

G. GENERAL DISCUSSION

Although there is much disagreement as to which cycling velocities place minimal demands on the cardiovascular system (optimal velocity), many authors suggest that an optimal range exists. This study clearly demonstrated that 60 to 80 rpm are within the optimal range and that 40 and 100 rpm are outside the optimal range. While there is support for the present findings (Boning et al., 1984; Buchanan and Weltman, 1985; Coast et al., 1986; Gueli and Shephard, 1976), other cycling velocities have received support as being the least demanding on the cardiovascular system. Some investigators have found 40 to 80 rpm to be most efficient cycling velocities, that is reduced oxygen uptake (Eckermann and Millahn, 1966; Lollgen et al., 1980; Pandolf and Noble, 1973). Hagberg et al. (1981) found higher velocities (72-100 rpm) more efficient as did Moffatt and Stamford (1978; 80-97 rpm). Still other investigators have found lower velocities to be more efficient (Hess and Seusing, 1962: 40 to 60 rpm and Michielli and Stricevic, 1977: 40 to 60 rpm). Closer scrutiny is required of those studies which used a wider range of cycling velocities. Eckermann and Millahn (1967) used power output of low and medium range and found results that are consistent with the present findings, that is at low and medium power outputs no signficant differences existed between physiological responses to cycling velocities of 40 t 80 rpm. Pandolf and Noble (1973), using extremely fit subjects and a small sample, found no significant difference between oxygen uptake between 40, 60 70 and 80 rpm; however, at high power output 40 rpm resulted in increased oxygen uptake (the difference was not significant but may have been if a large sample had been investigated).

Therefore, the findings that at low and medium power outputs 40 to 80 rpm were similar and the least stressful in terms of physiological responses is well supported (Boning et al., 1984; Buchanan and Weltman, 1985; Eckermann and Millahn, 1967; Gueli and Shephard, 1976). The support for 60 to 80 rpm producing similar physiological responses, is also quite strong (Boning et al., 1984; Bannister and Jackson, 1976; Buchanan and Weltman, 1985; Gueli and Shephard, 1976).

The value of the present study is the observation of more cardiovascular variables. The addition of cardiac output, mean arterial pressure and systemic vascular resistance measures to the complex interaction, provided additional insight. While oxygen uptake and blood lactate at 40 rpm and higher power outputs were significantly higher than 60 or 80 rpm, no other physiological response demonstrated this. In fact, for cardiac output, stroke volume, heart rate and mean arterial pressure, no significant differences existed between cycling velocities of 40 to 80 rpm. This is not consistent with the suggestion by Lewis et al. (1983) that a 1:1 relationship exists between systemic oxygen transport and utilization. These findings would suggest that the rate of muscle contraction (due to cycling velocity) might alter this proposed relationship. Coast et al. (1986) ruled out increasing resistance per pedal revolution, or the difference in fiber type as probable causes of increased blood lactate concentrations and oxygen uptake at lower rates. They suggest the difference is due to increased metabolic costs of moving the muscles at greater or lesser than optimal speed. The increased metabolic cost in turn resulted in increased oxygen uptake and blood lactate levels at a given power output. Hagberg et al. (1981) suggest the possible cause of increased in oxygen uptake at cycling velocities which are not optimal might be a result of the employment of additional muscles to complete the task (that is of muscles to stabilize the body). Observation of subjects in the present study tends to confirm Hagberg et al.'s (1981) suggestion. At high cycling rates body stabilizers appear to play a greater role, with added upper body rigidity (isometric contraction of upper body muscles). At high power outputs and low velocity (40 rpm), more gross movement of trunk and the inclusion of the arm and shoulder muscles appeared necessary to maintain the cycling rate. In both situations, a higher intensity of work for body stabilizing muscles was employed and thus a probable increase in oxygen uptake was required. Increased work was accompanied by elevated blood lactate levels and likely increased vasodilation. In this study, it would appear that there was not an equivalent increase in cardiac output to meet the increased oxygen uptake. Coast et al.

(1986) found that although oxygen uptake varied with cycling velocities, circulatory catecholamines did not. They suggested that the difference in cycling velocities may not been sufficient to elicit a differential in norepinephrine response in spite of small oxygen uptake differences. The present data tends to support the findings of Coast et al. (1986), in that, cardiac outputs were not significantly larger with small increases in oxygen uptake (40 rpm compared to 60 and 0 rpm), but in larger oxygen uptake differences (100 rpm compared to 40 to 80 rpm) cardiovascular output was significantly different.

While cyclists tend to suggest that they feel most efficient at high cycling rates, most researchers have found lower rates (60 to 80 rpm) more efficient. As Hagberg et al. (1981) and Coast et al. (1986), suggest, preferred cycling rate may coincide with the predominance of muscle fiber type and vary from individual to individual. Previous research tends to suggest that a true variance exists in the most economical rate for each individual. It is further suggested that in normal individuals not trained or genetically equipped for higher muscular contraction efficiency, slower cycling velocities may be more efficient.

G. SUMMARY

This study clearly demonstrates that an equivalence in cycling velocities for most cardiovascular responses does exist over a wide range of power outputs for young physically active males. Additionally, there are cycling velocities which are beyond this range. That is, 60 and 80 rpm produce very similar cardiovascular responses which are the most economical (lower cardiovascular demands) on the system. In contrast, 40 and 100 rpm are beyond the range of optimal cycling velocities.

At high power outputs (70% VO_2max), perceived exertion appears to be a good indicator of differences in both oxygen uptake and blood lactate concentrations.

From a practical standpoint, these data suggest that in submaximal testing (for example oxygen uptake prediction) on a cycle ergometer 60 and 80 rpm can be used interchangably for young, physically active males, and that beyond these velocities significant physiological variations may occur.

These data would suggest the need for further investigation in three areas. First, to determine if these finding are appropriate for untrained as well as trained. Work by Boning et al., (1984), suggests this is true for young untrained males. Secondly, to determine if gender is a factor. And thirdly, to investigate these physiological variables using numerous cycling velocities greater than 40 rpm and less than 100 rpm to identify the exact range of optimal cycling velocities.

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APPENDIX A

UNIVERSITY OF ALBERTA CONSENT FORM

I acknowledge that the research procedures described on the attached form and of which I have a copy, have been explained to me and that any questions that I have asked have been answered to my satisfaction. I have been informed of the alternatives to participation in this study. I also understand the benefits (if any) of joining the research study. The possible risk and discomforts have been explained to me. I know that I may ask now, or in the future, any questions I have about the study or the research procedures. I have been assured that personal records relating to these experimental protocols will be kept confidential and that no information will be released or printed that would disclose personal identity without my permission.

I understand that I am free to withdraw from the study at any time. I further understand that if the study is not joined, or if there is withdrawal from it at any time, the quality of medical care will not be affected.

The person who may be contacted about the research is:

____
NAME of PERSON to CONTACT

____
NAME of SUBJECT (print)

____
SIGNATURE of SUBJECT

____
Telephone # subject

____
Signature of Witness

____
Date

INFORMATION SHEET

TITLE OF PROJECT: Cardiovascular response to 4 different cycling ve ities on the bicycle ergometer

INVESTIGATORS: Dr. H.A. Quinney; Dr .T. Kappagoda; Dr. R. Macnab Dr. S. terson; Dr. R G Ha nel and Mr. .V. Et inger

This project is designed to ine the cardiovascular response to four di cycling velocities and to provide a ine for cardiovascular response in normals to imum stress test in normals.

The study consists of th phases; a familiarization phase, a maximum s test and a treatment phase. The familiarizat phase is a one hour session during which you will oriented to the testing equipment and procedures of the study. The stress test phase is a one hour session in which you will be required to pedal to exhaustion on the bicycle ergometer (VO_2max test). For this test you will rest for 30 minutes and then begin pedaling at 60 rpm and a low resistance. The resistance will be increased every 2 minutes until you are unable to continue or maximum consumption has been reached. During this test respiratory gases and heart rate will be monitored continually; blood samples will be drawn from the anticupital vein prior to exercise and 5 min post-exercise. The third phase consists of 4 separate tests to be conducted 24 to 48 hours apart. On each occasion, you will cycle at a different cycling velocity (randomly assigned from 40, 60, 80 and 100 pm). Each test will consist of 14 minutes of cycling with heart rate and respiratory ga being monitored continually; impedance cardiography measures will be taken six times during the test, and blood lactate and hematocrit samples taken up to five times from the anticupital vein. You will cycle for 4 minutes at a resistance equal to 40% VO_2max, 5 minutes at 55% and 5 minutes at 70%.

Any information obtained from this study will be kept confidential and will be released only to those conducting the study. This information will be made available to others only with your approval. The group results will be used in publication.

You will be required to make six visits to the

U of A Hospital (Cardiology stress lab) over approximately a 2 week period. Each visit will take approximately an hour.

Please be assured that your participation in this study is entirely voluntary and refusal or withdrawal from it will not jeopardize you in any way. The investigators would be pleased to clarify any concerns you may have prior to or during the study.

APPENDIX B

SUBJECT EXPOSURE ORDER TO TREATMENTS

TREATMENTS - CYCLING RATE AT EACH TREATMENT

POST-ID #	#1	#2	#3	#4	COMMENTS
1 (AM)	80	100	40	60	Completed
2 (BS)	80	100	60	40	Completed
3 (CS)	100	80	40	60	Completed
4 (DR)	40	60	100	80	Completed
5 (DS)	40	60	80	100	Completed
6 (DE)	80	60	100	40	Completed
7 (DB)	80	40	60	100	Completed
8 (GB)	100	40	80	60	Completed
9 (JE)	40	80	100	60	Completed
10 (JB)	60	100	40	80	Completed
11 (JC)	40	80	60	100	Completed
12 (KR)	40	100	80	60	Completed
13 (MD)	60	80	100	40	Completed
14 (ND)	40	100	60	80	Completed
15 (PN)	80	40	100	60	Completed
16 (PC)	60	40	80	100	Completed
17 (RO)	100	60	40	80	Completed
18 (RH)	60	40	100	80	Completed
19 (TD)	80	60	40	100	Completed
20 (BW)	80	60	100	40	Injured
21 (KI)	60	100	80	40	Injured
22 (ROH)	100	60	80	40	Moved
23 (RD)	100	40	60	80	Injured
24 (DE)	60	80	40	100	Injured

NOTE: 1) There were 24 permutations of 4 cycling rates (ie. 4!=24). 24 subjects were accepted as volunteers and the order for cycling rate was a number draw.

2) Only 19 of the 24 finished all phases; 4 were injured during the study and one moved away

APPENDIX C

MAXIMUM OXYGEN UPTAKE TEST PROTOCOL

Maximum oxygen uptake testing was conducted on a constant load cycle ergometer. Each subject was given a VO_2max test (before any treatments to allow power output determination). The testing protocol utilized was as follows:

Resting Protocol

1) the weigh scale and metabolic cart were calibrated;
2) the subject was weighed to the nearest 0.1 kg;
3) the impedance cardiography (IC) tape and sports tester was affixed;
4) the subject rested for 30 minutes;
5) resting blood samples were taken;
6) the subject mounted the cycle;
7) respiratory gas analysis was initiated (averaged measures 30 sec);
8) the subject remained at rest for 2 min (to determine 2 min resting VO_2);
9) resting heart rate and blood pressure was taken;
10) resting impedance cartiography measures were

taken;

Test Protocol

11) subject peddled at 60 rpm and 100 watts for 2 minutes;

12) respiratory data was collected continually and averaged over 30 sec and HR recorded every 30 sec;

13) blood pressure was recorded in the last 30 sec of each load;

14) at the end of each workload the subject stopped peddling and IC was recorded over 5- 10 cardiac cycles (at end exhalation with breathhold ~5-10 sec);

15) resistance was increased by 50 watts and peddling resumed at same rate;

16) repeat steps 12 to 15 until maximum criteria attained;

Recovery Protocol

17) subject remained seated on the cycle;

18) BP was taken at 30 sec recovery;

19) 1 min recovery respiratory gases and HR were recorded;

20) 2:30 min recovery BP was taken;

21) 3 min recovery respiratory gases and HR were

recorded;

22) 5 min blood sample was drawn;

23) the metabolic cart was calibrated; and

24) subject remained in lab until HR and BP returned to normal values (HR $<$ 100 and BP $<$ 140/90).

Notes: Maximum criterion (one of following):

1) VO_2 increased less than 50 ml with increase in workload;

2) attained HR equal to 90% of age predicted VO_2max; or3) unable to continue.

APPENDIX D

TREATMENT TEST PROTOCOL

Subjects attended four treatment tests 2 to 4 days apart. The subject cycled at only one pre-designated cycling velocity (60, 70, 80, or 90 rpm) for each of these treatments. Each treatment was conducted at a different but pre-designated velocity. Each test consisted of cycling 4 minutes at a work load equated to 40% of VO_2max followed by an IC recording (10 sec of inactivity), 5 minutes at 55% of VO_2max and IC recording, 5 minutes at 70% of VO_2max and then IC recording followed by 3 minutes of recovery recordings. The protocol utilized was as follows:

Resting Protocol

1) the weigh scale and metabolic cart were calibrated;
2) the subject was weighed to the nearest 0.1 kg;
3) the impedance cardiography (IC) tape and sports tester was affixed;
4) the subject rested for 30 minutes;
5) resting blood samples were taken;
6) the subject mounted the cycle;

7) respiratory gas analysis was initiated (averaged measures 30 sec);

8) the subject remained at rest for 2 min (to determine 2 min resting VO_2);

9) resting heart rate and blood pressure was taken;

10) resting impedance cardiography measures were taken;

Stage One

11) adjust resistance to calculated equivalent power output of 40% VO_2max;

12) subject began peddling at treatment cycling velocity for 4 minutes with cycling rates being closely monitored;

13) respiratory data was collected continually and averaged over 30 sec and HR recorded every 30 sec (simultaneous HR readings were recorded in the last 15 sec of each workload using the impedance cardiograph and the sports tester);

14) blood pressure was recorded in the last 30 sec of each workload;

15) at the end of each workload the subject stopped peddling and IC was recorded over 5 - 10 cardiac cycle (at end exhalation with breathhold ~5-10 sec);

16) once IC data was collected the subject began stage

2 at same rate;

Stage Two

17) adjust resistance to calculated equivalent power output of 55% VO_2max;
18) subject began peddling at treatment cycling velocity for 5 minutes with cycling rates being closely monitored,
19) respiratory data was collected continually and averaged over 30 sec and HR recorded every 30 sec (simultaneous HR readings were recorded in the last 15 sec of each workload using the impedance cardiograph and the sports tester);
20) blood pressure was recorded in the last 30 sec of each load;
21) at the end of each workload the subject stopped peddling and IC was recorded over 5 - 10 cardiac cycles (at end exhalation with breathhold ~5-10 sec);
22) once IC data was collected the subject began stage 3 at same rate;

Stage Three

23) adjust resistance to calculated equivalent power output of 70% VO_2max;
24) subject began peddling at treatment cycling

velocity

for 5 minutes with cycling rates being closely monitored,

25) respiratory data was collected continually and averaged

over 30 sec and HR recorded every 30 sec (simultaneous HR read[illegible] recorded in the last 15 sec of each work[illegible] using the [illegible] cardiograph and the sports tester);

26) blood pressure was recorded in the last 30 sec of each load;

27) at the end of each workload the subject stopped peddling and IC was recorded over 5 - 10 cardiac cycles (at end exhalation with breathhold ~5-10 sec);

28) once IC data was collected the subject remained on the cycle for the recovery protocol;

29) the metabolic cart was checked for calibration and; results recorded;

Recovery Protocol

30) subject remained seated on the cycle;

31) BP was taken at 30 sec recovery;

32) 1 min recovery respiratory gases and HR were recorded;

33) 2:30 min recovery BP was taken;

34) 3 min recovery respiratory gases and HR were recorded;

35) 5 min post-exercise blood sample was drawn.

36) subject remained in lab until HR and BP returned to normal values (HR < 100 and BP 140/90).

APPENDIX E

INDIVIDUAL SUBJECT CHARACTERISTICS

SUB (#)	AGE yrs	HT cm	WT kg	VO2max (l/min)	VO2max ml/kg/m	MAX PO (W)	MAX HR (bpm)
1 (AM)	35	175	71.7	3.48	48.54	350	177
2 (BS)	35	191	96.5	4.68	48.50	400	191
3 (CS)	24	188	77.2	4.24	54.92	400	186
4 (DR)	31	180	81.4	3.82	46.93	325	177
5 (DS)	23	183	78.2	4.35	55.63	400	185
6 (DE)	17	173	64.2	3.52	54.83	300	199
7 (DB)	27	188	91.2	3.89	42.65	350	191
8 (GB)	30	165	70.4	4.27	60.65	400	199
9 (JE)	21	190	85.5	4.63	54.15	400	191
10(JB)	34	196	86.5	4.33	50.06	400	175
11(JC)	24	168	54.5	3.07	56.33	250	181
12(FR)	31	173	69.4	3.23	46.54	300	192
13(MD)	25	177	75.0	3.68	49.07	350	188
14(ND)	24	173	62.7	3.80	60.61	350	191
15(PN)	30	175	65.9	3.37	51.14	375	188
16(PC)	29	185	75.6	3.00	39.68	250	198
17(RO)	22	174	71.1	3.56	50.07	300	203
18(RH)	23	185	83.5	3.76	45.03	300	189
19(TD)	26	186	78.3	3.09	39.46	350	184
MEAN	27	180	76	3.93	52.10	355	187
STD	5	9	11	0.49	5.22	46	7.37
SEM	1.23	2	3	0	1	11	1.69

APPENDIX F

RAW DATA

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	W
1	40	40	1.13	87	109.76	8.67	142	72	6	95
1	40	60	1.35	101	90.67	9.07	152	84	7	95
1	40	80	1.47	102	90.23	8.75	146	76	9	95
1	40	100	1.81	116	118.15	13.00	166	84	7	95
1	55	40	1.67	116	110.95	13.09	178	82	6	145
1	55	60	1.93	123	105.70	13.11	176	68	7	145
1	55	80	2.08	133	106.30	13.71	182	78	9	145
1	55	100	2.39	133	118.52	15.41	178	66	7	145
1	70	40	2.54	143	106.30	15.20	184	74	7	195
1	70	60	2.36	148	105.22	15.36	182	70	9	195
1	70	80	2.54	152	104.27	15.64	190	78	11	195
1	70	100	2.93	152	118.15	18.20	186	74	9	195

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
2	40	40	1.73	92	156.01	13.26	146	56	7	125
2	40	60	1.72	89	174.32	12.73	152	82	10	125
2	40	80	2.01	101	161.68	13.10	158	78	10	125
2	40	100	2.29	100	155.64	14.79	178	92	10	125
2	55	40	2.81	121	147.51	17.41	184	88	13	215
2	55	60	2.83	121	153.48	17.96	178	76	12	215
2	55	80	2.94	126	147.21	17.81	184	82	13	215
2	55	100	3.15	145	132.28	18.52	188	90	13	215
2	70	40	4.13	164	135.59	22.64	206	90	15	295
2	70	60	3.91	161	139.44	22.31	192	80	15	295
2	70	80	4.05	162	120.31	19.37	208	88	15	295
2	70	100	4.06	174	136.89	23.96	196	88	17	295

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
3	40	40	1.83	129	96.71	11.99	144	68	9	140
3	40	60	1.79	122	97.79	11.83	144	72	6	140
3	40	80	1.85	135	92.99	12.18	130	60	9	140
3	40	100	1.99	140	96.98	13.77	156	70	9	140
3	55	40	2.71	152	114.98	17.25	164	70	12	205
3	55	60	2.57	149	107.95	15.76	172	66	8	205
3	55	80	2.59	157	107.60	16.57	158	64	9	205
3	55	100	2.66	157	108.48	17.14	164	70	10	205
3	70	40	3.21	165	115.63	18.50	174	78	14	240
3	70	60	3.01	162	113.00	18.31	176	64	9	240
3	70	80	2.96	169	104.33	17.63	176	64	11	240
3	70	100	3.08	166	110.03	18.27	170	66	11	240

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
4	40	40	1.44	106	100.19	10.22	154	82	7	95
4	40	60	1.46	102	113.53	11.01	160	80	9	95
4	40	80	1.44	107	108.47	10.96	150	74	9	95
4	40	100	2.04	123	127.24	15.01	166	80	7	95
4	55	40	2.31	137	111.68	15.19	174	84	11	165
4	55	60	2.28	130	123.12	15.51	184	78	11	165
4	55	80	2.34	138	113.45	15.32	180	78	11	165
4	55	100	2.91	150	127.26	18.96	176	78	9	165
4	70	40	3.05	159	117.45	18.79	182	74	13	210
4	70	60	2.92	152	122.07	18.19	188	76	13	210
4	70	80	2.83	159	118.12	18.31	18[illegible]	[illegible]	11	210
4	70	100	3.34	167	131.69	21.[illegible]	184	76	12	210

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
5	40	40	1.76	96	199.60	12.77	142	66	9	130
5	40	60	1.61	93	137.17	11.11	145	80	9	130
5	40	80	1.82	96	165.60	12.[illegible]5	142	70	8	130
5	40	100	2.41	100	172.14	16.70	158	66	7	130
5	55	40	2.50	113	169.78	16.81	148	74	13	190
5	55	60	2.31	107	174.47	15.70	156	80	11	190
5	55	80	2.54	119	179.68	16.89	178	70	10	190
5	55	100	3.34	125	182.10	20.39	164	70	9	190
5	70	40	3.23	145	162.41	20.46	186	78	17	250
5	70	60	3.08	138	140.86	18.88	178	84	14	250
5	70	80	3.18	157	137.14	20.57	182	72	14	250
5	70	100	3.95	159	141.01	22.00	176	78	15	250

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
6	40	40	1.48	128	123.13	11.08	134	54	7	105
6	40	60	1.55	123	124.65	12.59	124	56	8	105
6	40	80	1.57	124	119.91	12.11	118	60	8	105
6	40	100	1.81	158	101.92	14.78	118	62	10	105
6	55	40	2.21	159	108.86	16.76	148	52	11	145
6	55	60	2.09	150	106.07	15.38	134	58	11	145
6	55	80	2.19	147	111.61	16.07	138	56	11	145
6	55	100	2.31	176	99.25	16.87	134	58	13	145
6	70	40	3.02	178	105.16	18.51	152	56	17	195
6	70	60	2.95	175	109.92	18.69	148	56	14	195
6	70	80	3.00	175	112.81	18.61	152	54	14	195
6	70	100	2.92	190	96.87	18.40	158	56	17	195

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
7	40	40	1.60	103	122.37	11.99	142	72	8	105
7	40	60	1.41	103	117.31	11.26	144	62	8	105
7	40	80	1.61	116	110.68	12.40	138	74	8	105
7	40	100	2.31	130	121.54	15.80	168	86	6	105
7	55	40	2.34	126	130.31	15.64	184	58	11	160
7	55	60	2.04	124	118.17	14.65	168	66	10	160
7	55	80	2.17	136	116.15	15.45	164	70	11	160
7	55	100	2.83	152	118.08	17.95	186	76	7	160
7	70	40	3.12	160	120.32	19.01	188	68	15	215
7	70	60	2.71	154	113.16	17.09	182	74	17	215
7	70	80	2.74	163	110.86	17.63	178	68	16	215
7	70	100	3.20	177	115.84	20.73	194	86	12	215

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
8	40	40	1.71	106	121.91	12.19	144	58	10	130
8	40	60	1.76	102	144.36	12.27	144	68	10	130
8	40	80	1.73	101	121.16	12.12	148	62	10	130
8	40	100	1.98	122	133.24	13.46	166	70	11	130
8	55	40	2.51	132	128.57	16.59	178	66	13	175
8	55	60	2.18	120	131.67	15.80	162	54	12	175
8	55	80	2.28	120	134.69	16.43	176	56	12	175
8	55	100	2.45	142	117.20	16.53	180	66	12	175
8	70	40	3.24	170	113.27	19.03	198	68	14	240
8	70	60	3.03	150	125.20	18.28	184	62	13	240
8	70	80	3.11	162	119.92	18.71	192	60	13	240
8	70	100	3.37	176	115.[illegible]8	20.00	[illegible]	64	14	240

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
9	40	40	1.48	109	125.86	12.84	144	78	6	100
9	40	60	1.52	103	144.13	12.68	120	58	7	100
9	40	80	1.68	120	150.98	13.74	132	64	6	100
9	40	100	1.88	134	122.67	15.70	132	68	6	100
9	55	40	2.86	145	128.11	18.19	154	68	13	205
9	55	60	2.65	134	139.89	17.63	156	58	10	205
9	55	80	2.74	148	131.12	18.75	148	66	9	205
9	55	100	3.04	162	125.34	20.43	154	68	9	205
9	70	40	3.79	175	131.97	22.83	172	78	17	275
9	70	60	3.57	162	143.40	22.66	168	68	14	275
9	70	80	3.62	174	124.79	21.59	166	64	13	275
9	70	100	3.72	178	128.34	22.97	184	66	18	275

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
10	40	40	1.84	104	135.10	12.97	142	66	9	140
10	40	60	1.81	102	137.95	12.55	144	72	10	140
10	40	80	2.08	124	123.26	14.54	148	58	11	140
10	40	100	2.30	117	139.99	14.98	156	72	11	140
10	55	40	2.47	129	139.29	15.74	182	76	12	195
10	55	60	2.36	122	133.59	15.50	162	64	13	195
10	55	80	2.61	145	127.73	17.50	152	58	13	195
10	55	100	3.09	137	141.46	18.67	160	68	12	195
10	70	40	3.22	151	127.06	18.42	184	76	15	240
10	70	60	3.04	138	134.00	17.55	188	68	14	240
10	70	80	3.19	159	129.33	19.40	182	50	15	240
10	70	100	3.63	151	137.32	20.05	178	72	13	240

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
11	40	40	1.47	119	89.[illegible]	10.65	148	85	11	95
11	40	60	1.41	124	82.70	10.17	152	76	9	95
11	40	80	1.45	119	86.35	10.45	148	72	9	95
11	40	100	1.64	136	90.98	12.46	140	76	8	95
11	55	40	2.06	152	92.21	13.92	182	74	13	140
11	55	60	1.98	151	87.90	13.27	176	72	11	140
11	55	80	2.05	149	92.78	13.73	174	72	11	140
11	55	100	2.17	159	94.35	14.91	160	78	11	140
11	70	40	2.84	183	89.83	16.44	182	86	14	185
11	70	60	2.67	172	92.22	15.95	182	64	15	185
11	70	80	2.64	177	91.98	16.19	176	72	15	185
11	70	100	2.74	176	93.37	16.62	178	64	15	185

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
12	40	40	1.27	114	95.46	11.17	146	68	10	90
12	40	60	1.28	109	98.09	10.79	124	66	10	90
12	40	80	1.35	114	96.50	11.29	138	60	10	90
12	40	100	1.57	133	94.53	12.19	146	70	11	90
12	55	40	1.96	140	93.53	13.66	166	78	13	140
12	55	60	1.74	140	92.99	12.83	146	70	13	140
12	55	80	1.76	140	91.16	12.58	142	64	13	140
12	55	100	2.15	151	92.88	14.12	158	64	13	140
12	70	40	2.83	177	95.66	17.12	182	74	15	200
12	70	60	2.58	169	87.51	15.31	182	78	15	200
12	70	80	2.59	169	88.51	15.14	164	64	15	200
12	70	100	2.80	173	99.28	16.98	172	64	17	200

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
13	40	40	1.51	106	122.53	12.50	146	68	6	105
13	40	60	1.65	108	124.68	13.09	146	68	6	105
13	40	80	1.61	104	127.30	10.82	144	62	6	105
13	40	100	1.86	121	127.41	14.53	166	72	7	105
13	55	40	2.13	132	119.34	15.51	148	68	11	155
13	55	60	2.10	131	118.47	15.64	154	64	10	155
13	55	80	2.09	129	121.62	14.84	168	72	10	155
13	55	100	2.45	144	117.49	16.80	178	66	11	155
13	70	40	2.78	161	117.08	18.85	174	66	15	200
13	70	60	2.68	152	117.44	18.09	180	62	9	200
13	70	80	2.70	157	118.18	18.44	178	64	9	200
13	70	100	2.95	161	118.19	19.03	176	88	13	200

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
14	40	40	1.85	119	111.36	13.03	140	70	11	125
14	40	60	1.57	119	94.76	11.18	156	64	6	125
14	40	80	1.64	116	113.75	11.72	146	66	7	125
14	40	100	1.99	140	113.36	14.62	158	74	9	125
14	55	40	2.34	137	125.17	16.65	170	66	10	170
14	55	60	1.98	135	106.29	14.24	162	50	10	170
14	55	80	2.18	141	107.98	14.36	146	60	11	170
14	55	100	2.50	162	111.57	17.41	162	60	9	170
14	70	40	3.10	171	112.02	19.16	172	68	14	240
14	70	60	2.74	167	106.96	17.54	194	58	13	240
14	70	80	2.83	168	108.71	18.37	182	64	13	240
14	70	100	3.32	187	110.04	20.69	188	70	15	240

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
15	40	40	1.58	112	98.78	10.17	162	86	8	125
15	40	60	1.58	115	92.06	10.03	162	68	11	125
15	40	80	1.50	114	93.73	10.03	154	72	7	125
15	40	100	1.97	138	101.61	13.51	162	72	8	125
15	55	40	2.28	142	109.48	15.44	170	82	13	180
15	55	60	2.27	143	108.47	15.29	172	64	15	180
15	55	80	2.15	148	100.75	14.91	174	72	11	180
15	55	100	2.56	158	112.07	17.26	178	56	11	180
15	70	40	3.00	171	113.24	19.02	188	76	15	250
15	70	60	3.03	169	107.97	18.25	178	58	14	250
15	70	80	2.97	170	105.52	18.04	194	68	14	250
15	70	100	3.32	175	116.69	20.54	182	60	14	250

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
16	40	40	1.27	122	78.84	9.30	124	74	10	85
16	40	60	1.34	114	81.05	9.00	148	70	9	85
16	40	80	1.41	131	72.38	9.63	130	68	8	85
16	40	100	1.69	145	86.20	12.33	148	68	7	85
16	55	40	1.82	155	84.28	13.06	130	66	13	120
16	55	60	1.83	139	96.39	13.01	154	76	13	120
16	55	80	1.82	161	85.90	13.57	160	64	11	120
16	55	100	2.33	173	89.69	15.52	162	70	11	120
16	70	40	2.69	197	87.29	16.93	150	70	19	170
16	70	60	2.40	179	91.99	16.10	182	78	14	170
16	70	80	2.54	187	90.16	16.95	16[illegible]	64	15	170
16	70	100	2.90	195	89.42	17.44	16[illegible]	72	17	170

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
17	40	40	1.57	131	89.31	11.16	138	90	9	105
17	40	60	1.58	139	81.20	11 04	168	100	7	105
17	40	80	1.59	142	80.62	11.29	162	84	11	105
17	40	100	1.87	1[illegible]6	93.13	13.22	168	96	12	105
17	55	40	1.98	165	88.68	14.19	182	96	12	140
17	55	60	2.12	164	103.04	15.77	194	88	13	140
17	55	80	2.01	165	90.46	14.74	176	82	12	140
17	55	100	2.47	178	92.28	16.61	170	92	10	140
17	70	40	2.90	182	102.27	18.61	190	98	16	195
17	70	60	2.76	185	93.43	16.44	198	96	15	195
17	70	80	2.71	185	100.26	18.35	198	88	14	195
17	70	100	3.08	194	95.17	18.56	192	94	13	195

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
18	40	40	1.53	102	118.14	11.58	164	74	7	100
18	40	60	1.40	100	110.51	10.72	156	76	8	100
18	40	80	1.51	97	120.80	11.48	148	66	6	100
18	40	100	1.80	105	120.23	12.74	166	74	8	100
18	55	40	2.28	130	125.13	15.89	186	78	12	160
18	55	60	2.15	126	126.17	15.27	198	84	13	160
18	55	80	2.21	119	135.14	15.54	186	[illegible]2	11	160
18	55	100	2.44	132	125.77	16.60	192	78	10	160
18	70	40	3.20	165	123.29	20.34	198	82	15	220
18	70	60	2.89	159	121.42	19.18	206	80	15	220
18	70	80	2.92	155	124.53	18.80	192	84	13	220
18	70	100	3.13	164	120.91	19.83	208	78	16	220

ID	%MX	RPM	VO2	SSHR	SV	CO	SBP	DBP	RPE	WATTS
19	40	40	1.48	115	104.31	11.16	142	80	7	120
19	40	60	1.70	117	105.11	11.88	142	76	9	120
19	40	80	1.65	118	100.22	11.63	144	84	11	120
19	40	100	1.76	139	88.59	12.14	144	78	11	120
19	55	40	2.21	150	101.99	15.20	158	84	11	175
19	55	60	2.35	143	104.77	15.51	166	72	11	175
19	55	80	2.19	145	106.20	15.08	166	82	11	175
19	55	100	2.36	165	95.13	15.60	158	74	13	175
19	70	40	3.02	172	101.95	17.74	178	90	17	230
19	70	60	3.01	167	106.31	17.97	180	74	14	230
19	70	80	2.89	165	103.66	17.41	176	86	14	230
19	70	100	3.07	179	98.89	17.80	170	78	17	230

APPENDIX G

BLOOD LACTATE RESULTS AT 5 MIN POST-EXERCISE (mM)

ID	PRE	MAX	40 RPM	60 RPM	80 RPM	100 RPM
1	0.68	8.44	1.53	2.09	2.51	3.07
2	1.19	11.16	2.93	2.31	2.57	3.73
3	1.10	9.14	2.45	1.19	2.06	2.04
4	0.67	7.08	3.18	3.66	2.44	5.35
5	1.30	8.59	2.19	1.78	3.34	3.50
6	0.98	10.13	2.30	2.12	2.06	3.20
7	1.04	9.31	1.81	2.76	2.16	4.72
8	1.10	12.73	1.70	1.65	1.88	2.32
9	1.62	12.08	4.16	3.68	2.61	4.04
10	1.24	7.58	2.83	2.77	2.71	4.70
11	1.52	7.61	6.60	4.30	4.51	4.72
12	1.46	9.95	3.98	3.73	3.[illegible]	4.39
13	1.27	8.11	3.24	2.72	2.44	3.75
14	1.30	11.25	5.48	3.77	3.78	3.67
15	0.88	13.22	2.39	1.21	1.41	1.65
16	1.36	8.40	6.63	4.50	5.93	6.81
17	1.44	10.20	5.60	5.60	4.22	5.80
18	0.99	7.85	4.06	4.17	4.21	4.64
19	1.52	9.92	3.72	2.64	2.52	4.05
SUM		172.75	66.78	56.65	56.90	76.15
MEAN		9.09	3.51	2.98	2.99	4.01
STD		2.52	1.54	1.18	1.09	1.24
SEM		0.58	0.35	0.27	0.25	0.28

APPENDIX H

BLOOD LACTATE CORRELATION

RUN 1 AND RUN 2

RUN 1	RUN 2	RUN 1	RUN 2
4.63	5.48	4.14	4.21
3.29	3.77	5.44	4.64
3.31	3.18	7.40	7.36
4.12	3.67	1.41	0.99
8.88	11.25	1.89	2.35
1.07	1.30	7.36	7.69
3.07	4.14	4.43	4.16
10.14	12.59	1.81	3.68
2.04	2.93	1.74	2.61
2.41	2.31	2.91	4.04
3.51	3.46	11.82	12.08
3.73	3.36	1.40	1.62
11.16	9.84	4.40	4.67
0.84	1.19	9.08	11.40
2.40	2.04	1.31	1.70
12.93	9.24	1.25	1.65
3.25	4.06	1.49	2.32
3.34	4.17	1.32	1.88

Regression Output:

Constant	0.61	0.61
Std Err of Y Est	1.08	1.08
R Squared	0.90	0.90
No. of Observation	36.00	36.00
Degrees of Freedom	34.00	34.00
X Coefficient(s)	0.94	
Std Err of Coef.	0.05	

APPENDIX I

STATISTICAL ANALYSIS

ANOVA RESULTS

VARIABLE	SOURCE	VARIANCE SUM	DF	VARIANCE ESTIMATE	F	PROBABILITY
VO2	VO2/PO	72.42	2	36.21	113.16	p<.01
	VO2/CR	4.82	6	1.61	105.35	p<.01
	VO2/PO/CR	805.29	108	0.07	4.48	p<.05
HR	HR/PO	99811.48	2	49905.74	78.77	p<.01
	HR/CR	9190.43	6	3063.48	22.78	p<.01
	HR/PO/CR	805.29	108	134.21	4.96	p<.01
SV	SV/PO	416.80	2	208.40	0.12	p>.05
	SV/CR	224.69	6	74.90	113.04	p<.01
	SV/PO/CR	156.62	108	26.10	4.93	p<.01
CO	CO/PO	1645.10	2	822.55	96.77	p<.01
	CO/CR	155.36	6	51.79	94.37	p<.01
	CO/PO/CR	13.02	108	2.17	3.94	p<.01
MAP	MAP/PO	5152.80	2	2576.40	8.77	p<.01
	MAP/CR	641.67	6	213.89	22.78	p<.01
	MAP/PO/CR	3065.04	108	81.90	2.89	p<.01
SVR	SVR/PO	1357385.	2	678692.7	31.33	p<.01
	SVR/CR	152328.3	6	5260.88	27.67	p<.01
	SVR/PO/CR	31565.28	108	1835.29	2.87	p<.05
RPE	RPE/PO	1199.07	2	599.54	66.99	p<.01
	RPE/CR	16.20	6	5.40	3.16	p<.01
	RPE/PO/CR	32.40	108	5.40	3.16	p<.01
BDLA	BDLA/CR	101.25	54	1.87	13.36	p<.01

2 WAY ANOVA REPEATED

VARIABLE/PO: p(.01) = 3.17 p(.05) = 5.01
VARIABLE/CR: p(.01) = 2.70 p(.05) = 3.97
VARIABLE/PO/CR: p(.01) = 2.19 p(.05) = 2.98

1 WAY ANOVA REPEATED

VARIABLE/CR: p(.01) = 2.78 p(.05) = 4.17

APPENDIX I (PAGE 2)

POST HOC MEAN COMPARISON - NEWMAN-KEULS

Q TABLE - STUDENTIZED RANGES

VARIABLE	40-60	40-80	40-100	60-80	60-100	80-100
VO2 (LOW)	0.01	1.61	8.10**	1.59	8.09**	6.49**
VO2 (MED)	1.83	0.56	6.65**	1.27	8.48**	7.21**
VO2 (HIGH)	3.90**	3.08*	2.88*	0.82	6.78**	5.96**
HR (LOW)	0.38	2.83	8.95**	2.40	9.33**	6.93**
HR (MED)	2.03	0.83	6.80**	2.85	8.83**	5.98**
HR (HIGH)	3.25	0.60	2.93*	2.65	6.18**	3.53*
SV (LOW)	1.46	1.94	0.21	0.48	1.67	2.15
SV (MED)	0.27	0.70	0.89	0.97	1.17	0.20
SV (HIGH)	0.20	2.01	0.13	1.81	0.07	1.88
CO (LOW)	0.69	0.42	8.79**	1.11	9.49**	8.37
CO (MED)	1.56	0.43	5.52**	1.13	7.08**	5.95**
CO (HIGH)	2.90	2.33	3.09*	0.56	5.97**	5.40**
MAP (LOW)	0.81	1.77	4.60**	2.58	3.79**	6.37**
MAP (MED)	3.17	2.88	1.69	0.29	1.40	1.19
MAP (HIGH)	2.27	3.30	1.27	1.03	1.00	2.03
SVR (LOW)	1.54	1.89	7.31**	3.43*	8.86**	5.43**
SVR (MED)	0.67	1.30	5.04**	0.63	4.37**	3.74*
SVR (HIGH)	0.83	0.20	2.30	1.03	3.13	2.11
PRE (LOW)	0.33	0.58	1.41	0.25	1.08	0.83
PRE (MED)	1.50	2.24	3.72	0.75	2.24	1.50
PRE (HIGH)	4.53**	4.91**	1.41	0.37	3.12*	3.49*
BDLA (HIGH)	5.10**	4.97**	5.72**	0.13	9.81**	9.69**

MEAN COMPARISON:	DF	L TABLES p<.05	p<.01
Q_2 - Smallest mean comparison	2,72	3.75	2.82
Q_3 - Larger mean comparison	3,72	4.29	3.39
Q_4 - Largest mean comparison	4,72	4.58	3.73

** - $p<.01$
* - $p<.05$

APPENDIX J

LITERATURE REVIEW

The primary focus of this review of literature will be the cardiovascular and perceived exertion responses to cycling velocities. The review will also include submaximal and maximal determination of oxygen uptake and general physiological responses to dynamic exercise with specific concentration on cycle ergometry literature.

a. Cardiovascular Response to Dynamic Exercise

Some evidence suggests that the cardiovascular demands of short duration dynamic exercise can be met by the sympathetic drive (central control mechanisms) (Lehmann et al., 1981). Patterson (1928) suggests that the needs may be met by reflex afferent neural activity from skeletal muscle and joint receptors. Mitchell (1985) demonstrated that both central and reflex neural mechanisms can be responsible for cardiovascular changes during dynamic exercise. He contends that both the central neural control mechanism (central command, related to motor unit recruitment) and reflex neural control mechanism (activation of mechanoreceptors connected to group III skeletal muscle afferents) initiate the cardiovascular response and determine the beginning level of efferent activity of the autonomic nervous system to the heart and blood vessels. During exercise, Mitchell (1985) and others (Rowell et al., 198[illegible] Perez-Gonzalez, 1981) suggests a reflex neural mechanism exists which works on a feedback system from the exercising muscles and responds to metabolic changes. Therefore, during light-intensity work this reflex neural mechanism may not be activated; however, during moderate or high intensity dynamic exercise this mechanism appears to be sending signals to elicit efferent autonomic changes to meet the exercise demands (Rowell, 1980; Mitchell, 1985).

During dynamic exercise the cardiovascular response may be described by indicating the individual physiological responses as follows. Stroke volume reaches a maximum at an approximate heart rate of 110 to 120 beats per minute (Astrand et al., 1964). Once the maximum or peak stroke volume has been achieved, it is maintained at this level as long as the exercise intensity remains at this level or increases to submaximal levels (Clausen, 1976). Consequently, further increases in cardiac output must be achieved by increases in heart rate. Systolic blood pressure increases linearly with workload increas [illegible] oderate increases in mean arterial blood pressure occur and diastolic blood pressure decreases slightly or remains unchanged (Bezucka et al., 1982). The changes in mean arterial pressure are a result of changes in cardiac output and systemic vascular resistance. While cardiac output is related to external workload, in spite of the muscle mass employed, mean arterial pressure is influenced by both (Clausen, 1977). Exercise induced vasodilation is dependent on the size of the muscle mass employed and the intensity at which the muscle is working (Astrand, 1960).

A summary of the central and peripheral circulatory adjustment to dynamic exercise in normal healthy individual is as follows. Cardiac output increases linearly with oxygen uptake. This increase in cardiac output is accomplished by increased heart rate and stroke volume. The cardiac output is directed in a biased manner to the exercising muscles, the myocardium and other essential body functions by reducing blood flow to non-exercising tissue, and organs through sympathetic vasoconstriction (Clausen, 1976; Mitchell, 1985).

Further cardiovascular responses to exercise are discussed under each subheading.

b. Oxygen Uptake

The ability to perform activities of greater than 90 seconds duration depends on the ability to take in and utilize oxygen (Brooks and Fahey, 1984). Oxygen uptake increases as the intensity of dynamic exercise increases until it reaches a plateau or peaks where in spite of increasing exercise intensity, oxygen uptake does not increase (Astrand, 1960). oxygen uptake shows little variance for equal work and consequently, is an excellent indicator of work equivalency (Astrand, 1960).

i) Determination of Maximal Oxygen Uptake

Many investigators have devised maximal oxygen uptake tests for the treadmill and cycle ergometer (Astrand, 1956; Binkhorst, 1963; Taylor, 1955). These methods have been compared extensively. Astrand (1956) found no significant difference in maximum oxygen uptake determined on the treadmill (elevation of one degree) and the bicycle ergometer. Other researchers found that significantly higher VO_2max results are obtained in running on the treadmill (with an elevation greater than 3 degrees) than on the cycle ergometer. Astrand and Saltin (1961) found 5%

differences while Glassford et al. (1965) reported 8% differences. These findings agreed with Hermansen and Saltin's (1969) findings of 7%. Chase et al. (1966) found much higher differences (15%) in subjects unfamiliar with cycling. Astrand and Saltin (1961) found no difference in maximum oxygen uptake when skiing or cycling (employing the arms). Hermansen (1973) found significantly higher results by using a ski-walking technique (subject treadmill walking with slightly bent knees and using ski-poles) at 12 degrees incline, than normal treadmill running.

In general, maximum oxygen uptake tests using a cycle ergometer, produce lower maximum oxygen uptake values than using a treadmill. The difference is reduced, and in some instances negated, if the subjects are skilled cyclists (Astrand, 1956; Hermansen, 1969; Potirin and Josse, 1983).

There are various procedures for maximal oxygen uptake testing on the cycle ergometer. With careful and judicious control similiar results have been produced (Binkhorst, 1963; McArdle and Magel, 1971; Stamford, 1976). Protocols for VO_2max tests can be found in MacDougall et al. (1983).

ii) Prediction of Maximum Oxygen Uptake from Submaximal Tests

Direct determination of maximum oxygen uptake is not always practical or realistic for the population being tested. Therefore, the development of a simple, valid, reliable and safe method of determining maximum oxygen uptake was necessary. Numerous tests have been developed (Astrand and Ryhming, 1954; Sjostrand, 1949; Wahlund, 1948). Cardiac output and oxygen uptake are the most reliable and valid measures of cardiorespiratory function (Astrand and Ryhming, 1954; Cummings and Cummings, 1963). However, the expense and the practical difficulties of measuring oxygen uptake as a field test has resulted in the measurement of physiological parameters which are easier to measure and allow prediction of VO_2max. The linear relationship between oxygen uptake and heart rate during steady-state heavy exercise has clearly been demonstrated (Astrand and Ryhming, 1954; Cummings and Cummings, 1963). Based on this relationship between heart rate and oxygen uptake, numerous reseachers (Andersen, 1965; Assmussen and Hemmingsen, 1958; Workman and Armstrong, 1964) have developed submaximal tests which predict oxygen uptake from the relationship heart rate to power output.

In 1954, Astrand and Ryhming developed a nomogram to facilitate the prediction of VO_2max from the heart rate as determined at a known work rate on various pieces of apparatus. Adjustment, for age (Astrand, Astrand and Rodahl, 1959) and sex (Astrand, Astrand and Rodahl, 1959; Taylor, 1941) were made to account for the gradual decline in maximal heart rate with age and higher heart rate for equivalent work for females. Later, Astrand (1960) provided more direction for the use of the nomogram to increase the accuracy of its prediction. He suggested that the lowest submaximal load used to predict oxygen uptake should not be lower than the power output needed to elicit a heart rate of 125 beats per minute (b/min). Validity measures were performed comparing the nomogram to the treadmill and cycle ergometer maximum tests (Astrand and Ryhming 1954) and found a seven to nine percent error. Hettinger et al. (1961) reported a significant difference ($p<0.05$) between predictive and maximal testing of 28 policemen, ages 20 to 30. They suggested the difference was possibly caused by poor physical condition of the subjects and consequently maximal uptake may not have been attained. The nomogram's reliability was also verified on more policemen (Rodahl et al., 1961) and on older men (Astrand et al., 1959). The differences were 5% and 1% respectively. Reliability and validity measures were conducted on 78 young military trainees (Boning and Dahlstrom, 1962). The correlation coefficients were 0.97 for validity and 0.98 for reliability at heavier loads (150 watts) and 0.94 an. 0.90 at lighter loads (100 watts). Six maximal tests of working capacity were compared by de Vries and Klafs (1964). They found the Astrand-Astrand nomogram method and Sjostrand work capacity test (Sjostrand, 1947) produced the highest predictive values. The best correlations they attained between the submaximal test and maximal test w[illegible] 67 and 0.85. Rowell et al. (1964) found that [illegible] mogram underestimated VO_2max by between 5 and [illegible]% with one as high as a 27% underprediction. Glassford et al. (1965) compared three maximal oxygen uptake tests to the nomogram predictions on the Astrand six minute bicycle test (Astrand and Ryhming, 1954) on 24 healthy males. All tests produced significantly different results ($p<0.01$), however, when compared separately no significant differences existed. Correlations between the predictive test and each VO_2max test were 0.65 with the Astrand bike test (as described by Astrand, 1956), 0.72 with the treadmill test described in Taylor et al. (1955) and 0.78 with the treadmill test of Mitchell et al. (1958). It is also interesting to note that the submaximal values correlated as well as any correlation of the two direct measures. Many factors affect submaximal prediction of oxygen uptake from heart rate. A number of authors (Astrand, 1950; Toner et al., 1982; Taylor et al., 1955) have demonstrated that temperature, time of day, fatigue, mechanical efficiency, dehydration, meals, smoking, altitude, age, sex, emotional condition, exercise condition and muscle mass employed, affect heart rate and therefore, affect submaximal prediction of maximum oxygen uptake from heart rate.

c. Physiological and Perceived Exertion Responses to Cycling Velocities

i) Background of Cycling Velocity in Bicycle Ergometry

In 1899, Zuntz was one of the first scientists to investigate cycling efficiency. He measured oxygen uptake of walking and cycling at various speeds and found cycling to be 2.5 times more efficient than walking, in terms of oxygen uptake. The study of efficiency (oxygen cost) of cycling in relation to pedalling speed was first conducted on a stationary bicycle by Benedict and Cathcart (1913). They found that slower cycling rates produced greater efficiency, that is, a lower oxygen cost (this was with no resistance). Duffield and MacDonald (1923), looked at the relationship between speed and efficiency. Using one subject, the braking resistances were varied and oxygen uptake measured using cycling velocities of 40, 63 and 85 rpm. They concluded, similar to Benedict and Cathcart (1913), that the cost of cycling varies linearly with the rate of cycling.

Dickinson (1929) provided an indepth evaluatio of cycling efficiency in relation to speed a resistance, however, the range of these parameters wa not provided. She found the optimal cycling speed using a 7 inch crank, was 33 rpm for minimal oxygen cost over the range of 5 to 26 kilogram (kg) force on the pedal.

In the late nineteen fifties and early sixties, ergometer testing became extremely popular for submaximal and maximal determination of maximum oxygen uptake. In 1966, the Research Committee of the Internationl Council of Sport and Physical Education on standardization of ergometry proposed cycling frequencies appropriate for various power outputs which were: 30 rpm for 0 - 100 W (600 kpm/min), 40 rpm for 100 W - 200 W (1200 kpm/min), 50 rpm for 200 W - 300 W (1800 kpm/min) and over 60 rpm for over 300 W. As a result of these recommendations, numerous studies ensued investigating various physiological and stress perception responses to cycling velocities.

ii) Oxygen Uptake and Cycling Velocity

During steady rate cycling, oxygen uptake increases to meet the oxygen demand and then levels off at low and medium power output; at high power output (above anaerobic threshold) oxygen uptake continues to rise in spite of constant work being performed (Astrand, 1960). Some authors felt that variations in oxygen uptake for different cycling rates was a result of different mechanical efficiencies (Grosse-Lordemann and Muler, 1937; Eckermann and Millahn, 1967). There is however, a great disparity in the literature on oxygen uptake and cycling velocity.

Hess and Seusing (1962) investigated oxygen uptake and cycling velocities of 30, 60 and 90 rpm at power outputs of 30 and 60 W in 5 young (mean age 24) untrained male subjects. Oxygen uptake was significantly higher ($p<0.05$) for 90 rpm than for 30 or 60 rpm. No significant difference existed between 30 and 60 rpm.

Twelve untrained males (age 18-32) cycled for 6 minutes at cycling velocities of 30, 40, 60 and 90 rpm and power outputs of 100 and 150 W (Eckermann and Millahn, 1967). At low (100 W) and medium (150 W) power outputs the subject utilized more oxygen at 30 and 90 rpm than at 40 or 60 rpm ($p<0.01$). No significant difference was observed in oxygen uptake between cycling velocities of 40 and 60 rpm.

Banister and Jackson (1967) varied power output and cycling velocity and observed the oxygen uptake results of one subject, a 25 year old gold medalist oarsman. They experimented with cycling velocities of 50, 60, 70, 80, 100 and 120 rpm and power outputs at 60, 120, 180, 240 and 300 W and found that oxygen uptake at high PO was much higher for faster cycling rates (100 and 120 rpm). Oxygen uptake for 50 and 60 rpm was slightly lower than oxygen uptake for 70 and 80 rpm at low power outputs. At high power outputs, 50 to 80 rpm were very similar while oxygen uptake for 100 and 120 rpm were much higher. By using the Astrand and Astrand nomogram, they pointed out that the point of entry workrate and heart rate must be common to produce similiar VO_2max estimates. This does not occur at high cycling speeds.

Pandolf and Noble (1973), studied the response of fifteen male athletes to cycling speeds of 40, 60 and 80 rpm at three different power outputs (60, 125 and 175 W). They found no significant difference in oxygen cost between any of the cycling frequencies at each power output.

Twelve well-conditioned males (ages 19-24) were studied by Gaesser and Brooks (1975). Power outputs of 0, 33, 65, 100 and 130 W at cycling velocities of 40, 60, 80 and 100 rpm were investigated. Subjects pedalled at work rates of 33 and 65 W for 6 minutes each and at 100 and 130 W for 8 minutes each with a rest period between bouts (until oxygen uptake returned to pre-exercise values). Only one cycling frequency was performed on each visit to the laboratory. Net efficiency was calculated as work accomplished divided by energy expended above rest. Change or delta efficiency was the change in work accomplished divided by the change in energy expenditure. At 60 rpm net efficiency remained relatively constant with increased workloads. At other speeds, work efficiency tended to increase with lower work rate (33 - 65 W) and decrease with higher work rate (100 - 130 W). Delta efficiency remained relatively constant at 60 rpm for all work rates whereas, delta efficiency for 40, 80 and 100 rpm decreased as metabolic load increased. They postulated that the most linear relationship between caloric output and work rate occurred at 60 rpm.

McKay and Banister (1976) wanted to determine if higher cycling velocities on the ergometer could more closely approximate the higher VO_2max values attained on the treadmill. They had 5 male athletes perform maximally on a cycle ergometer at cycling velocities of 60, 80, 100 and 120 rpm and running speeds of 6, 6.5, 7, 7.5 mph on the treadmill. Treadmill VO_2max results were significantly higher than cycle ergometer results. no differences existed between the VO_2max results for different treadmill running speeds. Cycling velocities of 80 and 100 rpm produced significantly higher VO_2max results than 60 or 120 rpm. The mean maximum oxygen uptake values for 80 rpm were 4.70 l/min (±0.1) compared to treadmill values 4.89 l/min (±0.13) at 7.5 mph and 50.6 l/min (±0.13) at 7.0 mph.

Various cycling velocities (50, 60, 75, 85 and 100 rpm) at work rates estimated to equate to 60% of VO_2max, were investigated (Gueli and Shephard, 1976) on ten healthy, young (ages 22-31 years) non-smoking male physical education students. The subjects warmed up for seven minutes at 50 rpm, adjustin[illegible] load to reach a final heart rate 140-145 beats [illegible] and then exercised at power output assigne[illegible] to latin squares design at each of th[illegible]ng velocities (two minutes at each ve[illegible]ll subjects showed a decrease in breathing [illegible] at

60 and 70 rpm, however, at 85 rpm respiratory minute volumes were reduced (although not significantly). Oxygen uptake costs at 50 and 100 rpm were significantly higher than 60, 70 and 85 rpm; 70 and 85 rpm were lower (but not significantly) than 60 rpm in terms of oxygen uptake.

The difference between increasing cycling velocity or increasing resistance (load) in determining VO_2max was investigated by Moffatt and Stamford (1978) on the bicycle ergometer using discontinuous maximal tests. Twenty-nine males and nine female university students participated in the study. There was no significant difference between the two methods. However, for males in re-testing, there was a significant difference between the resistance methods of increasing load. Therefore, increasing cycling velocity for males was the more reliable method Oxygen costs of high work rates were higher at 60 rpm for men and lower at 60 rpm for women. For men performing at high power outputs (175-265 W) higher frequencies (60-90 rpm) were most efficient (the lowest oxygen cost).

Lollgen et al. (1980) investigated cycling velocities of 40, 60, 80 and 100 rpm at work rates equated to 0, 70 and 100% of VO_2max on 6 physically active males (ages 24-33). VO_2max was determined at

60 rpm. The subjects were tested on four occasions at a different frequency. Each test consisted of 6 minutes of pedalling with no resistance, 5 minutes at a power output equal to 70% of VO_2max followed by 10-20 second pause and 2.5 to 5 minutes at 100% of VO_2max. At a power output which would require 100% of VO_2max, no difference occurred in oxygen uptake as a result of cycling velocity. At 70% of VO_2max, no difference existed in oxygen uptake between 40, 60 and 80 rpm; however, 100 rpm was significantly higher.

Nine well-trained male cyclists (mean age 17.8) and six untrained male medical students (mean age 21.7) were subjects in a study investigating the relationship of power output (50, 100 and 200 W) and cycling velocity (40, 60, 70, 80 and 100 rpm) to oxygen uptake (Boning et al., 1984). At low power outputs (100 watt or less), oxygen uptake increased with increased cycling frequencies. At 200 watts, cycling velocities of 60 and 80 rpm required significantly lower oxygen uptake.

Buchanan and Weltman (1985) also used highly trained male cyclists using 60, 90 and 120 rpm and power outputs that elicited blood lactate (BdLa) levels of lactate threshold (lt), 2 millimoles/liter (mM) and 4 mM and maximum exhaustion. These levels resulted in different power outputs at different cycling velocities to elicit the desired BdLa levels. At 1t, power outputs were 210, 185 and 165 W for 60, 90 and 120 rpm respectively; at 2mM were 250, 235 and 195 W; at 4mM were 290, 280 and 245 W; and at maximum the power outputs were 333, 335 and 307 W respectively. They found oxygen uptake higher ($p<0.05$) for 60 rpm than 90 or 120 rpm; however, the power output between cycling velocities at each level were significantly different which is a confounding variable. When power output of near equal ranges were compared, oxygen uptake differed only slightly between 60 and 90 rpm; 120 rpm required higher quantities of oxygen for the same work (no test of significance w conducted on these values - means were compared).

iii) Heart Rate and Cycling Velocity

It has been demonstrated that a linear relationship exists between power output in ergometry performance and heart rate over the heart rate range of 100 to 170 beats per minute (Berggren and Christensen, 1950).

During cycling on an ergometer once heart rate has reached steady state it increases directly with oxygen uptake (Brooks and Fahey, 1984); this is especially true in trained subjects. Mellerowicz and

Smodlaka (1981) suggest that at low power output (50-100 W) heart rate steady states in 2 to 3 minutes and at medium (150 W) and high power output (over 200 W) there is a steady rise in heart rate. In aerobically fit subjects the heart rates at equivalent loads are lower. Increases in heart rates for equal increases in power output are also lower for trained subjects (Astrand and Ryhming, 1954).

Using untrained males cycling at low (100 W) and medium power outputs (150 W) at rates of 30, 40, 60 and 90 rpm, Eckermann and Millahn (1967) found no significant difference in heart rates elicited at 40 or 60 rpm. Higher heart rates ($p<0.001$) resulted at 30 and 90 rpm than at 40 and 60 rpm. Heart rates were lowest at 40 rpm for both PO.

Low (95 W), medium (125 W) and high (195 W) power outputs were investigated in 15 male athletes at cycling velocities of 40, 60 and 80 rpm (Pandolf and Noble, 1973). No significant difference was observed between heart rate and cycling velocity. The trend however, was that low velocities elicited the lowest heart rates at low and medium power outputs (40 rpm lowest heart rates, then 60 rpm and highest 80 rpm). Heart rates were 8 to 10 beats per minute higher for 80 rpm than for 40 and 60 rpm. At high power outputs, 60 rpm produced the lowest heart rates.

During VO_2max tests 5 male athletes cycled at 60, 80, 100 and 120 rpm and produced statistically similar heart rates (McKay and Banister, 1976). The mean heart rates were 82, 185, 183 and 182 for cycling velocities of 60, 80, 100 and 120 respectively.

Ten physically fit males cycled at 60% of VO_2max using cycling velocities of 50, 60, 70, 85 and 100 rpm for 2 minutes at each velocity after a 7 minute warm-up (Gueli and Shephard, 1976). Cycling rates of 70 and 85 rpm produced minimal heart rates; 40 rpm produced the highest heart rates and 60 and 100 rpm were very similar (statistical analysis was not performed on heart rate).

Michelli and Stricevic (1977) used power output of 100 W to evaluate cycling velocities of 40, 50, 60, 70 and 80 rpm on 15 healthy young male subjects. heart rates were recorded for each minute of the 6 minute rpm test. The heart rate, in all instances, steady-stated before the 4th minute of recording (with a mean increase of 4 beats per minute or less between the last three minutes of each test). A test of significance was conducted between all six minutes for all cycling velocities. The heart rate response to 40, 50 and 60 rpm were not significantly different, although 50 rpm produced the lowest mean heart rates. Higher heart rates ($p<0.05$) were elicited at 70 and 80 rpm with 80 rpm values being the highest.

Power outputs equated to 0, 70 and 100% of VO_2max at cycling velocities of 40, 60, 80 and 100 rpm were investigated on 6 healthy males (Lollgen et al., 1980). Heart rates at 0% of VO_2max (no load) increased as cycling velocities increased, that is, heart rates for 40, 60 and 80 rpm were significantly lower than those for 100 rpm ($p<0.05$) and heart rates for 40 and 60 rpm were lower than for 80 rpm ($p<0.05$). The lowest heart rates were obtained at 40 rpm. At high power outputs (70% of VO_2max), heart rates increased with cycling velocities but was only significantly lower for 40 and 60 rpm compared to 100 rpm. This trend continued at VO_2max except no comparisons produced a significant difference.

Seven elite male cyclists pedalled at 80% of VO_2max using road cycles on a treadmill selecting preferred gear ratios (mean rate of 91 rpm and a range of 72-102 rpm), 1 or 2 gears above and 1 or 2 gears below the preferred rate (Hagberg et al., 1981). The heart rates were lowest at preferred rates, slightly elevated at lower rates (+1.5%) and highly elevated at higher rate (+5.1%). No test of significance was conducted.

Boning et al. (1984) compared 9 trained (T) cyclists and 6 untrained (UT) subjects at cycling velocities of 40, 60, 70, 80 and 100 rpm and power outputs of 50, 100 and 200 W. Heart rates for trained were significantly lower than for untrained for all cycling velocities and all power outputs. Definite similar trends existed when the trained and umtrained were investigated separately (not analyzed statistically). At very low power outputs (50 W), heart rate increased as cycling velocity increased. At higher power outputs but still low range (100 W), 40, 60, 70 and 80 rpm produced very similar heart rates while 100 rpm produced much higher heart rates. At high power outputs (200 W), the untrained group registered the lowest heart rates at 60 rpm while heart rates for 80 rpm were slightly higher, heart rates at 40 and 70 rpm were slightly increased over 80 rpm and heart rates at 100 rpm were very high. For the trained group 70 rpm produced lowest heart rates closely followed by those for 60 rpm with HR's for 40 and 80 rpm higher still and heart rates at 100 rpm were extremely high in comparison.

Five trained cyclists worked at a power output of 85% of VO_2max for various cycling velocities (40, 60, 80, 100 and 120 rpm) to allow comparisons of 10 and 20 minute bouts of cycling (Coast et al., 1986). They found that heart rates were significantly greater for 20 minutes than 10 minutes of cycling at every velocity (approximately 10 beat difference). Heart rates at 80 rpm were lower than for all other cycling velocities ($p<0.05$) after both 10 and 20 minutes exercise. No significant difference existed between heart rate and any other cycling velocity, although the highest heart rates were recorded at 120 rpm. After 10 minutes of cycling, 100 rpm elicited higher heart rates than 40 or 60 rpm; this was reversed after 20 minutes of cycling.

iv) Stroke Volume and Cardiac Output

The ability to measure stroke volume and, or cardiac output directly (Direct Fick Method or Dye-Dilution Technique) during bicycle ergometry under normal circumstances is either not possible or feasible in healthy subjects. Numerous indirect methods of measurement have been developed including echocardiography (measuring end diastolic and end systolic volumes), radionuclide imagery, ballistocardiography, carbon dioxide rebreathing an impedance cardiography (Little, 1985; Mellerowicz and Smodlaka, 51). The following section shall only discuss impedance cardiography, its validation, reliability and reproducibility.

Impedance cardiography is based on the theory that the recorded impedance variations are caused primarily by changes in volume of the intrathoracic arteries due to the pumping action of the heart. Four electrode bands are placed around the neck and thorax and a weak alternating current is sent through the outer two bands which is undetectable by the subject and the high frequency of alternation is incapable of stimulating the heart. The changing amounts of current are recorded and stroke volume is calculated according to the formula of Kubicek et al. (1966) (Lamberts et al., 1984):

$$SV = \frac{P \cdot L^2 \cdot dZ/dt_{min} \cdot T}{Z_o^2}$$

where P = electrical resistivity of the blood at body temperature ($P = 53.2e^{0.022H}$) (Gebbes and Saddler, 1973), H = hematocrit (%), L = average distance (cm) between the inner bands measured at the anterior and posterior midline, Z_o = the mean transthoracic impedance (ohms) between the inner bands, dZ/dt_{min} = minimum value for the rate of change of impedance (ohms) occuring during the cardiac cycle, and T = the left ventricle ejection time (seconds). In order to reduce artifact during exercise, the system has been further refined (Teo et al., 1985; Hetherington, et al., 1986) by having the subjects stop all movement, remain motionless and hold their breath at the end of a normal expiration for five to ten cardiac cycles (approximately 5 seconds). Using impedance cardiography to determine stroke volume (and therefore cardiac output) has been validated with direct methods in over sixty studies on humans by forty different investigators with validation correlations varying from .49 to .96 (Lamberts et al., 1984). With refined techniqes, the validity measures have been greatly improved (.85 - .96).

A comparison between the cardiac outputs determined by dye-dilution and impedance cardiography methods was conducted on ten healthy active young males (mean age 20) on a cycle ergometer in an upright position at rest, 100, 195 and 295 W (Denniston et al., 1976). They found a significant correlation between both stroke volume (r=.90) and cardiac output (r=.84) as determined by t two methods.

Cardiac output measures were compared using two non-invasive methods, namely, an indirect Fick technique, the Gould 9000 IV system using carbon dioxide rebreathing and electrical bioimpedance cardiography using the Bo Med NCCOM3 machine (Smith et al., 1988). The study was conducted in 2 parts; first a resting study and later an exercising study. In the resting study, the subjects (15 males and 4 females, mean age 43.5, range 18-76 of varying medical and health conditions) rested for 10 minutes then cardiac output was determined with 3 to 8 pairs of simultaneous measurements taken every 5 min during supine resting. Eleven of the original subjects participated in the exercise comparison of the study (9 males and 2 females, mean age 28 and range 18 to 39). They found a significant correlation between the two methods ($r=.56$, $p<0.001$) with 73% of the values lying outside limits defined by lines of identity ±20%. Statistical significance was greatly reduced when stroke volume index was correlated ($r=.24$, $p<0.05$). They observed the stroke volume determined by impedance was consistently lower than stroke volume determined by an indirect Fick method (carbon dioxide rebreathing). They also found that the indirect Fick results were linearly related to oxygen uptake while impedance measures were not. They concluded that further validation is required before either method (machines) could be recommended as an alternative to invasive measurement. It should be mentioned that the method used in this paper (Sramek et al., 1983; taken from the published study being referenced) to calculate stroke volume and administering the test from impedance cardiography differs greatly from the Kubicek formula (Lamberts et al., 1984). The major differences being the calculation of the distance that the electrical impulse is impeded is less accurate (they used height and weight instead of actual measured distances) and they failed to stop all movement and breathhold at the end of normal exhalation. Breath holding establishes a stable baseline and reduces artifact thereby increasing the accuracy.

Teo et al. (1985) used the direct Fick method and impedance cardiography simultaneously on 4 patients with coronary artery disease (CAD) at rest and during cycle ergometry at 80 and 130 W. The correlation coefficient between the two methods for 40 measures over a cardiac output range of 3.5 to 18 litres per minute was r=.93. The systematic error was less than 5% in each subject. The reproducibility of impedance cardiography was also evaluated in this study on 6 healthy well-conditioned males (mean age 26.4). Measures were recorded at 30, 50, 80 W and additional 50 W increments to exhaustion. The correlation coefficient between the tests (test and re-test) was 0.84 for stroke volume, 0.98 for cardiac output and 0.98 for oxygen uptake. The cardiac output values were similar to those found by Astrand et al. (1964).

The stroke volumes and cardiac outputs of 20 patients with coronary artery disease was determined at rest and during supine cycle ergometry using direct Fick and impedance cardiography and the reproducibility of results was assessed in 5 patients randomly selected (Hetherington et al., 1985). No systematic error existed and the correlation between the two methods was 0.93. Test re-test correlation was high (r=.94).

v) Stroke Volume, Cardiac Output and Cycling Velocities

Cardiac output was assessed using dye-dilution technique on 23 well-trained subjects (12 males, 11 females; ages 20-30) on the cycle ergometer from rest to maximum exercise (Astrand et al., 1964). Cardiac output increased linearly with oxygen uptake from rest to 70% of VO_2max and again linearly from 70% of VO_2max to VO_2max with a reduced slope (that is decreased cardiac output for equal increase in oxygen uptake). They determined that during a maximum test to attain maximum levels over resting values, heart rate increased 4 fold, stroke volume increased less than 2 fold, cardiac output increased 6 fold and oxygen uptake increased 6 fold (average values for normal healthy subjects). They further surmised that arterial venous oxygen difference (a-vO_2 diff; the difference in oxygen content between the blood entering and that leaving the pulmonary capillaries) had to increase 3.3 fold since $VO_2 = CO \cdot a\text{-}vO_2$ difference. In these well-trained subjects, they found that stroke volume peaked or leveled off at a heart rate of about 110 or 120 beats per minute until near maximum exhaustion when in some cases stroke volume dropped slightly. Heart rate increases accounted for all the increases in cardiac output.

Grimby et al. (1966) examined heart rate, stroke volume and cardiac output in 11 healthy males (22-39 years of age) on a cycle ergometer at rest, 195 and 295 W using the dye-dilution technique. They found similar results, in that, stroke volume peaked within two minutes at 195 W and did not increase with a subsequent increase in power output while heart rate and cardiac output did increase. At 295 W, heart rate and cardiac output increased between minutes 5 and minutes 7 of exercise and remained unchanged for 30 minutes of exercise (stroke volume decreased gradually after 7 minutes work at higher power outputs).

No studies were found relating to cycling velocity and cardiac output or stroke volume measurement.

vi) Blood Pressure

Arterial blood pressure can be measured directly or indirectly during cycle ergometry. The indirect method is used most often. In healthy young males at the start of exercise, systolic blood pressure, like heart rate, increases rapidly and tends to mimic heart rate changes during exercise (Astrand et al., 1965). Systolic blood pressure reaches steady state within a few minutes at low and medium power outputs but rises continually at high power output (Astrand et al., 1965).

Hallmann and Hettinger (1980) found that diastolic blood pressure fell slightly as exercise increased when measured indirectly (with a blood pressure cuff); whereas it rose slightly (20 mmHg) (Astrand et al., 1965) when measured directly (peripheral artery). A moderate increase in mean arterial pressure (MAP - DBP + 1/3 [SBP-DBP]) occurred during exercise since cardiac output and systemic vascular resistance both increase (Bezucha et al., 1982). The amount of increase in mean arterial pressure is determined by the power output and exercise induced vasodilation of the arteries to the working muscles (Astrand, 1960). Vasodilation is dependent upon the muscle mass employed in the exercise. Higher pressures are produced by equal increases in cardiac output if a smaller muscle mass is employed (Astrand, 1960).

During exercise increased blood flow to the exercising muscles is achieved by both an increase in cardiac output and a redistribution of blood flow (Olsson, 1981). At low levels of exertion little redistribution of blood occurs; at moderate and high levels of dynamic exercise the blood flow is increased to the working muscles by vasoconstriction in the visceral bed and non-exercising muscles (Clausen, 1976).

The literature revealed no papers discussing blood pressure, mean arterial pressure or systemic vascular resistance and cycling velocities.

vii) Blood Lactate and Cycling Velocities

At rest, blood lactate levels are about 1 mM. During exercise an increase in lactate concentration in the working muscles indicates that the formulation of lactate exceeds its removal. Blood lactate concentrations have been used to indicate that diffusion into the blood exceeds removal or utilization. Blood lactate levels of 4 mM have been referred to as the lactate threshold or the onset of blood lactate accumulation (Karlsson and Jacobs, 1982). This increased lactate level in muscle and blood is indicative of anaerobic metabolism necessitated to supplement the aerobic production of ATP to meet the added demands of exercise (Astrand and Rodahl, 1986). Trained individuals can exercise at higher oxygen uptake levels than untrained individuals without an increase in blood lactate accumulation. In untrained subjects, blood lactate accumulation begins to increase at approximately 50 to 60% of VO_2max, whereas in conditioned individuals blood lactate begins to accumulate at 80-85% of VO_2max (Hurley et al., 1984).

Seven trained road cyclists pedalled at a preferred speed and 1-2 gears above and below the preferred speed at power output equated to 80% of VO_2max (Hagberg et al., 1981). The lowest blood lactate levels were recorded at the preferred cycling velocity while slower speeds resulted in slightly elevated levels and higher speeds produced extremely elevated blood lactate levels. They concluded that from a blood lactate standpoint, road-cyclists were more efficient (mean cycle velocity of 91, range 72-102 rpm).

The lactate levels in trained male cyclists and untrained males were evaluated at 40, 60, 70, 80 and

100 rpm at power outputs of 50, 100 and 200 W (Boning et al., 1984). They found no significant difference in lactate levels for trained subjects at any power output (lactate consistent around 1.0 mM at 50 and 100 W and below 2 mM at 200 W). In untrained subjects no significant difference existed between 50 and 100 W and cycling velocities which resulted in lactate levels of just over 1 mM and just under 2 mM respectively. However, in untrained subjects at 200 W, higher cycling velocities (70, 80 and 100 rpm) produced higher lactate levels (greater than 6 mM). The lowest results were obtained at 60 rpm (4.5 mM) and 40 rpm (5 mM).

Buchanan and Weltman (1985) found no significant difference in blood lactate levels in 9 male road cyclists between cycling rates of 60, 90 and 120 rpm and power output between 0 and 120 W. At 150 W, 120 rpm produced higher lactate levels than 60 rpm ($p<0.05$). Blood lactate concentration were significantly higher at higher power outputs (240 W and 300 W) for 120 rpm than 60 and 90 rpm. No significant difference was found between 60 and 90 rpm at any power output.

Blood lactates were investigated in 5 trained cyclists at 85% of VO_2max using cycling velocities of 40, 60, 80, 100, and 120 rpm (Coast et al., 1986) after 10 and 20 minutes of cycling. A significant difference was observed ($p<0.05$) between cycling velocity and blood lactate. After 10 minutes of cycling blood lactate concentrations at 120 rpm were higher than any other cycling velocity ($p<0.05$). Although no significant difference other than those already mentioned were evident, trends were apparent. At 10 minutes 80 rpm produced the lowest values (4.5 mM), 40 and 60 rpm next lowest (5.6 mM), 100 rpm slightly higher (6 mM) and 120 rpm excessively high values (7.5 mM). After 20 minutes of exercise the trend was quite different with lactate levels of 6.0 mM at 80 rpm, 6.5 mM at 120 rpm, 7.0 mM at 100 rpm, 7.5 mM at 60 rpm and 7.8 mM at 40 rpm.

Trained and untrained subjects cycling at 60 rpm did not differ significantly during VO_2max tests in blood lactate concentrations, although the values were higher at each power output for untrained subjects (Simon et al., 1986). Recovery profiles were similar, but blood lactate levels dropped more quickly in trained than untrained subjects (no significant difference).

viii) Perceived Exertion and Cycling Velocity

The degree of difficulty perceived by the subject during physiological exertion has been speculated to correlate with various physiological parameters. McKay and Banister (1976) found that athletes preferred cycling rates of 60 and 80 rpm over 100 or 120 rpm, and treadmill running at slower speeds over faster one during VO_2max tests.

In 1962, Borg, as part of his thesis developed a rating scale of rate of perceived exertion (RPE), also referred to as The Borg Scale, which allows the subject to associate a numerical score to levels of difficulty was published as follows (Borg, 1970):

6	
7	Very, very light
8	
9	Very light
10	
11	Fairly light
12	
13	Somewhat hard
14	
15	Hard
16	
17	Very hard
18	
19	Very, very hard
20	

This scale has been found to correlate better with heart rate than any other physiological measure. Many investigators have used the Borg Scale in cycling velocity studies.

The influence of training on the rate of perceived exertion investigated (Ekblom and Goldbarg,

1971) on 19 healthy males, showed that when blocking agents were used rate of perceived exertion correlated better with blood lactate than with heart rate. After training the rate of perceived exertion and heart rate decreased in a similar manner over pre-training results at equal workloads. But the rate of perceived exertion was the same at the same percentage of maximum oxygen uptake.

Gamberale (1972) compared 12 subjects on a cycle ergometer, wheelbarrow work and lifting weight and found that the rate of perceived exertion and heart rate were fairly linear for all types of work.

The rate of perceived exertion, heart rate, blood lactate and leg pains were shown to highly correlate (p<0.05) on cycle ergometry work in 28 healthy males by Borg et al. (1985). They stress that these results not be interpreted as causal relationships, but rather a complex physiological interaction of central and peripheral factors. Still a powerful prediction factor was observed between heart rate and blood lactate on the cycle ergometer.

Significant correlations (p<0.05) during intermittent and continous work (indicated respectively) between the rate of perceived exertion and power output (0.94, 0.96), oxygen uptake (0.91, 0.97), heart rate (0.85, 0.87) and blood lactate

(0.63, 0.77) resulted during cycle ergometry work at 60 rpm for 3 male subjects (Edwards et al., 1972). Work rate averaged from 80 W to 325 W.

Pandolf and Noble (1973) investigated the rate of perceived exertion for cycling velocities of 40, 60 and 80 rpm at power output of 90 W, 125 W and 195 W. They observed that at low, medium and high power outputs, 40 rpm was perceived most difficult (significantly different at $p<0.05$) and although no significant difference existed between 60 and 80, 60 was perceived the easiest at low power output and 80 the easiest at high power output. They found that heart rate and the rate of perceived exertion were not closely associated for different cycling velocities; in fact, the lowest heart rates (40 rpm) were associated with the highest rate of perceived exertion values (40 rpm).

Six active, healthy male subjects pedalled at 40, 60, 80 and 100 rpm at power outputs equal to 0, 70 and 100% of VO_2max to determine the rate of perceived exertion in conjunction with other physiological measures (Lollgen et al., 1980). The rate of perceived exertion was not correlated with heart rate or oxygen uptake. At 0 load, 80 rpm was perceived the easiest and 100 rpm was significantly different from 80 rpm. At VO_2max, 80 rpm ($p<0.05$)

was perceived the easiest while 60 and 80 were lower than 100 rpm ($p<0.05$). At high power outputs (70% VO_2max) 60 rpm was perceived the easiest and 40 the most difficult (significant at $p<0.05$). Also 80 rpm was perceived more difficult than 60 rpm but less difficult than 100 rpm (not significantly different).

Seven male cyclists with racing cycles on a treadmill pedalled at preferred ra s and 1-2 gears above and below this level at approximately 80% of VO_2max (Hagberg et al., 1981). The preferred rate was 91 rpm (range of 72-102 rpm) and slower rates ranged 34 rpm slower and higher rates ranged 45 rpm above preferred rates. The rate of perceived exertion and heart rate were lowest at preferred rates, but only slightly lower than slower rates. Higher rates of cycling elicited much higher results.

Coast et al. (1986) had 5 male racers cycle at 85% of VO_2max at cycling velocities of 40, 60, 80, 100 and 120 rpm and determined the rate of perceived exertion after 10 and 20 minutes of cycling on a Monark ergometer. They found 80 rpm at 10 and 20 minutes least stressful ($p<0.05$) and 40 and 120 rpm the most difficult at both time intervals. After 10 minutes the order of cycling velocities were perceived from easiest to most difficult as 80, 100, 60, 40 and 120 rpm. The only difference after 20 minutes compared to after 10 minutes was 60 and 40 rpm were perceived more difficult than 100 or 120 rpm. The authors felt that the lack of agreement between efficiency and the rate of perceived exertion might be explained by peripheral input. Nor did they find a significant difference between catecholamine levels and pedalling rates.

A significant difference was found between cycling rates of 30 and 60 rpm and the rate of perceived exertion at low power outputs (45 - 115 W) and high PO (230 W) (Henriksson et al., 1972). A cycling rate of 30 rpm at both low and high power outputs was perceived more difficult.

d. Summary

Extensive contradictory findings have been reported in the literature concerning the physiological and perceived exertion responses to cycling velocities in both trained and untrained healthy young males.

Despite the lack of total clear cut evidence, the majority of authors tend to agree on certain findings. At low power outputs (~100 W or less, <45% VO_2max), cycling rates of 40 to 60 rpm (some authors include 70 to 85 rpm) produce the lowest physiological responses while cycling rates of 100 rpm and over yield excessively high physiological responses. At high power outputs (over 180 W, bove 65% of VO_2max), the lowest physiological responses to cycling rates were obtained at 60 to 85 rpm (some authors include the range 50 to 90 rpm); and the extremely high physiological responses were obtained at cycling rates of greater than 100 rpm. For low power outputs (~100 to 180 W, between 45 and 65% of VO_2max) the findings are as varied as are the number of studies conducted.