National Library of Canada

Acquisitions and Bibliographic Services Branch

395 Wellington Street
Ottawa, Ontario
K1A 0N4

Bibliothèque nationale du Canada

Direction des acquisitions et des services bibliographiques

395, rue Wellington
Ottawa (Ontario)
K1A 0N4

Your file Votre référence

Our file Notre référence

NOTICE

The quality of this microform is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

If pages are missing, contact the university which granted the degree.

Some pages may have indistinct print especially if the original pages were typed with a poor typewriter ribbon or if the university sent us an inferior photocopy.

AVIS

La qualité de cette microforme dépend grandement de la qualité de la thèse soumise au microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction.

S'il manque des pages, veuillez communiquer avec l'université qui a conféré le grade.

La qualité d'impression de certaines pages peut laisser à désirer, surtout si les pages originales ont été dactylographiées à l'aide d'un ruban usé ou si l'université nous a fait parvenir une photocopie de qualité inférieure.

Canada

University of Alberta

Exercise Testing and Training in Traumatic Brain Injury

by

Gary James Rowland

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Science

Department of Occupational Therapy

Edmonton, Alberta

Spring, 1996

ISBN 0-612-10753-1

University of Alberta

Library Release Form

Name of Author: **Gary James Rowland**

Title of Thesis: **Exercise Testing and Training in Traumatic Brain Injury.**

Degree: **Master of Science**

Year this Degree Granted: **1996**

Permission is hereby granted to the University of Alberta Library to reproduce single copies of this thesis and to lend or sell such copies for private, scholarly, or scientific research purposes only.

The author reserves all other publication and other rights in association with the copyright in the thesis, and except as hereinbefore provided, neither the thesis nor any substantial portion thereof may be printed or otherwise reproduced in any form whatever without the author's prior written permission.

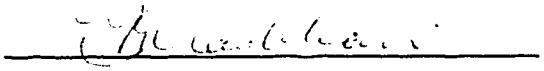
5301-55 Avenue
Leduc, Alberta, Canada
T9E - 5N8

Date: January 19/1996

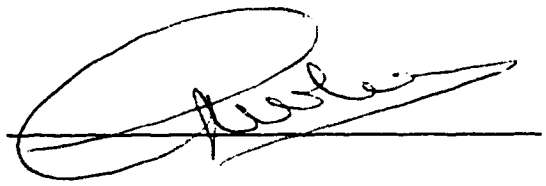
University of Alberta

Faculty of Graduate Studies and Research

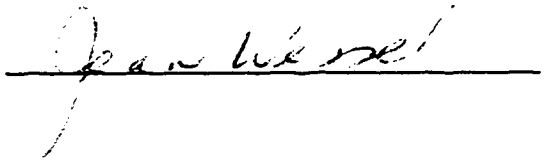
The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled "Exercise Testing and Training in Traumatic Brain Injury" submitted by Gary James Rowland in partial fulfillment of the requirements for the degree of Master of Science.



Y. Bhambhani, Ph.D.
Supervisor



Garry Wheeler, Ph.D.
Committee Member



Jean Wessel, Ph.D.
Committee Member

Date 21 December, 1994

Abstract

To examine the reliability of physiological responses during exercise in patients with moderate to severe traumatic brain injury, informed consent was obtained from 13 ambulatory subjects enrolled in an inpatient long term rehabilitation program. Subjects completed three identical symptom-limited incremental tests to exhaustion on a cycle ergometer. In addition, they completed two test sessions in which two cardiac output maneuvers were completed at each of three exercise intensities using the CO_2 rebreathing method. Physiological responses were continuously monitored using a metabolic cart, ECG, wireless heart rate monitor, and automated blood pressure apparatus. In addition to the physiological responses, body composition was estimated by bioelectrical impedance analysis. Pearson product-moment reliability coefficients for peak heart rate, oxygen uptake, ventilation volume, tidal volume and oxygen pulse measured during the three trials, ranged between 0.86 and 0.97 and were significant ($p<0.001$). Intraclass correlation coefficients for these variables were equal to or greater than 0.84.

Pearson product-moment correlation coefficients for cardiac output and stroke volume obtained in the second of two maneuvers performed at 50 % of peak $\dot{V}O_2$ were significant ($r= 0.85$, $p<0.01$). Intraclass correlation coefficients for these variables were 0.86 and 0.85 respectively. Neither mean arterial blood pressure or subjective rating of perceived exertion were found to be reliable in this study. Bioelectrical impedance analysis of body composition was highly reliable with Pearson and intraclass correlation coefficients equal to 0.99 ($p<0.001$).

Repeated measures analysis of variance indicated no significant differences ($a=0.05$) among the group means for the variables examined in the three graded exercise tests and the two cardiac output test sessions. However, considerable within subject variation between trials was observed when individual subject data were examined. The use of mean

data from two trials was found to substantially reduce the within subject variation for peak physiological responses.

Data from subjects with signs of brainstem damage indicated that, although reliability coefficients for this subgroup were similar to those of the subjects that did not show evidence of brainstem involvement, they demonstrated lower tidal volumes.

The results of this study suggest that maximal and submaximal physiological responses can be reliably monitored during cycle exercise in persons with TBI. However, given the within subject variability, caution is advised when generalizing group data to individual responses.

Preface

The information in this thesis is reported in the form of two paper:

Part A. Reliability of Physiological Responses During Cycle Exercise in Persons with Traumatic Brain Injury

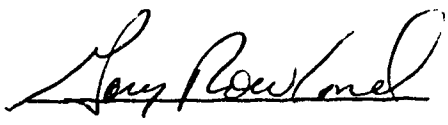
Part B. Aerobic Training Effects in Adults with Traumatic Brain Injury

ACKNOWLEDGMENTS

I am most appreciative of the support and guidance provided by Dr. Yagesh Bhambhani during the completion of this study. I would also like to thank the other members of my committee, Dr. Jean Wessel and Dr. Garry Wheeler, for their encouragement and advice. I would also extend my gratitude to the Department of Occupational Therapy, Faculty of Rehabilitation Medicine for the use of their equipment and arranging its transportation to and from Alberta Hospital Ponoka.

I am indebted to the men and women with traumatic brain injuries who volunteered for this study. My sincerest thanks are extended to these volunteers for their cooperation and effort. I would, in addition, like to thank the administration and staff of the Brain Injury Rehabilitation Program at Alberta Hospital Ponoka for their support. In particular, I wish to thank Sheila Ogilve-Zielke for her assistance in supervising the subjects in the exercise training program and Dr. Shawn Gray for his advice during the conception of this study.

I am deeply indebted to my dear wife, Theresa, who has supported my roles as student and therapist and helped me to balance these with the new role of parent.



Gary Rowland

Table of Contents

List of Tables

List of Figures

University of Alberta

Part A

Reliability of Physiological Responses During Cycle Exercise in Persons with Traumatic Brain Injury

by

Gary James Rowland

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Science

Department of Occupational Therapy

Edmonton, Alberta

Spring, 1996

Part A. Reliability of Physiological Responses During Cycle Exercise in Persons with Traumatic Brain Injury

INTRODUCTION

Persons who have sustained a moderate to severe traumatic brain injury (TBI) typically demonstrate a low tolerance for physical activity and are quickly fatigued during activities of daily living and exercise (Bray, Carlson, Humphrey, Mastrilli, & Valko, 1987; Perry, 1983). A lack of energy is a frequent and enduring symptom of even mild head injuries (Novak, Roth, & Boll, 1988). Fatigue, severe enough to impair work performance, is a common consequence of mild head injury (Giles, 1994). Being 'easily tired' was identified as a problem for individuals with TBI at six months (Oddy, Humphrey & Uttley, 1978) and at seven years (Oddy, Coughlan, Tyerman & Jenkins, 1985) by approximately 40 % of their relatives. When compared to healthy control subjects, brain injured subjects who were tested 20 days to 5.5 years post injury, displayed a 25 % to 40 % reduction in total lung capacity, vital capacity and forced expiratory ventilation (Becker, Bar-Or, Mendelson and Najensen (1978). Weakness in the respiratory muscles and reduced compliance of the rib cage in persons with TBI were suggested as possible reasons for these lower values. The subjects with TBI also had a lower oxygen pulse ($\dot{V}O_2$/HR) and a higher ventilatory equivalent for oxygen ($\dot{V}_E/\dot{V}O_2$), indicating that physiological changes had occurred which impaired ability to obtain and utilize oxygen (Becker, et al., 1978).

A review of the literature reveals a paucity of research pertaining to the reasons for low aerobic fitness in persons with TBI. It is reasonable to assume that the location and size of brain lesions and degree of diffuse axonal damage makes a significant contribution. Brain damage can result in paralysis or paresis of muscles of the trunk and limbs resulting in localized atrophy, a decrease in the muscle mass available to perform exercise and a reduction in the range of activities that can be performed. Paralysis or paresis can also affect muscles involved in breathing. Lesions in the cortex affecting input to or within the brainstem can affect cardiovascular and respiratory reflexes resulting in a diminished cardiorespiratory response to exercise (Lindsley & Holmes, 1984).

Prolonged recovery from the injuries to both the central nervous system and other parts of the body can result in a marked degree of deconditioning. It has been shown in healthy young adults that maximal aerobic power, measured as the maximum volume of oxygen consumed per minute ($\dot{V}O_2$ max, l/min), dropped by an average of 28 % after 20 days of bed rest (Saltin, et al., 1968). The period of bedrest for approximately 10 % of persons with TBI exceeds 3 weeks (Kalsbeek, McLaurin, Harris, & Miller, 1980) and for many the length of coma exceeds this time frame.

Cognitive deficits including amotivation, adynamia, poor self-awareness and self-monitoring and insight into their abilities and limitation (Levin, Benton, & Grossman, 1982; Prigatano, 1986; Prigatano & Altman, 1990) can play a role in the deconditioning process. Many individuals with TBI can be described as sedentary, and as a result of a decreased ability or willingness to maintain an appropriate level of activity, secondary health issues arise. Loss of lean body mass, increased obesity, decreased muscle strength and range of motion in the trunk, neck and limbs, further decreases in respiratory status and increased risk for cardiac disease and stroke may occur (Patterson, Pearson, & Fisher, 1985). Recently, an impairment in food intake regulation has been presented as another secondary factor associated with TBI. Subjects with TBI consumed larger meals than control subjects resulting in a 14 % higher caloric intake per day (Henson, De Castro, Stringers & Johnson, 1993). A decrease in physical activity, particularly upright ambulation, can reduce the stresses on the supporting bones to a level insufficient to maintain normal mineralization and the resulting decrease in bone density increases the risk for fractures (Astrand & Rodahl, 1986). Secondary changes, such as decreased muscle strength, range of motion and cardiorespiratory fitness may further reduce the TBI survivor's ability to engage in strenuous activity. Over protection by family and caregivers (Becker, et al., 1978) and a lack of self-confidence (Cohadon, 1981) are also thought to contribute to a reduction in activity. A progressively worsening cycle is thereby set into motion, where decreased activity leads to further declines in health and quality of life.

The use of physical conditioning programs has been recommended to improve the physical work capacity of persons with TBI (Cohadon, 1981; Howard, Huijbregts, & Miller, 1988; Moran, 1976; Sullivan, Richer, & Laurent, 1990). Two recent investigations reported that individuals with TBI could improve their aerobic fitness, measured as VO_2 max, through regular participation in strenuous exercise (Jankowski & Sullivan, 1990; Hunter, Tomberlin, Kirkikis, & Kuna, 1990). Subjects in these studies completed two graded exercise tests, one prior to and one following a period of exercise training. Both studies reported significant improvement in mean $\dot{V}O_2$ max; 15.2 % in relative VO_2 (Jankowski & Sullivan, 1990) and 14.4 % in absolute VO_2 (Hunter et al., 1990). Although the results of these two studies of persons with TBI appear to confirm the earlier subjective observations supporting the effectiveness of exercise training to improve aerobic fitness and physical work capacity (Cohadon, 1981; Howard, Huijbregts, & Miller, 1988; Moran, 1976), caution must be used before generalizing their results to all persons with TBI. Sample sizes in these studies were small and individual variability in improvement in VO_2 max ranged from - 1.6 % to + 42.1 %. For almost 60 % of the subjects in the study by Hunter et al. (1990) and 40 % of the subjects in the Jankowski and Sullivan (1990) study, the improvement in maximum oxygen consumption was less than 10 %. Both studies used subjects as their own control and compared peak physiological responses between single graded exercise tests performed before and after participation in an exercise program. This design is susceptible to threats to reliability, such as practice effects and between-test variations in motivation. Furthermore, factors such as maturation and statistical regression could affect the validity of the these results.

Although the reliability of $\dot{V}O_2$ max has been established for healthy subjects (Astrand & Rodahl, 1986) and for some populations with disabilities such as spinal cord injury (Bhambhani, Eriksson, & Steadward, 1991), cerebral palsy (Bhambhani, Holland, & Steadward, 1993) and mental retardation (Fernhall, Millar, Tymeson, & Burkett, 1990), it has

not been established that aerobic fitness can be reliably measured in persons with moderate to severe brain injuries.

It is essential from both a research and clinical perspective to establish the reliability of measurements of aerobic fitness in the TBI population. By establishing the reliability of submaximal and maximal physiological responses to exercise in this population, future research evaluating the effects of physical exercise programs can be more confidently considered and applied in rehabilitation. The primary purpose of this study therefore was to examine the test-retest reliability of: 1) peak physiological and perceptual responses during incremental cycle exercise in persons with TBI and 2) cardiorespiratory responses at three submaximal intensities in this population. A secondary purpose of this study was to examine the difference in physiological responses to exercise between subjects with clear evidence of brainstem injury and those where brainstem involvement could not be confirmed.

METHODS

Subject Selection

Subjects were recruited from the in-patient population of a post acute rehabilitation facility. Thirteen subjects, 9 males and 4 females, participated in this study after they, and where appropriate, their guardian provided written informed consent. To be considered for this study, subjects had to demonstrate the following criteria:

1) presence of traumatic brain injury rated as moderate to severe, based on the Glascow Coma Scale, duration of coma and/or post traumatic amnesia (Annegers, Grabow, Kurkland, & Laws, 1980; Jennet, 1976; Jennet, Snoak, Bond, & Brooks, 1982; Jennet & Teasdale, 1981),

2) absence of cardiorespiratory disease,

3) medically stable,

4) ability to perform exercise on a cycle ergometer and

5) ability to functionally communicate.

The procedures for choosing and testing of subjects was approved by the ethics committees of Alberta Hospital Ponoka and the Department of Occupational Therapy, University of Alberta. Some pertinent characteristics of the subjects are provided in table 1.

Testing took place in a laboratory set up in the rehabilitation facility using equipment on loan from the University of Alberta. All subjects included in the study completed three identical graded exercise tests and two cardiac output determination protocols consisting of two maneuvers performed at each of three intensities.

Test Protocols

Body Composition

Upon arrival to the laboratory, each subject's weight and height were recorded and their body composition estimated using a RJL body composition analyzer[a]. Bioelectrical impedance analysis (BIA) of body composition is a quick and non-invasive method to estimate body composition based on the principle that resistance to a low current (50 kHz) varies between lean body mass (body cell mass, extracellular water and the skeleton and other extracellular solids) and body fat (Twyman & Liedtke, 1988). Studies employing the RJL system have validated BIA determination of body composition against hydrostatic weighing in healthy adult males and females (Segal, Gutin, Presta, Wang, & Van Itallie, 1985) as well as in children and adolescents (Wu, Nielson, Cassady, Cook, Janz, & Hansen, 1993) with correlation coefficients of 0.91 or larger.

To obtain measurements of the total body resistivity, subjects were required to lie supine on a plinth. The placement of electrodes has been described previously (Segal, et al., 1985; Wu, et al., 1993). Two current injector electrodes were placed on the dorsum of the right hand and foot, over the distal end of the second and third metacarpals and

metatarsals, respectively. One detector electrode was placed on the dorsal surface of the wrist, on a line bisecting the styloid processes of the radius and ulna. A second detector electrode was positioned on the superior surface of the ankle on a line bisecting the medial and lateral malleoli. Resistance and reactance readings were obtained three times over the course of several minutes. Segal et al. (1985, pp. 1566) provide the following formulas for the determination of body density in adult males and females from resistance measurements, height and weight:

density (males) = 1.1554 - 0.0841 (weight x resistance) / height^2

density (females) = 1.1113 - 0.0556 (weight x resistance) / height^2

From the body density, the percent body fat (% fat) and lean body mass (% lbm) was determined using the formulas:

% fat = [(4.95/density) - 4.5] x 100 and

% lbm = 100 - % fat (Siri, 1956).

Graded Exercise Tests

Each subject completed three identical graded exercise tests (GXT) tests to volitional fatigue on an electronic cycle ergometer[b], with a minimum of two days and a maximum of one week between tests. Subjects were tested at approximately the same time of the day to avoid any circadian rhythm changes (such as variation in alertness or energy level between mornings and afternoons) that could affect their performance. Testing was scheduled a minimum of 90 minutes after meal time. Subjects were encouraged to drink only water and cautioned to avoid consuming food or sugary beverages during the 90 minute period before their test sessions.

The continuous graded exercise protocol was initiated with the subjects pedaling at 60 rpm at zero load for two minutes. This was followed by an increase of 25 watts for the male subjects and 15 watts for the female subjects every two minutes until volitional fatigue

was reached. As subjects approached their peak power output, they were verbally encouraged to exert their best effort. Metabolic and cardiorespiratory measurements were continuously monitored using an automated metabolic cart[c], an electrocardiograph[d] with bipolar leads in the CM_5 position, a wireless heart rate monitor[e] (WM), and an automated blood pressure cuff[f]. A description of the metabolic cart and the procedure used to calculate absolute oxygen consumption ($A\dot{V}O_2$, l/min) and carbon dioxide production ($\dot{V}CO_2$, l/min) using the Haldane transformation has been previously described by Bhambahani, Eriksson and Steadward (1991). The metabolic cart was programmed to obtain measurements averaged over 30 seconds. Heartrate (HR) was recorded from the WM during the last 15 sec of each minute of exercise. During the last 15 sec at each power output, a blood pressure reading was obtained and the subjects were asked to indicate their overall rate of perceived exertion (RPE) using the Borg scale (Borg, 1970; 1982).

The highest value recorded for oxygen consumption during the test was considered to be the peak oxygen uptake ($p\dot{V}O_2$). The values of the other variables reported correspond to the time at which $p\dot{V}O_2$ occurred.

Cardiac Output Maneuvers

After completing the three graded exercise tests, subjects returned to the laboratory and performed two identical protocols in which cardiac output (Q) was estimated twice at three submaximal intensities during each protocol. Cardiac output was first determined while the subjects were sitting at rest in a chair and then at two submaximal levels while cycling; 1) at zero load and 2) at a resistance set to elicit 50 % of the mean $p\dot{V}O_2$ obtained from the three graded exercise trials. This last resistance level was chosen for two reasons: (1) maximum stroke volume (SV) is reached at between 40 % and 50 % of $\dot{V}O_2$ max and (2) concerns that the steady state metabolic rate required for an accurate evaluation of Q and SV (McArdle, Katch & Katch, 1991) may not be attainable by subjects at an exercise intensity above this level . Resistance was closely monitored and adjusted in an attempt to keep the

level . Resistance was closely monitored and adjusted in an attempt to keep the subject's oxygen consumption near the 50 % of pVO_2 level and their cycling speed at 60 rpm.

Submaximal cardiorespiratory measurements were obtained using the same equipment employed in the three graded exercise tests. Cardiac output was determined using the automated CO_2 rebreathing procedure (Wilmore, et al., 1982) as previously described by Bhambhani, Norris and Bell (1994), using the Advanced Exercise Testing Program supplied with the metabolic cart. This procedure used the equilibrium method (Collier, 1956) for estimating mixed venous CO_2 tension.

Each Q maneuver was initiated once the subject has attained a steady state while resting and during submaximal cycle exercise. A steady state condition was assumed when the values for minute ventilation ($\dot{V}_E$), absolute $\dot{V}O_2$, HR and end tidal CO_2 concentration ($PetCO_2$) were consistent for one to two minutes. Subjects were then switched, from breathing room air, to breathing from a five liter rebreathing bag containing a mixture of 8 % to 13 % CO_2 and balance O_2. Varying concentrations of CO_2 were required in order to obtain a rapid equilibrium between the gas in the bag and the subject's lungs. A concentration of 8 % CO_2 was used during maneuvers at rest. Higher concentrations of CO_2 were selected based on the metabolic rate of each subject during the cycle exercise. Subjects were instructed to hyperventilate into the rebreathing bag until a 'plateau' in the partial pressure of CO_2 between the lungs and the bag was attained (Jones, 1985). An equilibrium in the CO_2 concentration between the mixed venous blood and alveolar gas was assumed when, during the first six to eight seconds of the rebreathing maneuver, the difference between their concentrations was less than one mm of Hg. When a 'plateau' was not achieved, an estimate of this value for PCO_2 was made using an extrapolation procedure (Jones, 1985, pp. 195). The value for arterial PCO_2 was assumed to equal the steady state $PetCO_2$, obtained immediately prior to the maneuver. The value for Q was calculated using

the steady state values for CO_2 production and the estimated difference in the venous and arterial CO_2 concentrations in the indirect Fick equation (McArdle, Katch, & Katch, 1991).

From these measurements of Q, SV was calculated as the ratio between Q and HR, while the arteriovenous oxygen difference [(a-v)O2 diff] was computed as the ratio between $\dot{V}O_2$ and Q. In order to control for the effect that body size might have on Q and SV, cardiac index (QI) and stroke volume index (SVI) were calculated by dividing Q and SV by the subject's body surface area (BSA). BSA was computed using the formula originated by (DuBois & DuBois, 1916);

$$BSA = [(weight)^{0.425} \times (height)^{0.725} \times 71.84].$$

Determination of Brainstem Damage

Injury to the brainstem can result in damage to the vagus nerve (cranial nerve X) and affect both the motor vagus fibers to the pharynx and larynx and the autonomic fibers to the heart and circulatory system (Farber, 1982; Gilroy & Holliday, 1982). The approximation within the medulla oblongata of the respiratory control centers to the nuclei and neural fibers of cranial nerves X and XII (Farber, 1982; Gilroy & Holliday, 1982), suggests that the presence of signs characterizing impairment to these cranial nerves could indicate an impairment in the cardiorespiratory responses to exercise. Thus, within the population of persons with TBI there could be individuals with brainstem damage, indicated by damage to these cranial nerves, that could have a markedly altered response to exercise. In the comatose patient ataxic breathing, characterized by an irregular rhythm and depth of respiration, is associated with damage to the respiratory centers (Gilroy & Holliday, 1982). Any residual inability to coordinate the rate and depth of respiration in individuals recovering from brain damage that included brainstem involvement, could result in decreased tidal volume (TV), minute ventilation ($\dot{V}_E$) and $\dot{V}O_2$ and increased $\dot{V}_E/\dot{V}O_2$ ratio.

In order to examine the effects that a brainstem injury has on overall fitness and the reliability of measures of physiological responses to exercise, subjects were classified into two groups on the basis of the presence or absence of brainstem injury. Several factors were taken into consideration in the determination of brainstem involvement, including: 1) the presence of overt signs of damage to cranial nerves X and XII such as dysarthria, dysphagia, dysphonia, inability to elevate the soft palate and asymmetrical wasting or deviation of the tongue (Farber, 1982; Gilroy & Holliday, 1982), 2) documentation of apneustic or ataxic breathing or abnormal eye reflexes such as ocular bobbing or skew deviation (Gilroy and Holliday, 1982) or 3) radiological evidence in the acute care medical record. The reliability of peak and submaximal physiological responses to exercise was compared between this subgroup of five subjects and all thirteen subjects as a single group.

STATISTICAL ANALYSIS

Several method were used to examine the reliability of peak and submaximal physiological responses to exercise. Repeated measures analyses of variance were calculated to examine the difference between the group means for the various peak and submaximal physiological responses. Pearson product-moment correlation coefficients were calculated to enable a comparison of the reliability of these responses with the reliability reported for able-bodied subjects and individuals with other forms of disability. Pearson correlations are frequently reported in studies examining the reliability of cardiorespiratory responses (Bhambhani, Eriksson, & Steadward, 1991; Bhambhani, Holland, & Steadward, 1993; Fernhall, Millar, Tymeson, & Burkett, 1990).

In order to compensate for two serious disadvantages described by Sale (1991) in using the correlation coefficient to judge the reproducibility of measurements; 1) sensitivity to the range of the values used in its calculation and 2) inability to provide a clear indication of the percent variation between trials, additional statistical analyses were performed. The intraclass correlation coefficients (Bartko, 1976), which takes into account the variability

(mean and standard deviation) between test trials, were calculated. In addition the method error, expressed as a coefficient of variation, was computed to provide an indication of the variability between trials. The percent difference between trials was calculated so that both the group means and individual subject data could be compared between trials. Because the possibility of making a Type 1 error increases with the number of test performed, statistical significance was determined using the Bonferroni adjustment (Ottenbacher, 1991) for an α value of 0.05 (two-tailed).

Graded Exercise Tests

Pearson product-moment correlation coefficients (Pagano, 1990) were used to examine the reliability of the peak value of selected variables obtained during the three graded exercise trials (HR; $\dot{V}O_2$; relative oxygen consumption, $R\dot{V}O_2$; carbon dioxide production, $\dot{V}CO_2$; V_E; TV; breathing frequency, F; O_2 pulse; respiratory exchange ratio, RER; mean blood pressure, MBP (0.67 x DBp + 0.33 x SBp); & rate of perceived exertion, RPE). Test-retest reliability of body composition (percent body fat, % fat) was also examined using Pearson correlations. Repeated measures analysis of variance was used to compare the means of the three trial for each of these variables. A *post hoc* analysis using the Scheffe procedure was performed on all significant F ratios to determine the effect of trial. SPSS (1988) statistical software was used to perform all statistical analyses, with the exception of the *post hoc* analyses which were examined using Statistica (1991) software.

Intraclass correlation coefficients (ICC) were calculated using the formula:

$$ICC = (MSB - MSW)/(MSB + (C-1)MSW),$$

where MSB = mean square between, MSW = mean square within and

C = the number of trials or maneuvers (Bartko, 1976).

The method error expressed as a coefficient of variation (CV) was also calculated using the formula:

CV = (SD/SQR(N-1)/((mean X_1 + mean X_2)/2), where

SD = the standard deviation of the difference between the two trials in the comparison, SQR = the square root, N = the number of trials and X_1 and X_2 refer to the two trials in the comparison (Sale, 1991). Differences for both group mean and individual subject data between the three trials were calculated as a percentage by the equations: [{(Trial 2-Trial 1)/Trial 1} x 100], {[(Trial 3-Trial 2)/Trial 2} x 100], {[(Trial 3-Trial 1)/Trial 1} x 100]}. The averages of Trials 1 and 2 and of Trials 2 and 3 were calculated and the percent difference between these two averages was obtained by the equation: [{(B-A)/A} x 100], where A = mean of Trials 1 and 2, and B = mean of Trials 2 and 3.

Cardiac Output Maneuvers

The same statistical analyses utilized in the examination of peak physiological responses were used to examine the test-retest reliability of variables obtained at a submaximal exercise level during the two Q output determination test sessions. Selected variables included HR, $A\dot{V}O_2$, $\dot{V}CO_2$, O2 pulse, $\dot{V}_E$, Q, QI, SV, SVI, $(a\text{-}v)O_2$ diff and TV. The reliability among the four maneuvers (Test 1a, Test 1b, Test 2a & Test 2b) performed at each of the three sub-maximum intensity levels (rest, cycling at zero load and cycling at an intensity equal to 50 % of $p\dot{V}O_2$) were examined using Pearson correlations, ICCs, coefficients of variation and the percent difference between maneuvers.

The percent difference between the four combinations of interest among the four maneuvers was calculated using the formulas:[{(Test 1b-Test 1a)/Test 1a} x 100], [{(Test 2b-Test 2a)/Test 2a} x 100], [{(Test 2a-Test 1a)/Test 1a} x 100], [{(Test 2b-Test 1b)/Test 1b} x 100]. The mean of Tests 1a and 1b was compared to the mean of Tests 2a and 2b and the percent difference between the means calculated as [{(Y-X)/X}x100], where X = mean of Tests 1a and 1b and Y = mean of Tests 2a and 2b.

Brainstem Damage

Separate two-way analyses of variance (maneuver or trial x presence or absence brainstem injury) were carried out to examine the effects that brainstem injury might have on the determination of peak physiological variables obtained from the three graded exercise trials and on submaximum responses obtained during the two trials to determine cardiac output and stroke volume. A Scheffe *post hoc* analysis was performed on all significant F ratios to determine the effect of brainstem injury on the physiological responses to exercise. In order to examine whether brainstem damage affects the reliability of measurements of peak physiological responses to exercise, Pearson product-moment correlation coefficients were calculated for; 1) the peak values of HR, $A\dot{V}O_2$, and $R\dot{V}O_2$ and 2) the submaximal values for Q, SV and $(a\text{-}v)C_2$ diff.

RESULTS

Graded Exercise Tests

The means and standard deviations for the peak values of the physiological and perceptual responses monitored during the three graded exercise trials are presented in table 2, along with the percent differences and correlation coefficients between the trials. Data from 12 of the 13 subjects were used in the calculation of statistics for RPE as one individual was unable to comprehend the Borg scale due to marked alexia.

The usual criteria that should be met to achieve a measurement of maximum $\dot{V}O_2$, were not met by the subjects in this study. Few subjects were able to meet the criterion of showing a plateau or decrease in $\dot{V}O_2$ while maintaining cycling (Astrand & Rodahl, 1986, McArdle, Katch, & Katch, 1991; Thoden, 1991). The mean values for peak HR indicates that subjects were also unable to meet the criterion of achieving their age-predicted maximum heart rates (Thoden, 1991). RER values greater than 1.0 indicate that the muscle cells are unable to meet the additional energy requirements aerobically and increasingly utilize anaerobic glycolysis (McArdle, Katch, & Katch, 1991). An increase in expired CO_2 results as

lactate is buffered by sodium bicarbonate. Although the mean RER values were just above the criterion value of 1.0 (McArdle, Katch, and Katch, 1991), the standard deviation of RER values indicates that many subjects were unable to meet this criterion. An RER value of 1.0 is considered to be low as a criterion for maximal effort and values exceeding 1.1 are often used (Bhambhani, Norris, & Bell, 1994). None of the subjects in this study were able to achieve RER values above 1.1 for all three trials. Therefore, the highest value of $\dot{V}O_2$ obtained while subjects were exercising represents their peak and not their maximum values. Therefore, it is likely that localized fatigue contributed to termination of testing. However, the high mean RPE values indicates that subjects perceived that they had reached the upper limits of their exercise tolerance on each of the three trials.

Repeated measures analysis of variance revealed that there were no significant differences between the means of the three trials for the variables in table 2. The Pearson correlation coefficients shown in table 2 indicate that the reliability of most of the variables was very high (>0.80) and significant ($p<.001$). Pearson 'r's ranged from a low of 0.82 for F (Trials 1 & 3) to 0.97 for absolute and relative $\dot{V}O_2$ (Trials 1 & 2). Intraclass correlation coefficients shown in table 3 were similar to the average Pearson 'r's for the 3 GXT trials. For the cardiorespiratory variables which showed high Pearson product-moment correlation coefficients, ICCs ranged from 0.85 for F to 0.94 for $\dot{V}O_2$ and $\dot{V}CO_2$.

The method error coefficient of variation for these variables are shown in table 4. For the cardiorespiratory variables which showed high reliability in table 2, coefficients of variation ranged from a low of 4.8 % for HR (Trial 1 & 2) to 13.4 % for $\dot{V}_E$ (Trial 1 & 3). For consecutive trials, all coefficients of variation were under 10 %. The fifth column in table 4 shows that the lowest coefficient of variation occurred when the means of consecutive trials were compared.

Pearson correlation coefficients (table 2) were low and non-significant for the variables RER, MBP and RPE. Intraclass correlations coefficients for these variables(table 3) were also low, indicating poor test-retest reliability.

For some variables, individual peak physiological responses for the three graded exercise trials are provided in Appendix 1. For each of the subjects, the percent differences between trials for these variables are provided in Appendix 2. The individual mean values for Trials 1 and 2 and for Trials 2 and 3 and difference between them are shown in Appendix 3. The range of within subject individual variation between trials is shown in table 5, where the variation between individual trials is also compared to the variation between the means of Trials 1 and 2 and Trials 2 and 3. While the percent differences in the group means for these variables (table 2) were low, ranging from -3.4 % (F) to +10.6 ($\dot{V}_E$), individual changes in responses between trials were much greater (table 5) and ranged from -28.8 % (F) to +52.4 % ($\dot{V}_E$). The fifth column in table 5 shows that the within subject individual variation was reduced for all variables when the means of consecutive trials (1 & 2, 2 & 3) were compared, for example -12.9 % (F) and +22.6 ($\dot{V}_E$). The range in percent difference was lowest for HR and RER across all three trials and also, when the means of consecutive trials were compared. Scatterplots for the peak cardiorespiratory variables which showed high reliability coefficients (HR, $A\dot{V}O_2$, $\dot{V}CO_2$, V_E, TV, F and O_2 pulse) are presented in Appendix 4. Separate scatterplots were not prepared for RVO_2 as the correlation coefficients were very similar to those for $A\dot{V}O_2$. Individual variation between trials can be readily seen in an examination of these scatterplots.

Cardiac Output Maneuvers

The reliability of the values obtained for selected cardiorespiratory variables obtained at rest and during cycling at zero load was determined to be poor, with low, inconsistent and often non-significant Pearson product-moment correlation coefficients. Only data obtained when subjects were cycling at a resistance level at which their oxygen

consumption was approximately 50 % of their peak $\dot{V}O_2$ showed consistently high correlations and were further analyzed. The means and standard deviations for the variables obtained during the four maneuvers at this intensity, as well as the percent differences and Pearson product-moment correlation coefficients between maneuvers are presented in table 6. Repeated measures analysis of variance indicated that there were no significant differences between the means of the four maneuvers for these variables. The Pearson correlation coefficients for measurements taken on the same day (1a-1b, 2a-2b) were significant ($P< 0.01$) and ranged from 0.76 ($[a-v]O_2$ diff, day 2) to 0.98 ($A\dot{V}O_2$, day 2). There were also high correlations between the second maneuvers performed during each test session (1b-2b), with correlation coefficients ranging from 0.74 for $(a-v)O_2$ diff to 0.95 for $A\dot{V}O_2$ and O_2 pulse. Comparison of the first maneuver between the two days (1a-2a), revealed that only HR, AVO_2, VCO_2, O_2 pulse and V_E showed strong correlations. While values for SV and $(a-v)O_2$ diff were not correlated in this comparison, Q showed a weak correlation.

The high correlations for $A\dot{V}O_2$ across all comparisons were expected as this variable was monitored closely and maintained at a level close to 50 % of $p\dot{V}O_2$, by adjusting the power output of cycling. Heart rate was also highly correlated due to the similarity in power output across the maneuvers. The Pearson 'r's for Q (0.89, 0.93, 0.86), SV (0.85, 0.81, 0.85) and $(a-v)O_2$ diff (0.90, 0.76, 0.74), for comparisons (1a-1b), (2a-2b) and (1b-2b) respectfully, indicate that the test-retest reliability of values for these variables is high and similar between maneuvers performed within the same test session and in test sessions separated by two to seven days.

Intraclass correlation coefficients using a) all four maneuvers and b) the second of the two maneuvers performed during each test session are presented in table 7, along with the average Pearson 'r's. The intraclass correlation coefficients for the four maneuvers ranged from 0.61 for SVI to 0.93 for $\dot{V}O_2$. These ICC values are similar to the average

Pearson 'r's but lower than the individual Pearson 'r's for the comparison between maneuvers performed on the same day (1a-1b, 2a-2b) and the second maneuver performed on each day (1b-2b), shown in table 6. The ICCs for the two second maneuvers (1b & 2b), also presented in table 7, were higher than the ICCs for all four maneuvers and similar to the Pearson 'r's for this comparison. The two maneuver ICCs ranged from a 0.74 for (a-v)O_2 diff to 0.95 for $A\dot{V}O_2$ and O_2 pulse. The ICCs for Q, SV, and (a-v)O_2 diff were 0.78, 0.68 and 0.70 respectfully, for the comparison using all four maneuvers, including comparison (1a-2a) which demonstrated low Pearson 'r's for some variables. The ICC's for the comparison (1b-2b) for these variables were higher, 0.85, 0.84 and 0.75 respectfully, for Q, SV and (a-v)O_2 diff. These ICC values are similar to the Pearson 'r' values for this comparison and give added support that test-retest reliability is significantly high for these variables.

The coefficients of variation calculated using the method error (Sale, 1991) are shown in table 8. The lowest variability for SV occurred between maneuvers 1b and 2b, where as that for Q and (a-v)O_2 diff occurred between two maneuvers performed on the same day. However, the difference between coefficients of variation (CV) for the comparisons (1a-1b), (2a-2b) and (1b-2b) for these variables was less than 3 %, indicating similar reliability between these comparisons. The CVs for Q ranged from 7.7 % to 10.5 %. The CVs for SV varied from 8.8 % to 10.8 % and those for (a-v)O_2 diff ranged from 6.2 % to 9.1 %.

Individual values for the variables under investigation during the four maneuvers to determine Q and SV are displayed in Appendix 5. The percent difference between maneuvers is shown in Appendix 6. The average of each days 2 trials and the percent difference between these two means are presented in Appendix 7. The range of within subject variation between maneuvers and between the average of the two maneuvers performed on each of the two days is shown in table 9. As was seen with the variables from the graded exercise tests, the percent difference between group data across the four

maneuvers was much lower (ranging from -9.4 % for $\dot{V}CO_2$ to +7.0 % for V_E) than the percent difference between maneuvers when individual data was examined (-39.9 % for SV to +66.8 % for (a-v)O_2 diff). Within subject variation was lowest for comparison of maneuvers performed on the second day (Trials 2a-2b), followed by the comparison of the second maneuvers performed on each day (Trials 1b-2b) and then the two maneuvers performed on day one (Trials 1a-1b). Each of these three comparisons resulted in a lower percent difference between trials than did the comparison between the average of each days two trials (X-Y). Scatterplots displaying the within subject variability for the variables HR, $A\dot{V}O_2$, $\dot{V}CO_2$, Q, SV and (a-v)O_2 diff are displayed in Appendix 8. An examination of these scatterplots reveals the presence of more outliers than was seen in the scatterplots for peak physiological responses.

Effects of Brainstem Damage

Graded Exercise Tests

For the peak values obtained in the three graded exercise test, two-way analysis of variance (brainstem x trial) revealed some significant differences ($p<0.02$) between subjects with overt evidence of brainstem injury and those that did not. It can be seen from table 2, that for trial 1, there were significant differences between the two groups for $A\dot{V}O_2$ ($p<0.003$), $R\dot{V}O_2$ ($p< 0.02$), $\dot{V}CO_2$ ($p<0.001$), $\dot{V}_E$ ($p<0.003$) and TV ($p<0.0001$). In trial 2, significant differences occurred for $\dot{V}CO_2$ ($p<0.006$), $\dot{V}_E$ ($p<0.02$) and TV ($p<0.0001$). Only TV ($p<0.008$) showed a significant between group difference for trial 3. There was no significant group difference across all three trials in the remaining variables shown in table 2, including HR, F, O_2 pulse. Applying the Bonferroni adjustment (Ottenbacher, 1991) for a p value of 0.05 (two-tailed) to the eight cardiorespiratory responses examined resulted in no significant difference in $R\dot{V}O_2$ for trial 1, in $\dot{V}_E$ for trial 2 and in TV for trial 3 at the adjusted p value of 0.006. However, setting the p value at 0.006 may be too stringent a choice to ensure controlling for Type 1 errors, and it seems reasonable to conclude that there was a significant difference in TV between the two groups. The means and standard deviations

for the variables that differed between those with and without brainstem injuries are shown in table 10.

The Pearson correlations for the five subjects with brainstem damage, are shown in the top half of table 11. It can be seen, when these correlation coefficients are compared to those of the entire group shown in table 2, that the reliability coefficients of these measurements were slightly higher for the sub-group with brainstem involvement.

Cardiac Output Maneuvers

Two-way analysis of variance (brainstem x maneuver) did not reveal any significant differences between the two groups for any of the variables obtained at the time the cardiac output maneuvers were initiated. For the three variables of primary interest; Q, SV and (a-v)O_2 diff, Pearson 'r's are presented in the bottom half of table 11. As was the case with measurements of peak physiological responses to exercise, the reliability coefficients for these submaximal physiological responses to exercise were slightly higher for the five subjects with brainstem damage than for the entire group.

The finding that the test-retest reliability coefficients for both peak and submaximal responses to exercise were not lower in subjects with clear indications of brainstem injury was unexpected. However, these high reliability coefficients do give added support to the finding that the subjects with brainstem involvement had significantly lower values than the subjects without brainstem injury for the peak variables $A\dot{V}O_2$, $R\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}_E$ and TV on some of the trials.

DISCUSSION

Comparison with Previous Studies

Subjects in this study were similar in many respects to those in the studies by Jankowski and Sullivan (1990) and Hunter et al. (1990). All the subjects in the study by Jankowski and Sullivan (1990) were reported to have sustained a closed head injury. Hunter et al. (1990) included persons who had experienced either a traumatic brain injury or

cerebral vascular accident. With the inclusion of the two individuals who had neurosurgery for a cerebral vascular condition with the subjects who had sustained an accident related traumatic brain injury, the subjects in this study are more comparable with those of the Hunter et al. (1990) study.

Age averaged 28.8 years and ranged from 18 to 45 years in the 13 males and 1 female who participated in the Jankowski and Sullivan (1990) study. The 3 females and 9 males in the study by Hunter et al. (1990) averaged 31.0 years in age and ranged from 26 to 48 years. Although there were younger subjects in this study, the average age was comparable; the 4 female and 9 male subjects averaged 29.7 (+/- 10.4) years of age and ranged from 18 to 47 years old.

Body fat content ranged from 8 % to 28 % for the male subjects and from 22 % to 36 % for the female subjects in this study. Average percent body fat content for all subjects in this study was 20.7 (+/- 8.3) %, 21.4 (+/- 8.3) % and 21.5 (+/- 8.7) %, respectively for the three trials. Percent body fat, calculated using skinfold measurements, average 19.5 % and ranged from 3.3 % to 27.6 % for the male subjects in the study by Jankowski and Sullivan (1990). Their only female subject had an initial body fat content of 26.7 %. Subjects in the study by Hunter et al. (1990) averaged 102.7 % of their ideal body weight, with values ranging from 92% to 176%. Given that a desirable fat content is 15% or less for males and 25% or less for females of this age group (McArdle, Katch & Katch, 1991), there were subjects in all three studies who were above their desirable body weight and fat content.

Prior to training, the peak $R\dot{V}O_2$ for the subjects in the study by Jankowski and Sullivan (1990) averaged 31.3 ml/kg/min and ranged from 14.2 to 45.2 ml/kg/min. The peak $R\dot{V}O_2$ for the subjects in this study averaged 26.4, 27.1 and 28.0 ml/kg/min for trials 1, 2 and 3, respectively. Individual values ranged from 12.3 to 46.3 ml/kg/min. Initial $A\dot{V}O_2$ for the subjects in the Hunter et al. (1990) study averaged 2.13 l/min and ranged from 1.26 to 2.83 l/min. This also compares favorably with the $A\dot{V}O_2$ recorded for the subjects in this

study, which averaged 2.11 (+/- 0.75), 2.18 (+/- 0.73) and 2.25 (+/- 0.84) liters/min for trials 1, 2 and 3, respectively. The individual subject values ranged from 0.96 l/min to 3.35 l/min. The slightly lower average peak $R\dot{V}O_2$ values obtained by subjects in this study is consistent with the lower peak $\dot{V}O_2$ reported when cycling is compared to treadmill running (Hermansen & Saltin, 1969; Hunter et al., 1990).

The similarity in nature of injury, age, body composition and oxygen consumption rates between the subjects in this study and those in the two previous studies which provided individual subject data (Jankowski & Sullivan, 1990; Hunter et al., 1990), emphasizes that low peak $\dot{V}O_2$ values are typical of this population. In addition, the low peak exercise HRs displayed by the subjects in this study, which ranged from 55% to 92% and averaged 84% of the age adjusted maximum, is consistent with the lower exercise HRs reported by Becker, et al. (1978). The average peak HR for the three trials in this study (145, 148, & 146) was lower than that reported by Hunter et al. (1990), which was approximately 172 beats/min. While TVs above 2.0 liters are common during exercise in healthy individuals (McArdle, Katch & Katch, 1991), TVs for subjects in this study averaged 1.65 (+/- 0.42), 1.76 (+/- 0.46), and 1.77 (+/- 0.41) liters for trials 1, 2 and 3, respectively. Low TVs are also consistent with the decreased vital capacity reported by Becker et al. (1978), as vital capacity limits the TV during exercise (Astrand & Rodahl, 1986; McArdle, Katch & Katch, 1991).

Reliability of Physiological Responses

Graded Exercise Tests

From table 2, it can be seen that the test-retest Pearson product-moment correlation coefficients for the first eight cardiorespiratory variables for the three trials were highly significant ($r>0.82$, $p<0.001$). Body composition, measured as % fat determined by bioelectrical impedance analysis, also demonstrated very high correlations ($r=0.99$, $p<0.001$) across the three trials. The intraclass correlation coefficients given in table 3 closely

approximated the Pearson product-moment correlation coefficients, giving added confidence that measures of peak physiological responses to exercise can be reliably measured.

The Pearson reliability correlation coefficients determined in this study for peak values of $\dot{V}O_2$ (0.94 to 0.97), HR (0.86 to 0.91), $\dot{V}_E$ (0.87 to 0.96) and O_2 pulse (0.92 to 0.96) are similar to the values reported by Bhambhani, Eriksson and Steadward (1992) for persons with spinal cord injury (0.98, 0.97, 0.96 & 0.96, respectively) and by Bhambhani, Holland and Steadward (1993) for athletes with cerebral palsy (0.89 to 0.92, 0.82, 0.92, & 0.87 , respectively). The reliability correlation coefficients for $A\dot{V}O_2$, HR and $\dot{V}_E$ reported by Fernhall, Miller, Tymeson and Burkett (1990) for adults with mental retardation were 0.93, 0.82 and 0.92, respectively. Therefore, the test-retest reliability of peak physiological responses during cycle exercise for persons with TBI compares favorably with that of other populations with physical and cognitive disabilities.

Although the reliability correlation coefficients for most of peak variables were high and significant and there were no significant differences between the group means between the three trials, an examination of the within subject variation between trials (Appendix 2) revealed considerable difference for some subjects. The method error coefficients of variation presented in table 4 and the range of within individual variation (expressed as the percent difference between trials) given in table 5 indicate that the use of the average of at least two trials may be warranted when measuring the peak physiological responses to exercise in persons with TBI. This has particular relevance to single subject study designs.

The reliability of RER ($\dot{V}CO_2/\dot{V}O_2$), MBP and RPE were determined to be poor. From table 4, it can be seen that the variability between trials was low for RER and RPE, but extremely large for MBP. The range in peak RER and RPE data is typically low, resulting in low correlations. The poor reliability of RER is not surprising, since both $\dot{V}CO_2$ and $\dot{V}O_2$ showed high reliability, when examined independently. The low correlation coefficients

and high coefficient of variation for MBP at peak exercise intensity indicates that there were considerable differences between trials. Increased use of the arms and trunk was observed in many of the subjects as they approached their peak effort. Increased muscle activity in the arm wearing the blood pressure cuff may have affected the validity of the blood pressure readings. Poor reliability of RPE could be due to an altered ability to monitor and assess exertion in some of the subjects. There was a tendency for some of the male subjects to deny that they were working harder as the resistance was increased. Prigatano and Altman (1990) examined the tendency for some individuals with TBI to overestimate their behavioral competencies while others underestimate them. They suggest that impaired awareness of behavioral limitations may be related to bilateral impairment of heteromodal cortex where information from the external environment is integrated with information about the internal, homeostatic state of the individual.

The results of this study indicate that peak physiological responses to exercise can be reliably measured in this population. The poor reliability coefficients for RPE in this study suggests that individuals with moderate to severe brain injury are inconsistent in rating the level at which they are exercising. This suggests that external supervision should be provided to ensure that individuals with TBI exercise at a safe intensity level that will promote improvement in cardiorespiratory fitness.

Cardiac Output Maneuvers

Test-retest reliability measured using the Pearson product-moment correlation coefficient was high ($p<0.01$) for comparisons made between the two maneuvers performed during the same test session and between the second maneuvers performed during each test session on separate days. From table 6, it can be seen that these correlation coefficients ranged from a low of 0.74 for $(a\text{-}v)O_2$ diff to 0.98 for $A\dot{V}O_2$ and $\dot{V}_E$. As $\dot{V}O_2$ was the factor which was carefully monitored and adjusted by changing the resistance level, a high correlation coefficient between trials for $A\dot{V}O_2$ was expected and achieved.

Pearson 'r's for Q for the two maneuvers performed during the same test session (1a-1b, 2a-2b) and between the second maneuvers performed during each test session (1b-2b) were 0.89, 0.93 and 0.86. These test-retest correlation coefficients compare favorably with the range of 0.86 to 0.96 reported in a review article on the validity and reliability of Q determination by CO_2 rebreathing methods in able bodied subjects (Marks, Rocchini, Beekman, & Rosenthal, 1985) and with the reliability coefficient of 0.89 reported by Bhambhani, Norris and Bell (1994). Reliability of Q measurements obtained in the first maneuver of each test session (1a-2a) was low (0.57, $P<0.05$). The Pearson 'r's for SV were lower than those for Q, as the variability in HR across maneuvers was a factor. This can readily be seen in the comparison of the first maneuvers during each test session where the combination of low reliability for both Q and HR resulted in a Pearson 'r' for SV that was low and not significant ($r=0.51$, $p=0.08$).

The intraclass correlation coefficients given in table 7 were similar to the Pearson product-moment correlations obtained for similar comparisons between maneuvers. The lower ICCs using all four maneuvers can be attributed to the poor reliability for the comparison between the first maneuvers during each test session (1a-2a) as demonstrated by lower and sometimes non-significant Pearson 'r's. The higher ICC's for the second of the two maneuvers performed at 50 % of $p\dot{V}O_2$, which were in fact the last of the six maneuvers performed during each test session, suggests that a steady state was not being maintained during the first of the two maneuvers.

As was seen with the peak physiological responses, despite the highly significant reliability correlation coefficients demonstrated for three of the four comparisons of interest, considerable within subject variability occurred. An examination of the coefficients of variation presented in table 8 and the range of within subject variation (expressed as the percent difference between trials) presented in table 9 reveals that the majority of the lowest values occurred in the comparison between the two maneuvers performed during the

second test session. This was followed by the comparison of the two maneuvers performed during the first test session and then by the comparison of the second maneuvers performed during each session.

Several factors could account for the better reliability between the two maneuvers performed during the second test session than the other comparisons investigated. Although subjects had become very familiar with the equipment used in this study during the performance of the three graded exercise tests, the added requirement to control their breathing just prior to initiation of the cardiac output maneuver and to breath rapidly from the rebreathing bag while maintaining a steady cadence of cycling were new to this part of the study. While the entire cardiac output test protocol was new to the subjects during the first session, they had the knowledge of what was expected and that they could complete the testing requirements during the second test session. Thus practice may be an important factor.

The test protocol used in this experiment required the subjects to perform 6 cardiac output maneuvers during each test session. Two maneuvers were performed while sitting at rest in a chair, two while cycling at zero load and two while cycling at a resistance adjusted to produce a $\dot{V}O_2$ of approximately half of each subject's $\dot{V}O_2$ max. Total cycling time ranged between 20 and 30 minutes and the majority of subjects rated their exertion using the Borg (1970; 1982) scale as approaching their maximal exertion level. It is likely that the duration of cycling and not the resistance level was a major factor in raising the level of perceived exertion in these subjects. Several subjects expressed after the first Q test session that they had doubts whether they could complete the final stage of testing. Factors such as motivation, fear, pain and discomfort may have been different between the test sessions.

The determination of Q requires that a steady state level of exercise be reached and maintained (McArdle, Katch & Katch, 1991). As every subject in this study demonstrated some degree of impairment in motor control, it is probable that there would also be a

reduced ability to regulate their response to exercise. The regulation of the cardiorespiratory responses to exercise and maintenance of a steady state metabolic rate may have been especially difficult to achieve for those subjects who demonstrated very low levels of aerobic fitness. It is, therefore, likely that there would be greater similarity between the values for the variables investigated in this part of the study as cycling time and effort increased and the interplay between HR, $\dot{V}O_2$, $(a\text{-}v)O_2$ difference and SV adjusted to the exercise level. The low correlations for SV and $(a\text{-}v)O_2$ difference for the first maneuvers of each test session supports this hypothesis.

This problem of taxing the subjects' physical work capacity could be avoided in future testing by eliminating the maneuvers while cycling at zero load and proceeding to the resistance level required to elicit 50 % $\dot{V}O_2$ max after a short warm up period of cycling. The maneuvers performed while the subject is resting will still be required for determination of resting SV and for the subjects to gain experience with the requirements of the testing.

The correlation coefficients for the comparison of the second maneuver performed during each test session suggests that it is possible to obtain reliable measurements of Q (Pearson 'r' =0.86, ICC =0.85) and SV (Pearson 'r' = 0.85, ICC = 0.84) in persons with TBI. Although not as high, (a-v)O2 diff also demonstrated reliability (Pearson 'r' = 0.74, ICC = 0.75). Additional research in this area, using a shorter protocol that does not overly tax the aerobic and muscular endurance of the subjects is warranted.

Effects of Brainstem Damage

An examination of the peak physiological response to exercise for the five subjects with overt signs of brainstem damage revealed significantly lower peak responses for $\dot{V}O_2$ (trial 1), $\dot{V}CO_2$ (trials 1 & 2), $\dot{V}_E$ (trials 1 & 2) and TV (all 3 trials). The consistently lower TV is consistent with that found by Becker, et al. (1978). It is not possible to attribute these lower values to any one cause. Damage to the respiratory and cardiovascular control

centers in the brainstem may have contributed to these differences. The severity of the brain damage as measured by length of coma may also be a factor. The average length of coma for the subjects with brainstem damage was 40.0 days compared to just 7.9 days for the other subjects. This prolonged period of coma could produce weakness of the respiratory muscles and decreased compliance of the chest wall and account for the low TVs as suggested by Becker, et al. (1978). There were no significant differences between subjects with and without brainstem injury for any of the variables obtained during the measurement of Q during submaximal exercise.

For both peak and submaximal exercise levels, contrary to the prediction that subjects with brainstem damage might demonstrate lower reliability, it was found that the Pearson product-moment correlation coefficients (table 11) for peak HR, peak $A\dot{V}O_2$, peak $R\dot{V}O_2$, Q and SV exceeded those obtained for the entire group of subjects (including those with and without evidence of brainstem damage). Thus, while lower peak responses to exercise might be expected in persons with brainstem damage, the test-retest reliability of measurements of the physiological responses to exercise are not likely to vary from that of the general population of individuals with moderate to severe brain injury.

IMPLICATIONS OF THE RESULTS

A comparison of the characteristics of the subjects in this study to those from two other research studies (Hunter et al., 1990; Jankowski & Sullivan, 1990) revealed that there was much similarity between the three sets of subjects. Peak physiological responses to exercise obtained in this study were also similar in those found in these two studies. The similarity between the three studies supports and gives credence to the assumption that this sample is representative of the population of persons with moderate to severe TBI.

The high overall reliability of measurements of the peak physiological responses to exercise shown in this sample of subjects indicates that the cardiorespiratory fitness of

persons with moderate to severe TBI can be reliably determined. The most reliable cardiorespiratory variables were $p\dot{V}O_2$ and $p\dot{V}CO_2$. Peak HR also showed high reliability. The variables $p\dot{V}O_2$ and pHR can therefore be used with confidence when evaluating cardiorespiratory fitness, prescribing training intensity and monitoring outcomes of exercise based rehabilitation programs for the TBI population. Bioelectrical impedance analysis of body composition was also shown to be a reliable measure in this population. The use of reliable measurements of body composition can contribute to knowledge about a person's level of fitness and progress towards improvement.

The reliability of Q and SV was lower than that of measurements of the peak physiological responses. The requirement that subjects maintain a steady state metabolic level for the procedure to determine Q dictates that the exercise level must be submaximum. At submaximum exercise levels there could be a multitude of combinations of the degree to which various physiological responses to exercise can contribute to $\dot{V}O_2$ and Q. Therefore, it is not unexpected that the reliability of Q and SV would be lower than the reliability of variables observed at peak exercise levels. Although lower, the reliability of Q and SV was found to be high enough to warrant consideration of their use in the assessment of cardiovascular fitness and monitoring the outcome of rehabilitation efforts.

The presence of brainstem damage or possibly the length of coma was shown to be a factor that lowered the mean values for some of the peak physiological responses to exercise. However, there was no reduction in the reliability of measurements of these peak response or for Q and SV in the subjects with brainstem damage. Therefore, given that there are no physical or cognitive limitations that prevent a person from performing the test protocol, individuals with evidence of brainstem damage, who may appear very unfit, should not be excluded from fitness testing.

The degree of within subject variability between values for the physiological responses obtained during graded exercise trials and Q maneuvers suggests that caution

should be used when interpreting the results of single exercise tests. Within subject variation for peak physiological responses was reduced when the average of two trials was used. Similarly, the use of multiple measurements performed on separate days with a simple protocol that does not tax the subjects endurance is recommended for determination of Q and SV.

The results of this study should encourage additional research in persons with TBI. Caveness (1979) in a review covering the period between 1970 and 1976 reported that the annual incidence of head injury in the United States was 3.6 % (over 7 million reported cases) and estimated that about 25 % of these cases involved major head injuries with the potential for brain injury. More recently, Frankowski (1986), in a review of epidemiological studies of head injury in the United States, estimated that the annual incidence rate of TBI, defined as including evidence of intracranial injury, skull fracture, loss of consciousness, or posttraumatic ranged from 0.2 to 0.3 % of the population (Frankowski, 1986). Therefore, of the estimated 7 million Americans who sustain some type of head injury annually, there are more than 400,000 injuries requiring hospitalization for brain injury (Frankowski, 1986). It has been estimated that over 40,000 patients spent 3 weeks or more in hospitals in the United States as a result of head injuries (Kalsbeek, McLaurin, Harris, & Miller, 1980). Only approximately 10 % of brain injuries are fatal (Frankowski, 1986). Advances in life saving medical techniques are resulting in the survival of increasing numbers of seriously brain injured patients, many of whom have the potential to live normal life spans, resulting in a growing population of persons with brain injury (Smigielski, Malec, Thompson, & DePompolo, 1992). Unfortunately, the majority will experience long-term, if not permanent physical, cognitive and psychosocial deficits (Finlayson & Garner, 1994; Jennet & Teasdale, 1981; Levin, Benton, & Grossman, 1982; Prigatano, 1986; Ylvisiker & Gobble, 1987).

For persons in the United States with head injury, direct care costs ('the dollar value of real goods and services associated with health care related to injury') and indirect costs

('the monetary loss incurred by society when the productivity of the injured person has been interrupted') was estimated to be 2.6 billion dollars in 1974 (Kalsbeek, McLaurin, Harris, & Miller, 1980, pp. S29). Although initial studies (Hunter et al., 1990; Jankowski & Sullivan, 1990) have reported improvements in cardiorespiratory fitness in persons with TBI, and a relationship between improved physical fitness and alertness and attention (Smigielski, Malec, Thompson, & DePompolo, 1992) has been suggested, the benefits of including aggressive aerobic training in rehabilitation programs warrants continued research. Given the incidence rate, increasing prevalence and high costs of care for this population, research into the contribution that improved cardiorespiratory fitness can make in changing the lifestyle patterns and cognitive abilities of persons with TBI is greatly needed.

The results of this study indicate that peak and submaximal responses to cycle exercise can be reliably measured in persons with TBI. A review of the literature has not revealed any previous reports of the reliability of these measurements in a sample of persons with TBI.

Table 1
Characteristics of Subjects

Subject	Age	Time post-injury	Coma	Height	Weight	Cause of TBI, location of injury
No., Sex	(years)	(months)	(days)	(cm)	(kg)	
1, F	22	19	18	171.5	78.5	MVA, Lt. cerebral hemis, & brainstem
2, M	28	13	56	181.0	100.5	MVA, diffuse, global & brainstem
3, M	24	7	49	167.0	63.6	MVA, diffuse, global & brainstem
4, M	23	9	14	172.5	67.7	AVM surgery, Lt. CVA
5, M	20	50	8	177.0	80.9	MVA, Rt. frontal
6, M	27	6	21	183.0	88.0	MVA, Rt cerebral hemis, mild diffuse cerebellar atrophy
7, F	22	9	42	170.5	74.7	MVA, diffuse, global & brainstem
8, M	34	57	21	171.0	100.0	Rt. fronto-parietal cerebral abscess
9, F	47	13	14	158.0	56.0	MVA, anoxia, Lt. frontal & diffuse global,
10, F	47	45	1	160.0	70.0	MVA, diffuse, global
11, M	29	10	35	167.0	77.3	IA, Lt. frontal & brainstem
12, M	18	5	3	177.0	87.8	anoxia, global, diffuse
13, M	45	19	1	176.0	98.4	Fall, Lt. fronto-parietal
Mean	29.7	20.2	20.2	171.6	80.3	
SD	10.4	18.1	19.2	7.4	14.2	

MVA - motor vehicle accident

IA - industrial accident

CVA - cerebral vascular accident

AVM - arteriovenous malformation

Table 2

Comparison of the Mean Peak Physiological Responses During the Three Cycle Graded Exercise Test Trials

Variable	Trial 1	Trial 2	Trial 3	%diff 1-2[c]	r 1-2	%diff 2-3[c]	r 2-3	%diff 1-3[c]	r 1-3
HR beats/min	145.1 (24.07)	148.5 (21.04)	146.7 (22.64)	2.3	0.91[a]	- 1.2	0.86[a]	1.1	0.90[a]
$\dot{V}O_2$ (A) l/min	2.11[b] (0.75)	2.18 (0.73)	2.25 (0.84)	3.2	0.97[a]	3.2	0.96[a]	6.6	0.94[a]
$\dot{V}O_2$ (R) ml/kg/min	26.4[b] (8.98)	27.1 (8.2)	28.0 (9.71)	2.5	0.97[a]	3.4	0.95[a]	5.9	0.94[a]
$\dot{V}CO_2$ l/min	2.15[b] (0.78)	2.34[b] (0.90)	2.33 (0.88)	8.8	0.96[a]	- 0.4	0.93[a]	8.4	0.93[a]
$\dot{V}_E$ l/min	65.17[b] (22.19)	72.06[b] (27.95)	70.15 (25.59)	10.6	0.96[a]	- 2.6	0.94[a]	7.6	0.87[a]
TV l/breath	1.65[b] (0.42)	1.76[b] (0.46)	1.77[b] (0.41)	6.7	0.94[a]	0.6	0.94[a]	7.3	0.94[a]
F breaths/min	39.0 (8.4)	40.3 (9.9)	38.9 (8.3)	3.4	0.87[a]	- 3.4	0.86[a]	- 0.1	0.82[a]
O_2 PULSE ml/beat	14.4 (4.3)	14.5 (4.0)	15.1 (4.5)	0.3	0.94[a]	4.3	0.92[a]	4.6	0.96[a]
% FAT	20.7 (8.3)	21.4 (8.3)	21.5 (8.7)	3.3	0.99[a]	0.8	0.99[a]	4.1	0.99[a]
RER	1.02 (0.07)	1.06 (0.09)	1.04 (0.08)	3.9	0.18 p=0.56	- 1.9	0.18 p=0.56	2.0	0.27 p=0.37
MBP mm Hg	95.8 (33.8)	93.9 (24.5)	106.9 (25.0)	- 2.1	- 0.07 p=0.83	13.9	0.23 p=0.93	11.5	0.03 p=0.83
RPE	18.8 (1.0)	18.3 (1.2)	19.2 (0.8)	- 2.2	0.24 p=0.04	5.0	0.42 p=0.18	2.7	0.34 p=0.27

r-Pearson product-moment correlation coefficients

[a]correlation coefficients significant at the 0.001 level

[b]significantly lower scores were obtained by subjects with brainstem lesions ($p \leq 0.02$)

[c]no significant trial effects

Table 3
Comparison of Intraclass Correlation Coefficients and Average Pearson's r for Selected Peak Physiological Responses (nTrials = 3)

Variable	ICC	Ave Pearson 'r'
HR	0.86	0.89
$A\dot{V}O_2$	0.94	0.96
$R\dot{V}O_2$	0.94	0.95
$\dot{V}CO_2$	0.94	0.94
$\dot{V}_E$	0.90	0.92
TV	0.92	0.94
F	0.85	0.85
O_2 PULSE	0.93	0.94
% FAT	0.99	0.99
RER	0.19	0.21 (ns)
MBP	0.05	0.06 (ns)
RPE	0.23	0.33 (ns)

Table 4
Method Error Coefficients of Variation for Selected Peak Physiological Responses

Variable	Trial 1-2	Trial 2-3	Trial 1-3	Mean A - B
HR	4.8	5.9	6.8	3.4
$A\dot{V}O_2$	6.1	8.2	9.4	4.7
$R\dot{V}O_2$	6.1	8.1	8.9	4.4
$\dot{V}CO_2$	8.3	7.8	9.9	4.8
$\dot{V}_E$	9.1	9.6	13.4	6.5
TV	6.5	6.1	6.0	3.0
F	8.6	9.0	9.2	4.5
O_2 PULSE	7.2	8.3	6.2	3.1
% FAT	4.2	3.0	4.9	2.4
RER	7.1	7.5	6.0	3.0
MBP	32.1	21.6	29.0	15.0
RPE	5.0	4.1	3.7	1.9

A = mean trials 1 and 2
B = mean trials 2 and 3

Table 5
Comparison of the Percent Difference Between Individual GXT Trials and the Mean of Consecutive Trials

Variable		Trial 1-2	Trial 2-3	Trial 1-3	Mean 1-2
HR	low	- 9.2	- 11.4	- 13.5	- 7.1
	high	+16.7	+13.1	+ 18.6	+ 9.0
$A\dot{V}O_2$	low	- 10.9	- 15.0	- 15.8	- 8.1
	high	+30.2	+21.2	+ 41.7	+ 18.2
$R\dot{V}O_2$	low	- 10.9	- 15.0	- 15.8	- 8.3
	high	+30.2	+21.2	+ 41.7	+ 18.1
$\dot{V}CO_2$	low	- 10.9	- 21.0	- 12.7	- 6.7
	high	+ 36.1	+ 19.8	+ 44.0	+ 18.4
$\dot{V}_E$	low	- 24.0	- 18.6	- 27.3	- 14.5
	high	+ 31.7	+ 30.6	+ 52.4	+ 22.6
TV	low	- 7.8	- 8.3	- 4.0	- 2.0
	high	+ 29.3	+ 25.2	+ 27.1	+ 12.7
F	low	- 28.8	- 23.7	- 24.6	- 12.9
	high	+ 17.5	+ 32.5	+ 19.9	+ 9.2
O_2 PULSE	low	- 16.1	- 8.0	- 11.9	- 6.1
	high	+ 21.0	+ 35.4	+ 19.4	+ 8.8
RER	low	- 15.9	- 25.6	- 17.3	- 9.0
	high	+22.5	+ 9.8	+ 15.8	+ 7.4

Table 6 Comparison of Mean Physiological Responses During Determination of Cardiac Output

VARIABLE	TRIAL 1a (SD)	TRIAL 1b (SD)	TRIAL 2a (SD)	TRIAL 2b (SD)	% DIFF & r*(1a-1b)	% DIFF & r*(2a-2b)	%DIFF & r*(1a-2a)	% DIFF & r*(1b-2b)
HR beats/min	115.2 (18.7)	120.5 (19.1)	114.4 (19.2)	120.5 (19.1)	4.6 (0.93)	5.4 (0.95)	- 0.7 (0.69)	0.1 (0.93)
$\dot{V}O_2$ l/min	1.19 (0.46)	1.24 (0.45)	1.19 (0.39)	1.18 (0.41)	5.9 (0.97)	- 1.3 (0.98)	0.9 (0.87)	- 5.9 (0.95)
$\dot{V}CO_2$ ml/min	1277.7 (536.2)	1301.5 (493.4)	1216.2 (430.3)	1198.5 (449.3)	4.8 (0.94)	- 2.5 (0.97)	- 2.6 (0.85)	- 9.4 (0.92)
O_2 PULSE ml/beat	10.2 (3.4)	10.18 (3.0)	10.54 (3.6)	9.78 (2.9)	0.8 (0.93)	- 6.6 (0.94)	3.8 (0.80)	- 3.9 (0.95)
$\dot{V}_E$ l/min	37.78 (14.86)	40.42 (14.39)	37.35 (10.79)	37.60 (11.61)	7.0 (0.97)	0.7 (0.98)	- 1.1 (0.88)	- 7.0 (0.90)
Q l/min	13.47 (3.76)	13.54 (3.30)	13.43 (3.65)	13.08 (3.88)	1.8 (0.89)	- 3.0 (0.93)	0.4 (0.57) (p=0.04)	- 4.4 (0.86)
QI l/min/sq.m	6.98 (1.76)	7.00 (1.41)	7.00 (1.90)	6.78 (1.92)	1.5 (0.82)	- 3.6 (0.86)	0.9 (0.42) (p=0.15)	- 4.1 (0.85)
SV ml/beat	117.9 (32.0)	112.9 (23.6)	118.3 (29.9)	108.5 (26.00)	- 3.0 (0.85)	- 7.6 (0.81)	0.9 (0.51) (p=0.08)	- 3.9 (0.85)
SVI ml/beat/sq.m	61.2 (15.8)	58.5 (10.2)	61.6 (15.0)	56.3 (12.4)	- 3.3 (0.86)	- 8.0 (0.79)	1.2 (0.45) (p=0.12)	- 3.8 (0.80)
$(a\text{-}v)o_2$ diff ml /100 ml	8.7 (1.7)	9.0 (1.7)	8.9 (1.7)	9.0 (1.5)	3.3 (0.90)	1.1 (0.76)	2.6 (0.50) (p=.058)	0.4 (0.74)
TV ml/breath	1403.6 (334.0)	1424.8 (324.9)	1367.0 (305.1)	1398.7 (393.9)	2.8 (0.95)	- 1.7 (0.95)	- 1.3 (0.74)	- 5.7 (0.79)

r-Pearson product-moment correlation coefficients

*correlations significant at the 0.01 level except where indicated

Table 7
Intraclass Correlation Coefficients and Average Pearson 'r's for Some Variables during Determination of Cardiac Output

Variable	ICC[a]	ICC[b]	Ave Pearson 'r' [a]
HR (beats/min	0.84	0.93	0.87
$A\dot{V}O_2$ (l/min)	0.93	0.94	0.94
$\dot{V}CO_2$ (ml/min)	0.91	0.90	0.92
O_2 PULSE (ml/beat)	0.88	0.94	0.90
$\dot{V}_E$ (l/min)	0.89	0.87	0.93
Q (l/min)	0.78	0.85	0.81
QI (l/min/sq.m)	0.73	0.82	0.74
SV (ml/beat)	0.68	0.84	0.76
SVI (ml/beat/sq.m)	0.61	0.78	0.72
(a-v)O_2 DIFF (ml/100 ml)	0.70	0.75	0.72
TV (ml/breath)	0.82	0.79	0.86

[a] 4 maneuvers (1a, 1b, 2a, & 2b)

[b] 2 maneuvers (1b & 2b)

Table 8
Method Error Coefficients of Variation for some Variables During Determination of Cardiac Output

VARIABLE	Trial 1a-1b	Trial 2a-2b	Trial 1a-2a	Trial 1b-2b	X - Y
HR	4.4	7.2	10.1	4.6	6.4
$A\dot{V}O_2$	6.2	4.9	13.2	8.4	10.1
$\dot{V}CO_2$	8.0	5.3	14.6	10.6	11.7
O_2 PULSE	19.4	9.1	15.1	17.6	13.1
$\dot{V}_E$	6.9	4.9	13.8	11.4	11.9
Q	8.9	7.7	18.1	10.5	13.8
QI	8.9	8.8	18.4	10.6	14.0
SV	10.5	10.8	18.2	8.8	13.5
SVI	10.4	11.2	18.7	9.2	13.4
$(a-v)O_2$ DIFF	6.2	8.8	13.3	9.1	10.1
TV	5.2	7.2	11.8	12.1	11.3

Table 9
Comparison of the Percent Difference Between Individual Maneuvers and the Means of Each Days Two Maneuvers

Variable	Range	Trial 1a-1b	Trial 2a-2b	Trial 1a-2a	Trial 1b-2b	X - Y
HR	low	- 4.4	0.0	- 30.6	- 14.5	- 22.4
	high	+ 25.7	+ 26.6	+ 31.7	+ 11.0	+ 20.2
$\dot{V}O_2$	low	- 6.7	- 16.7	- 21.3	- 19.8	- 16.8
	high	+ 31.7	+ 10.2	+ 48.5	+ 28.3	+ 23.9
$\dot{V}CO_2$	low	- 14.4	- 15.1	- 34.0	- 29.9	- 25.4
	high	+ 33.3	+ 10.4	+ 37.1	+ 17.2	+ 20.2
O_2 pulse	low	- 15.2	- 24.1	- 20.0	- 18.1	- 5.6
	high	+ 37.4	+ 4.9	+ 20.7	+ 20.7	+ 25.8
$\dot{V}_E$	low	- 9.6	- 16.1	- 29.9	- 23.3	- 26.2
	high	+ 24.4	+ 10.7	+ 32.7	+ 17.2	+ 18.8
Q	low	- 15.6	- 27.9	- 38.6	- 20.7	- 30.1
	high	+ 25.1	+ 12.7	+ 42.5	+ 19.9	+ 28.4
QI	low	- 15.6	- 35.1	- 38.5	- 20.8	- 30.1
	high	+ 24.5	+ 12.7	+ 58.2	+ 20.6	+ 36.4
SV	low	- 18.6	- 28.3	- 39.9	- 20.0	- 30.4
	high	+ 31.6	+ 10.0	+ 45.9	+ 18.2	+ 29.2
SVI	low	- 18.6	- 27.9	- 39.9	- 19.4	- 30.4
	high	+ 30.8	+ 10.3	+ 46.8	+ 18.5	+ 29.8
(a-v) O_2 diff	low	- 9.8	- 22.9	- 27.1	- 27.4	- 27.2
	high	+ 28.2	+ 42.4	+ 66.8	+ 25.1	+ 36.0
TV	low	- 10.2	- 7.0	- 23.6	- 21.8	- 15.6
	high	+ 12.6	+ 23.4	+ 26.6	+ 39.9	+ 30.7

X = mean of maneuvers 1a & 1b, Y = mean of maneuvers 2a & 2b

Table 10
Comparison of Variables that showed Significant Differences Between Subjects With or Without Indications of Brainstem Injury

Variable	brainstem injury	Trial 1		Trial 2		Trial 3	
		mean	SD	mean	SD	mean	SD
$\dot{V}O2$	no	2.30	(0.73)	2.26	(0.75)	2.33	(0.84)
l/min	yes	1.80[a]	(0.75)	2.03	(0.76)	2.11	(0.92)
$\dot{V}O2$	no	28.3	(7.4)	27.6	(7.2)	28.5	(8.5)
ml/kg/min	yes	23.4[a]	(11.3)	26.1	(10.4)	27.1	(12.4)
$\dot{V}CO2$	no	2.39	(0.76)	2.54	(0.95)	2.46	(0.81)
l/min	yes	1.76[a]	(0.73)	2.02[a]	(0.81)	2.12	(1.03)
$\dot{V}_E$	no	73.28	(22.54)	78.79	(30.74)	73.82	(24.57)
l/min	yes	52.20[a]	(18.57)	61.30[a]	(21.34)	64.28	(28.94)
TV	no	1.80	(0.43)	1.94	(0.45)	1.87	(0.42)
l/min	yes	1.42[a]	(0.42)	1.47[b]	(0.32)	1.60[a]	(0.39)

[a] significantly lower for subjects with brainstem injury

Table 11

Reliability of Measures in Subjects with Brain Stem Injury

Graded exercise tests

Variable	r Trials 1-2	r Trials 2-3	r Trials 1- 3
HR	0.99	0.87	0.91
$A\dot{V}O_2$	0.98	0.99	0.96
$R\dot{V}O_2$	0.99	0.99	0.97

Cardiac output maneuvers

Variable	r Maneuver 1a-1b	r Maneuver 2a-2b	r Maneuver 1a-2a	r Maneuver 1b-2b
Cardiac output (Q)	0.94	0.98	0.76	0.91
Stroke volume (SV)	0.94	0.84	0.57	0.84
$(a\text{-}v)O_2$ diff	0.99	0.96	0.98	0.99

r = Pearson product-moment correlation coefficients

REFERENCES

Annegers, J., Grabow, J., Kurland, L., & Laws, E. Jr. (1980). The incidence, causes, and secular trends of head trauma in Olmsted County, Minneapolis, Minnesota. Neurology, 30, 912-19.

Astrand, P-O. & Rodahl, K. (1986). Textbook of work physiology (3rd ed.). Toronto: McGraw-Hill Book Company.

Bartko, J.J. (1976). On various intraclass correlation reliability coefficients. Psychological Bulletin, 5, 762-65.

Becker, E., Bar-Or, O., Mendelson, L., & Najenson, T. (1978). Pulmonary functions and responses to exercise of patients following cranio cerebral injury. Scandinavian Journal of Rehabilitation Medicine, 10, 47-50.

Bhambhani, Y.N., Eriksson, P., & Steadward, R.D. (1991). Reliability of peak physiological responses during wheelchair ergometry in persons with spinal cord injury. Archives of Physical Medicine and Rehabilitation, 72, 559-562.

Bhambhani, Y.N., Holland, L.J., & Steadward, R.D. (1993). Anaerobic threshold in wheelchair athletes with cerebral palsy: validity and reliability. Archives of Physical Medicine and Rehabilitation, 74, 305-11.

Bhambhani, Y.N., Norris, S., & Bell, G. (1994). Prediction of stroke volume from oxygen pulse measurements in untrained and trained men. Canadian Journal of Applied Physiology, 19, 49-59.

Borg, G. (1970). Perceived exertion as an indicator of somatic stress. Scandinavian Journal of Rehabilitation Medicine, 2, 92-98.

Borg, G. (1982). Psychophysical bases of perceived exertion. Medicine and Science in Sports and Exercise, 14, 377-381.

Bray, L.S., Carlson, F., Humphrey, R., Mastrilli, J.P., & Valko, A.S. (1987). Physical rehabilitation. In M.Ylvisaker & E.M.R. Gobble (Eds.). Community Re-entry for Head Injured Adults (pp. 25-85). Boston: Little, Brown and Company.

Caveness, W.F. (1979). Incidence of craniocerebral trauma in the United States in 1976 with trend from 1970 to 1975. Advances in Neurology, 22, 1-3.

Cohadon, F. (1981). The importance of rehabilitation programs in the prevention and alleviation of head injury sequelae. Progress in Neurological Surgery, 10, 344-384.

Collier, C.R. (1956). Determination of mixed venous CO_2 tensions by rebreathing. Journal of Applied Physiology, 9, 25-29.

DuBois, D. & DuBois, E.F. (1916). A formula to estimate the approximate surface area if height and weight be known. Archives of Internal Medicine, 17, 863-71.

Farber, S.D. (1982). Neurorehabilitation: A Multisensory Approach. Philadelphia: W. B. Saunders Company.

Fernhall, B., Miller, A.L., Tymeson, G.T., & Burkett, L.N. (1990). Maximal exercise testing of mentally retarded adolescents and adults: reliability study. Archives of Physical Medicine and Rehabilitation, 71, 1065-68.

Finlayson, M.A.J. & Garner, S.H. (1994). Brain Injury Rehabilitation: Clinical Considerations. Philadelphia: Williams & Wilkins.

Frankowski, R.F. (1986). Trauma treatment in practice: the head injured patient. Advances in Psychosomatic Medicine, 16, 153-72.

Giles, G.M. (1994). Functional assessment and intervention. In M. A. J. Finlayson & S. H. Garner (Eds.), Brain Injury Rehabilitation: Clinical Considerations, (pp. 124-56).. Philadelphia: Williams & Wilkins.

Gilroy J. & Holliday, P.L. (1982). Basic Neurology. New York: MacMillan Publishing Co., Inc.

Henson, M.B., De Castro, J.M., Stringer, A.Y., & Johnson, C. (1993). Food intake by brain-injured humans who are in the chronic phase of recovery. Brain Injury, 7, 169-178.

Hermansen, L. & Saltin, B. (1969). Oxygen uptake during maximal treadmill and bicycle exercise. Journal of Applied Physiology, 32, 467-73.

Howard, R.A., Huijbregts, M.P.J., & Miller, P.A. (1988). A walking program to re-train cardiorespiratory fitness after closed-head injury in young adults. Physiotherapy Canada, 8 (Suppl. 2), 4.

Hunter, M., Tomberlin, J., Kirkikis, C., & Kuna, S. (1990). Progressive exercise testing in closed head-injured subjects: comparison of exercise apparatus in assessment of a physical conditioning program. Physical Therapy, 70, 363-371.

Jankowski, L.W. & Sullivan, S.J. (1990). Aerobic and neuromuscular training: effects on the capacity, efficiency, and fatigability of patients with traumatic brain injuries. Archives of Physical Medicine and Rehabilitation, 71, 500-504.

Jennet, B. (1976). Assessment of severity of head injury. Journal of Neurology, Neurosurgery and Psychiatry, 39, 647-655.

Jennet, B., Snoak, J., Bond, M., & Brooks, N. (1981). Observations on the use of the Glasgow Outcome Scale. Journal of Neurology, Neurosurgery and Psychiatry, 44, 285-293.

Jennet, B. & Teasdale, G. (1981). Management of Head Injuries. Philadelphia: F. A. Davis Company.

Jones, N.L. (1988). Clinical Exercise Testing. Philadelphia: W. B. Saunders Company

Kalsbeek, W.D., McLaurin, R.L., Harris, B.S. & Miller, J.D. (1980). The National Head and Spinal Cord Injury Survey: major findings. Journal of Neurosurgery, 53, S19-S31.

Levin, H., Benton, A., & Grossman, R. (1982). Neurobiological Consequences of Closed Head Injury. Oxford: Oxford University Press.

Lindsley, D.F. & Holmes, J.E. (1984). Basic Human Neurophysiology. Amsterdam: Elsevier Science Publishing Co., Inc.

Marks, C., Katch, V., Rocchini, A., Beekman, R., & Rosenthal, A. (1985). Validity and reliability of cardiac output by CO_2 rebreathing. Sports Medicine, 2, 432-46.

McArdle, W.D. Katch, F.I., & Katch, V.L. (1991). Exercise Physiology: Energy, Nutrition, and Human Performance. Philadelphia: Lea & Febiger.

Moran, A.J. (1976). Six cases of severe head injury treated by exercise in addition to other therapies. Medical Journal of Australia, 1, 396-397.

Novack, T.A., Roth, D.L., & Boll, T.J. (1988). Treatment alternatives following mild head injury. Rehabilitation Counselling Bulletin, 31, 313-24.

Oddy, M., Coughlan, T., Tyerman, A., & Jenkins, D. (1985). Social adjustment after closed head injury: a further follow-up seven years after injury. Journal of Neurology, Neurosurgery, and Psychiatry, 48, 564-68.

Oddy, M., Humphrey, M., & Uttley, D. (1978). Subjective impairment and social recovery after closed head injury. Journal of Neurology, Neurosurgery and Psychiatry, 41, 611-16.

Ottenbacher, K.J. (1991). Statistical conclusion validity: multiple inferences in rehabilitation research. American Journal of Physical Medicine and Rehabilitation, 70, 317-22.

Pagano, R R. (1990). Understanding Statistics in The Behavioral Sciences (3rd ed). St. Paul, MN: West Publishing Company.

Patterson, R.P., Pearson, J., & Fisher, S.V. (1985). Work-rest periods: their effect on normal physiologic response to isometric and dynamic work. Archives of Physical Medicine and Rehabilitation, 66, 348-52.

Perry, J. (1983). Rehabilitation of the neurologically disabled patient: principles, practice and scientific basis. Journal of Neurosurgery, 58, 799-816.

Prigatano, G. (1986). Neuropsychological rehabilitation after brain injury. Baltimore: John Hopkins University Press Ltd.

Prigatano, G.P. & Altman, I.M. (1990). Impaired awareness of behavioral limitations after traumatic brain injury. Archives of Physical Medicine and Rehabilitation, 71, 1058-64.

Sale, D.G. (1992). Testing strength and power. In J.D. MacDougall., H.A. Wenger, & H.J. Green (Eds.), Physiological Testing of the High-Performance Athlete (2nd. ed.). (pp. 21-106).

Saltin, B., Blomqvist, G., Mitchell, J.H., Johnson Jr., R.L. Wildenthal, K., & Chapman, C.B. (1968). Response to exercise after bedrest and training. Circulation, Supplement VII, 1-78.

Segal, K.S., Gutin, B., Presta, E., Wang, J., & Van Itallie, T.B. (1985). Estimation of human body composition by electrical impedance methods: a comparative study. Journal of Applied Physiology, 58, 1565-71.

Siri, W.E. (1956). Gross composition of the body. In J.H. Lawrence & C.A. Tobias (Eds.), Advances in Biological and Medical Physics, (volume 4), New York: Academic Press.

Smigielski, J.S., Malec, J., Thompson, J.M., & DePompolo, R.W. (1992). Mayo Medical Center brain injury outpatient program: treatment procedures and early outcome. Mayo Clinic Proceedings, 67, 767-774.

SPSS, Inc. (1988). SPSS-X (Computer software, VAX/VMS release 3.0). Chicago: Author

Statistica (1991). (Statistics Software, release 3.1). Tulsa: Author

Sullivan, S.J. Richer, E., & Laurent, F. (1990). The role of and possibilities for physical conditioning programmes in the rehabilitation of traumatically brain-injured persons. Brain Injury, 4, 407-14.

Thodan, J.S. (1991). Testing aerobic power. In J.D. MacDougall., H. A. Wenger, & H.J. Green (Eds.), Physiological Testing of the High-Performance Athlete (2nd. ed.). (pp. 107-173).

Twyman, D.L. & Liedtke, R.J. (1987). Bioelectrical impedance analysis of body composition. Bioelectrical Impedance User's Manual. R. J. L. Systems, Inc. Author.

Wilmore, H., Farrell, P. A., Norton, A. C., Cote III, R W., Coyle, E.F., Ewy, G.A., Temkin, L.P., & Billing, J.E. (1982). An automated, indirect assessment of cardiac output during rest and exercise. Journal of Applied Physiology, 52, 1493-97.

Wu, Y-T., Nielson, D.H., Cassady, S.L., Cook, J.S., Janz, K.F., & Hansen, J.R. (1990). Cross-validation of bioelectrical impedance analysis of body composition in children and adolescents. Physical Therapy, 73, 320-328.

Ylvisaker, M. & Gobble, E.M.R. (1987). Community Re-Entry for Head Injured Adults. Boston: Little, Brown and Company.

Suppliers

a) Corival 400, Quinton Instruments Co., 2121 Terry Avenue, Seattle, Wa. 98121.

b) Model MMC Horizon, Sensormedics, 1630 South State College Boulevard, Anaheim, Ca. 98206.

c) Model 1500B, Hewlett Packard, 6877 Coreway Drive, Mississauga, Ontario, Canada L4V-1M8.

d) Sports Tester, Model PE3000, Potential Training Products Co. Inc., 2410 Speers Road, Oakville, Ontario L6L 5M2.

e) Blook Pressure Monitor, Model 9350, Paramed Technology Inc. 510 Logue Ave., Mountain View, CA. 94043.

f) R. J. L. Body Composition Analyzer BIA-101, R. J. L. Systems Inc., 9930 Whittier, Detroit, MI. 48224.

University of Alberta

Part B

Aerobic Exercise Training Effects in Adults with Traumatic Brain Injury

by

Gary James Rowland

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Science

Department of Occupational Therapy

Edmonton, Alberta

Spring, 1996

Abstract

The purpose of this study was to examine the changes in peak and submaximum physiological responses to exercise in subjects with severe to moderate brain injury following aerobic training. Eleven subjects with brain injuries were recruited from a rehabilitation program that specialized in providing treatment for this population. Subjects completed three identical incremental tests to volitional fatigue and two submaximal cardiac output protocols on a cycle ergometer in order to establish initial levels of cardiorespiratory fitness. Subjects were then matched for level of fitness, gender, and functional abilities. From pairs of cohorts, individuals were randomly assigned to either an experimental or control group. Subjects in the control group performed their regular rehabilitation program activities. Subjects in the experimental group continued with their regular rehabilitation and in addition, were prescribed aerobic fitness training three times per week for 10 to 12 weeks, at a minimum intensity equivalent to 50 % peak oxygen. Training intensity was monitored using wireless heart rate monitors.

Following training, the subjects performed the same incremental cycle test twice and the submaximal cardiac output protocol once. In all, subjects completed five identical incremental cycle tests and three cardiac output maneuver protocols. Individual test sessions were separated by a minimum of two days and a maximum of one week. An automated metabolic cart, electrocardiograph and wireless heartrate monitor were used to measure peak and submaximal physiological responses to exercise. An estimate of body composition was obtained using bioelectrical impedance analysis prior to and following the training period.

The exercise group subjects demonstrated a significant improvement (+ 20 bpm, $p<0.002$) in peak heart rate following the training program. Increases in O_2 uptake (+ 5 ml/kg/min), minute ventilation (+9 liters/min) and power output (+ 30 watts) approached statistical significance. When examined as the percent difference between pre- and post-

was obtained using bioelectrical impedance analysis prior to and following the training period.

The exercise group subjects demonstrated a significant improvement (+ 20 bpm, $p<0.002$) in peak heart rate following the training program. Increases in O_2 uptake (+ 5 ml/kg/min), minute ventilation (+9 liters/min) and power output (+ 30 watts) approached statistical significance. When examined as the percent difference between pre- and post-training mean values, improvement in peak oxygen uptake was significantly higher in the exercise group. There were no significant differences between pre- and post-training values for cardiac output and stroke volume during submaximal exercise. However, stroke volume at an intensity of 50 % of peak oxygen uptake increased in the exercise group (+9 ml/beat, 7.2 %) and decreased (-1.3 ml/beat, -1.2 %) in the control group, suggesting that the exercise program had a positive effect on the myocardium. There was no significant change in body weight or composition for either group.

The results of this study support previous reports that adults with traumatic brain injury can improve their level of cardiorespiratory fitness through aerobic exercise training. The decline in cardiorespiratory fitness observed in the control group, emphasizes the importance of including aerobic activities in rehabilitation and discharge planning.

Part B.

Aerobic Exercise Training Effects in Adults with Traumatic Brain Injury

INTRODUCTION

The annual occurrence of traumatic brain injury (TBI) in the United States in 1974 has been estimated at 0.2 % of the population (Kalsbeek, McLaurin, Harris, & Miller, 1980). From 1974 to 1984, the incidence of TBI, defined as including evidence of intracranial injury, loss of consciousness, skull fracture or posttraumatic amnesia, was estimated at between 0.2 % and 0.3 % (Frankowski, 1986). This translates to an estimation that annually over 400,000 Americans sustain some type of TBI requiring hospitalization (Frankowski, 1986). It has been estimated that approximately 44,000 of these cases fall into the moderately to severely impaired range (Kalisky, Morrison, Meyers, & Von Lufan, 1985). Data from Statistics Canada for 1988-1989, indicates that approximately 40,000 patients (incidence rate of approximately 0.2 %) are discharged from hospitals with a diagnosis of skull fracture or intracranial injury (Wong, Dornan, Schebtag, Ip, & Keating, 1993). In Alberta between 1979 and 1989, there were 46,080 hospitalizations with diagnoses including head injury, suggesting that annually between 4,000 and 5,000 Albertans sustain TBI (Helledie & Shane, 1992).

To put these numbers for TBI into perspective, an annual incidence rate of 0.2 % is 40 times greater than the annual incidence rate for spinal cord injury (0.005 %) determined by Kalsbeek et al. (1980). Advances in life saving medical techniques are resulting in the survival of increasing numbers of seriously brain injured patients, many of whom have the potential to live a normal life span (Smigielski, Malec, Thompson, & DePompolo, 1992). As a result, the population of individuals with brain injury is growing. The annual cost of health care for acquired brain injury and spinal cord injury in the United States is approximately $4 billion and has been increasing at the rate of 15 % per year (Cope & O'Lear, 1993). Costs for

inpatient rehabilitation programs can vary from $700 to $1500 per day (Cope & O'Lear, 1993).

Many individuals with moderate to severe TBI demonstrate a low tolerance for physical activities. Three subjects in the study by Jankowski and Sullivan (1990) obtained post-training peak relative oxygen consumption ($R\dot{V}O_2$) levels of 24.7, 25.7 and 27.0 ml/kg/min. These values are so low that these individuals would be working at 33 % to 35 % of their physical work capacity when engaged in self-care (dressing, grooming) or light household activities (food preparation, sweeping floors, dusting). These activities are rated at 2.5 METs which is equal to 8.75 ml O_2/kg/min (Ainsworth et al., 1993). A fourth subject, with a peak $R\dot{V}O_2$ of 15.4 ml O_2/kg/min would be taxed at 57 % of his physical work capacity while engaged in these activities. Seven of the 13 subjects in the study by Rowland (1995) had values for peak $R\dot{V}O_2$, ranging from 15.3 to 25.9 ml/kg/min and would also be severely taxed by activities taken for granted by healthy individuals.

When permitted, people performing manual labour for extended periods, will set the pace of the work and seldom accept working above 40 % of their physical work capacity (Astrand & Rodahl, 1986). Therefore, it is not surprising that many individuals with TBI do not voluntarily engage in more strenuous activities, such as work or exercise programs, as the O_2 demands for such activities would severely tax their cardiorespiratory system. For example, each of the following activities are rated at 5.0 METs (17.5 ml O_2/kg/min),

a) a work role involving lawn maintenance
b) a work role involving lifting and carrying objects weighing between 25 and 49 pounds, such as warehousing, or
c) performing light calisthenics (Ainsworth et al., 1993).

The four subjects in the study by Jankowski and Sullivan (1990) with low post-exercise oxygen consumption would be taxed at the rates of 70.1 %, 68.1 %, 64.8 % and 113.6 % of their physical work capacity ($\dot{V}O_2$ max) performing these types of activities. Those in the

study by Rowland (1995) would be working between 67.6 % and 87.9 % of their maximum physical work capacity.

Lack of energy is a frequently reported and enduring symptom of even mild TBI (Novak, Roth, & Boll, 1988) and is often severe enough to impair work performance (Giles, 1994). Being 'easily tired' was reported as a symptom by 40 % of relatives of persons with TBI at six months (Oddy, Humphrey, & Uttley, 1978) and seven years (Oddy, Coughlan, Tyerman, & Jenkins, 1985).

Many factors can contribute to the low level of aerobic fitness often found in persons with TBI. Brain injured subjects, who were tested 20 days to 5.5 years post injury, displayed a 25 % to 40 % reduction in total lung capacity and forced expiratory volume when compared to healthy control subjects (Becker, Bar-Or, Mendelson, & Najensen, 1978). Brain injured subjects in this study also had a lower oxygen pulse ($\dot{V}O_2$/HR) and higher ventilatory equivalent for oxygen ($\dot{V}_E/\dot{V}O_2$) during exercise, suggesting lower levels of cardiorespiratory fitness. Weakness in the respiratory muscles and reduced compliance of the rib cage in persons with TBI were suggested as contributing factors to these lower values (Becker et al., 1978).

Recovery from injuries to the central nervous system, internal organs and musculoskeletal system can result in a marked degree of deconditioning. Healthy young adults confined to bedrest for a 20 day period, demonstrated an average reduction in their $\dot{V}O_2$ max of 28 % (Saltin et al., 1968). In a study of Canadian hospitalizations for head injuries during the census period covering 1988 - 1989, 91.6 % of the individuals suffered loss of consciousness. Duration of coma averaged 21.9 days and ranged from 1 to 303 days (Wong et al., 1993). It is estimated that more than 40,000 patients in the United States are hospitalized for at least three weeks as a result of head injuries (Kalsbeek et al., 1980). Individuals with TBI whose injuries result in prolonged loss of consciousness and a lengthy

period of limited activity during their recovery will experience muscle weakness and cardiopulmonary deconditioning that can hinder rehabilitation efforts (Perry, 1983).

Cognitive and behavioral deficits also contribute to the adoption of a sedentary lifestyle in persons with TBI. Deficits such as amotivation, adynamia, poor-self awareness and insight into their functional limitations and abilities (Levin, Benton, & Grossman, 1982; Prigatano, 1986; Prigatano & Altman, 1990; Varney & Menefee, 1993) likely contribute to the person with TBI adopting a sedentary lifestyle. Psychiatric sequelae including depression and anxiety are common in persons with TBI (Rosenthal & Bond, 1990) and may also lead to a reduction in activity level.

Brain damage can affect innervation to muscles in the trunk and limbs producing impairments in postural control, voluntary movements including gait, sensation, tone/spasticity, range of motion, strength, coordination, control of involuntary movements and level of fitness (Gowland & Cambarotto, 1994). Any of these impairments, alone or in combination with others, could limit the range of activities that can be performed. Paralysis or paresis of muscles will result in a decrease in the muscle mass available to perform exercise. Paralysis or paresis can also affect the respiratory muscles.

Brain lesions can affect the cardiovascular and respiratory centers located in the medulla oblongata region of the brainstem as a result of physical damage to these centers or disruption of afferent and efferent innervation (Astrand & Rodahl, 1986; Lindsley & Holmes, 1984; McArdle, Katch & Katch, 1991). Brain damage that affects the cardiovascular and respiratory centers may affect heart rate, redistribution of blood flow to support exercising muscles, and rate and depth of breathing. Any of these changes may result in a diminished cardiorespiratory response to exercise.

Living a sedentary lifestyle, in and of itself, can contribute to secondary health issues such as loss of lean body mass, increased obesity, decreased muscle strength, decreased range of motion in the trunk, neck and limbs, further decreases in respiratory status and

increased risk for cardiovascular disease and stroke (Patterson, Pearson, & Fisher, 1985). A decrease in physical activity can reduce the stresses on the skeletal system to the point that normal mineralization of the bones is reversed, leaving the person at risk for fractures (Astrand & Rodahl, 1986). An impairment in food intake regulation was observed in a recent study in which subjects with TBI ate larger meals and consumed 14 % more calories per day than control subjects (Henson, De Castro, Stringers, & Johnson, 1993). An imbalance in the amount of calories consumed and expended by metabolic processes will affect body weight and composition.

It has been demonstrated in healthy sedentary subjects that regular participation in a vigorous exercise program results in improvement in the physiological responses to exercise (Astrand, 1987; Astrand & Rodahl, 1986; McArdle, Katch & Katch, 1991; Whipp & Wasserman, 1991). The capability to improve aerobic fitness has been reported in several populations with disabilities, including physical disabilities (Santiago, Coyle, & Kinney, 1993), chronic pain (Davis, Filligim, Doleys, & Davis, 1992), cerebral palsy (Cooper & Sherill, 1986), mental retardation (Fernhall, 1993; Fernhall, Miller, Tymeson & Burkett, 1990; Miller, Fernhall, & Burkett, 1993; Rintala, Dunn, McCubbin, & Quinn, 1992), multiple sclerosis (Ponichter-Mulcare, 1993) and spinal cord injury (Davis, 1991, 1993; Figoni, 1993; Hjeltnes, 1982).

A review of the literature suggests that individuals with TBI can improve their cardiorespiratory responses to exercise with training. It has been reported that subjects in a comprehensive rehabilitation program which included 10 weeks of exercise training demonstrated a significant improvement in oxygen consumption [ml/kg/min] (Scherzer, 1986; Sullivan, Richer and Laurent, 1990). Details regarding the nature of the exercise program and how the change in O_2 uptake was measured were not provided. Sullivan, Richer and Laurent (1990) also describe the physical rehabilitation component of a specialized rehabilitation center for persons with TBI, where physical activity programs

occupied 50 % of the patient's rehabilitation time and resulted in improvement in ambulatory status and endurance. Cohadon (1981) the founder of the program described above by Sullivan, Richer and Laurent (1990) has suggested that participation in physical activities can contribute to recovery in brain injured individuals' confidence in their physical abilities that can lead to a more active lifestyle and willingness to perform difficult work or leisure tasks.

Two studies have reported that individuals with TBI can improve their aerobic fitness by participating in vigorous exercise programs (Hunter, Tomberlin, Kirkikis, & Kuna, 1990; Jankowski & Sullivan, 1990). This finding has potential significance for brain injury rehabilitation programs. However several weaknesses in the design of these two studies need to be accounted for. Both studies (Hunter et al., 1990; Jankowski & Sullivan, 1990) had small samples (12 & 13, respectively) in which subjects served as their own controls. In each study, the results of a single incremental exercise test prior to and following training were used to evaluate changes in fitness. The use of one incremental test to establish the level of fitness of subjects with TBI is suspect, given the possibility of changes in the subject's comprehension of test requirements and their ability to perform the test (practice effects), motivation, and overall wellness between the two tests. Test-retest reliability of physiological responses to exercise had not been established for persons with TBI at the time these studies were performed. The reliability of key variables related to cardiorespiratory and physical fitness has only recently been established for cycle exercise in this population (Rowland, 1995).

In both studies (Hunter et al., 1990; Jankowski & Sullivan, 1990) there was a wide range in the change in oxygen consumption following the training program. The percent difference between the pre-training and post-training values in these studies ranged from 1.2 % to 42.1 % (Hunter et al., 1990) and from - 1.6 % to 36.2 % (Jankowski & Sullivan, 1990). Almost 60 % of the subjects (7/12) in the study by Hunter et al. (1990) and 40 % of the subjects (5/14) in the Jankowski and Sullivan study (1990) demonstrated improvement in

peak $\dot{V}O_2$ that was less than 10 %. Of these subjects, two in the first study and five in the second, showed less than 5 % improvement in peak $\dot{V}O_2$. Improvement in peak $\dot{V}O_2$ exceeding 30 % was reported in two subjects in each of these studies. The exercise program in both of these studies (Hunter, et al., 1990; Jankowski & Sullivan, 1990) appeared to meet the requirements recommended by Astrand (1987) with respect to frequency, duration and intensity. A 15 % to 20 % improvement in oxygen uptake can be achieved in healthy sedentary adults who meet these training recommendations (Astrand, 1987). However, increases in $\dot{V}O_2$ exceeding 30 % are usually associated with a large total body mass and fat weight loss in individuals who begin at a very low level of fitness (ACSM, 1990).

Although the authors (Hunter et al. ,1990; Jankowski & Sullivan, 1990) attributed the improvement in peak $\dot{V}O_2$ to participation in regular exercise, they did not provide enough information to determine the reasons for the large variability in the outcome of their training programs. Because of the design of their studies, one can not rule out that, in addition to the concerns discussed above, maturation effects and chance could also make significant contributions to any change in aerobic fitness between tests.

Given the incidence and increasing prevalence of TBI and the high costs of rehabilitation and other services for this population, research that contributes to a better understanding of the effects of regular exercise on cardiorespiratory fitness is valuable. Better awareness and understanding of the aerobic fitness of patients will contribute to decisions regarding rehabilitation program planning and utilization of both physical and human resources in order to maximize the benefits to participants. Knowledge about an individual's level of aerobic fitness can and should be considered in discharge planning with respect to obtaining the necessary support services and linking the person up with appropriate community agencies and services.

In order to address the need for a better understanding about the relationship between exercise and cardiorespiratory fitness, the primary purpose of this study was to

examine the effects of aerobic training on various physiological responses at peak and submaximum exercise intensities in individuals with moderate to severe TBI. Changes in body composition were also examined. Because of the importance of exercise intensity to the outcome of exercise programs, the validity of the wireless heart rate monitors used to monitor training heart rates was also examined.

METHODS

Subject Selection

Informed written consent to participate in this study was provided by 13 subjects with diagnosed moderate to severe TBI. When appropriate, guardians for some participants signed the consent form, acknowledging the individual's understanding and willingness to participate in this study. Subjects, nine males and four females, were recruited from a tertiary level rehabilitation program specializing in the treatment of brain injury. In order to be eligible for this study, subjects had to demonstrate:

1) the presence of a moderate to severe brain injury (Jennet, Snoak, Bond, & Brooke, 1982; Jennet & Teasdale, 1981),
2) the absence of cardiorespiratory disease,
3) a stable medical condition,
4) the ability to perform exercise on a cycle ergometer, and
5) the ability to functionally communicate.

The medical staff at the rehabilitation facility were consulted on each subject, before the selection process was completed. The procedures for selecting and testing of subjects was approved by the ethics committees of Alberta Hospital Ponoka and the Department of Occupational Therapy, University of Alberta.

After completing the pre-training series of tests to determine peak physiological and submaximal responses to cycle exercise, subjects were matched for gender, age, level of peak

absolute oxygen uptake ($pA\dot{V}O_2$) and presence or absence of brain stem injury. From six pairs of subjects, one individual was randomly selected to participate in an exercise program. The remaining member of each pair and the 13th subject were placed in the control group. Two subjects, one from each of the groups, completed the initial testing but were discharged from the rehabilitation center before the research project could be completed. A second member of the control group was also discharged from the facility but returned for follow-up testing. This individual had been employed as a laborer by a local farm and construction materials supply company since participating in the reliability study. Each group consisted of two females and three males. Pertinent characteristics of the ten subjects who remained in the rehabilitation program and completed pre- and post-training tests are provided in table 12.

Exercise Program

Subjects allocated to the exercise group were scheduled to participate in a one hour long program of aerobic and light resistance exercises three times per week for a period of 12 weeks. The exercise program was to include a warm-up period of stretches, 10 minutes of arm cycling on a Saratoga arm ergometer, 10 minutes of cycling on an exercise bike, 9 minutes of resistance exercises for the upper and lower limbs using a Hydra-fitness gym, 10 minutes of stair stepping or fast walking/jogging and a cool-down period. A schedule of the exercise program is shown in Appendix 9. Exercise equipment was arranged so that the exercises could be performed in a circuit, with each participant able to start at any of the four stations and then continue around the circuit until all exercises had been completed. However, subjects spent little time warming up and cooling down, as these activities were frequently constrained by a lack of time. This occurred because subjects frequently arrived late. Occasionally, subjects had to be sought out and verbally encouraged to attend the exercise session.

To ensure that subjects exercised at an intensity which would promote training effects, they were requested to maintain a minimum exercise HR equal to that obtained when they were exercising at 50 % of their pVO_2 (mean of the $pA\dot{V}O_2$s determined in the three graded exercise tests). An exercise intensity of 50 % of $\dot{V}O_2$ max represents a minimum intensity for improving aerobic fitness in healthy adults (ACSM, 1990; Astrand, 1987). Exercise intensity was monitored by the investigator with the assistance of one other rehabilitation program staff member using wireless monitors (Sports tester, Model PE3000[a]). HRs were recorded at rest and then after five minutes and ten minutes of arm and leg cycle exercise and stair stepping. HRs were also recorded after three minutes of each of the three exercises on the Hydra-fitness gym. Subjects indicated their perceived level of exertion on the Borg Rating of Perceived Exertion (RPE) scale (Borg, 1979, 1982) at the same time that HRs were recorded. Resistance on the exercise bike, arm ergometer and Hydra-fitness gym was individually adjusted to maintain the subject's exercising HR at or above the prescribed training level. Cycling speed on the exercise bike and arm cycle was maintained at approximately 50 rpm. Resistance on the Hydra-fitness gym was initially set at a level that each subject experienced as "13 -somewhat hard" using the Borg (1970, 1982) RPE scale. Resistance and or rate was then adjusted to maintain the desired training intensity. Subjects performed as many repetitions of each of the three exercises as they could in three minutes. Wrist weights were added for some subjects during stair stepping in order to increase their HRs to the desired training intensity. As time passed, subjects were encouraged to work at a higher intensity if they could comfortably do so during the exercise program.

In order to share three similar heart rate monitors and accommodate individual therapy schedules, subjects in the exercise group were divided into two groups. One group was scheduled to exercise in the morning and the other in the afternoon. The number of training sessions attended, initial and final mean training HRs, percent of maximum HR and percent of age-adjusted HR (220-subject's age) are presented in table 13.

Overview of Test Protocol

Prior to the commencement of the aerobic fitness training program for the experimental group, subjects completed three identical incremental exercise tests to volitional fatigue and two protocols to determine their cardiac output (Q) and stroke volume (SV) at submaximal exercise intensities. High test-retest reliability of these protocols was established using the data from these pre-training trials (Rowland, 1995). Immediately before each incremental or graded exercise test (GXT), each subject's height and weight were recorded. Also at this time, an estimation of each subject's body composition was obtained using a bioelectrical impedance analyzer (BIA)[b].

For selected variables, the mean values from the three GXTs and the two Q protocols (four maneuvers in total) were calculated and used as the pre-training measures of cardiorespiratory fitness. After a 12 week period of exercise training, subjects completed two additional GXTs and one more submaximal Q protocol. The mean post-training values for the peak physiological responses were calculated from the two GXTs. Post-training values for submaximal physiological responses were obtained from the two Q maneuvers performed at 50 % of the post-training p$\dot{V}$O2. In all, each subject completed five identical symptom-limited GXT's to volitional fatigue and three protocols to determine their Q. During both the pre-exercise and post-exercise testing, the period between test sessions was at least two days but no more than one week.

Peak physiological Responses

The GXTs were performed on an electronically braked uniwork bicycle ergometer[c]. The incremental protocols were initiated with the subjects pedaling at 60 rpm and zero load. Every two minutes, the resistance was increased by 25 watts for the male subjects and 15 watts for the female subjects until volitional fatigue was reached. All subjects were verbally encouraged to exert their best effort.

Metabolic and cardiorespiratory measurements were continuously monitored using an automated metabolic cart[d], an electocardiograph[e] with bipolar leads in the CM_5 position, and a wireless heart rate monitor[a]. A description of the metabolic cart and the procedure to obtain a calculation of absolute oxygen consumption ($A\dot{V}O_2$, l/min) and carbon dioxide production ($\dot{V}CO_2$, l/min) using the Haldane transformation has been previously described by Bhambhani, Eriksson and Steadward (1991). Metabolic measurements were averaged over 30 seconds. Heart rate (HR) was recorded from the wireless monitor during the last 15 seconds of each minute of exercise. As well, the subjects indicated their level of perceived exertion using the Borg scale (Borg, 1970, 1982) during the last 15 seconds at each power output. The highest value for absolute oxygen consumption ($A\dot{V}O_2$, l/min) while the subject was still able to pedal was considered to be the peak (p) level of oxygen consumption. The value of other cardiorespiratory variables reported correspond to the time that $pA\dot{V}O_2$ occurred.

Submaximal Physiological Responses

The protocol for determining submaximal responses to exercise has been described previously by Rowland (1995). Cardiac output was determined using the automated CO_2 rebreathing procedure (Wilmore, et al., 1982). The same equipment used in the graded exercise tests(cycle ergometer[c], metabolic cart[d], electrocardiograph[e]) was employed. The procedures used to obtain an estimate of Q and SV has been previously described by Bhambahani, Norris and Bell (1994). Cardiac index (QI) and stroke volume index (SVI) were calculated by dividing the Q and SV by the subject's body surface area. Total body surface area (BSA) was calculated using the following formula;

$$BSA=[(weight)^{0.425} \times (height)^{0.725} \times 71.84]$$ (DuBois & DuBois, 1916).

Each protocol for determination of Q consisted of two maneuvers performed at each of three exercise intensities(at rest, zero load, & 50 % of $pA\dot{V}O_2$). As maximum SV is

achieved between 40 % and 50 % of maximal $\dot{V}O_2$ (Astrand & Rodahl, 1986; McArdle, Katch, & Katch, 1991), the value for SV obtained at 50 % of each subject's $pA\dot{V}O_2$ was assumed to represent their peak SV. Although the inclusion of six Q maneuvers in each of the two pre-training test sessions was thought to tax the endurance of subjects and contribute to within subject variability between maneuvers (Rowland, 1995), it was necessary to use the same protocol for post-training to avoid any influence that changing the test protocol could have on the estimation of Q and SV. Only the data for the two maneuvers performed at an exercise intensity of 50 % of $pA\dot{V}O_2$ was analyzed as the reliability of Q and SV obtained at the lower exercise intensities was quite poor (Rowland, 1995).

Body composition

The procedure used to obtain bioelectrical impedance analysis (BIA) estimates of body composition has been previously described (Wu, Nielson, Cassady, Cooks, Janz, & Hansen, 1990; Segal, Gutin, Presta, Wang, & Van Itallie, 1985) and summarized by Rowland (1995). The procedure involves placement of transmitter and receiver electrodes on the wrist and ankle on one side of the body and recording the resistance and reactance values to a low electrical current. The resistance values are then entered along with the subjects height and weight into a computer program that generates an estimate of the subject's water content and body composition using empirical equations provided by the manufacturer of the bioelectrical impedance analyzer.

Validity of Wireless Heart Rate Monitors

Although the validity (correlation coefficients ranging from 0.93 to 0.98) and stability (standard error of estimate ranging from 3.7 % to 6.8 %) of the type of wireless heart rate monitor used in this study have been established against ECG monitored HRs for healthy subjects (Leger & Thivierge, 1988), it has not been examined in a population with TBI. HR data obtained by both an electrocardiograph and one of the wireless monitors during the

three pre-training incremental cycle protocols were examined in order to establish that the wireless monitors could accurately monitor exercise intensity in persons with TBI.

STATISTICAL ANALYSIS

Peak Physiological Responses

Descriptive statistics (means and standard deviations) were performed on selected peak physiological and perceptual responses. Selected peak exercise variables were HR, $A\dot{V}O_2$, relative oxygen consumption ($R\dot{V}O_2$, ml/kg/min), power output (watts), minute ventilation ($\dot{V}_E$, l/min), O_2 pulse (ml/beat), ventilatory equivalent for O_2 ($\dot{V}_E/\dot{V}O_2$ ratio), respiratory exchange ratio ($RER = \dot{V}CO_2/\dot{V}O_2$), tidal volume (TV, l/min), breathing frequency (F, breaths/min) and RPE. Although an attempt was made using pre-training data to equate the two groups with respect to fitness level, attrition of subjects resulted in significant differences between the experimental and control groups for several peak physiological responses. A single factor (group) analysis of variance [ANOVA] (Pagano, 1990) was performed to examine how equivalent the two groups were before the experimental group commenced the exercise training program. Significant differences were found between the two groups for several variables, including pHR ($p<0.001$) and $pA\dot{V}O_2$ ($p<0.02$) (see table 1).

Because of the initial difference in $A\dot{V}O_2$ between the two groups, a two-way analysis of covariance (ANCOVA), using $A\dot{V}O_2$ as the covariate, was performed on each of the selected variables to examine the difference between pre- and post-training mean peak values for the two groups. A two-way ANOVA (time X group) was used to examine the change in VO_2. *Post hoc* analysis of all significant F ratios was performed using the Scheffe procedure to determine the effects of time and group.

Improvement in cardiorespiratory fitness as a result of training is often expressed as a percentage increase in $\dot{V}O_2$ max (Astrand & Rodahl, 1986, Haskell, 1989; Jankowski & Sullivan, 1990; McArdle, Katch, & Katch, 1991). Therefore, in order to evaluate the

effectiveness of the prescribed exercise training program to improve cardiorespiratory fitness, the change in peak physiological responses to exercise were calculated as a percentage using the formula:

% diff =[(post-training mean value - pre-training mean value) /

pre-training mean value] X 100.

The means, standard deviations and ranges of the individual percent difference between pre- and post-training values for the selected peak physiological responses were calculated. The contrast between the groups for the mean % change was examined using a one-way ANCOVA (group).

The Bonferroni adjustment to correct for Type 1 errors for a p value of 0.05 (two-tailed) was applied to determine statistical significance of training effects (Ottenbacher, 1991). All statistical analyses were performed with the SPSS package (SPSS, 1988), with the exception of the *post hoc* Scheffe analyses which were computed using Statistica (1991) software.

Submaximal Physiological Responses

One of the five experimental group subjects was unable to successfully complete two post-training Q maneuvers, although three attempts were made. For the purposes of determining the effects of training, the values for the selected submaximal responses to exercise obtained from the one successful maneuver were used as this subject's post-training values.

This introduced a concern regarding the validity of a single Q maneuver to estimate SV. However, the values for Q and SV, recorded during the second of two Q maneuvers performed at 50 % of $pA\dot{V}O$, demonstrated high reliability ($r=0.85$, $p<0.01$) between test sessions separated by two days (Rowland, 1995). Therefore, an alternative to using the mean values from all four Q maneuvers for estimating pre-training levels, would be to use only the

second of the two maneuvers performed during each test session. A comparison of the mean value for SV obtained from the two second maneuvers with the single post-training value obtained in the successful second maneuver for this subject resulted in a decrease of 13.0 % in SV. Comparing the mean of all four pre-training maneuvers with the single post-training value resulted in a decrease of 16.7 %. The similarity in the decline in SV for this subject supports the use of a single Q maneuver as an estimate of his submaximal physiological responses to exercise.

Means and standard deviations were calculated for selected submaximal physiological and perceptual responses to exercise. Because of the significant difference between the groups for pre-training $pA\dot{V}O_2$, an ANCOVA, using $pA\dot{V}O_2$ as the covariate was used to examine the effects of training on selected responses to submaximal exercise. These variables included HR at the time the Q maneuvers were initiated, $A\dot{V}O_2$, power output (watts), O_2 pulse, $\dot{V}_E/\dot{V}O_2$ ratio, Q, Q', SV, SVI, $(a\text{-}v)O_2$ diff, and RPE. Two other variables were examined, the % pHR and the % $pA\dot{V}O_2$. These variables were calculated as the ratio of the value at the time the Q maneuver was initiated and the mean peak value from the preceding incremental cycle tests, multiplied by 100.

The same statistical procedures that were performed on the peak physiological responses were used to determine the effects of training on submaximal responses.

Body Composition

Means and standard deviations for body weight and % body fat were calculated. Pre-training data was analyzed using a t-test for independent groups (Pagano, 1990) to examine differences between the experimental and control group. A two-way ANOVA was used to examine differences between pre- and post-training values for these variables. The percent difference between pre- and post-training values was calculated for both group mean and individual subject data . The change in the % difference in body weight and % fat between groups was examined using a single factor (group) ANOVA.

Validity of Wireless Heartrate Monitors

The mean peak heart rate values were calculated from the peak values obtained by the ECG and wireless heart rate monitor during the three pre-training incremental cycle tests. The mean of a minimum of six submaximal heart rates obtained simultaneously by each instrument were computed for each subject. In order to examine the concurrent criterion-related validity of wireless monitoring of HRs, Pearson product-moment correlation coefficients and the percent difference between HRs recorded by the ECG and the wireless monitor were determined for both mean peak values and for the means of individual subject submaximal HRs. The percent difference in HR values between the electrocardiograph (ECG) and wireless monitor (WM) were calculated using the formula:

$$\% \text{ diff} = ((HR_{ECG} - HR_{WM})/HR_{WM})X100.$$

RESULTS

Peak Physiological Responses

Pre- and post-training means and standard deviations for the selected peak physiological responses for the two groups are presented in table 14. The single factor ANOVA revealed pre-training differences between the experimental and control group mean values for HR ($p<0.0001$), $A\dot{V}O_2$ ($p<0.02$), $R\dot{V}O_2$ ($p<0.0.02$), power ($p<0.02$), $\dot{V}_E$ ($p<0.005$), $\dot{V}_E/\dot{V}O_2$ ratio ($p<0.03$) and TV ($p<0.01$).

The two-way (group X time) ANCOVA, with pre-training $A\dot{V}O_2$ as the covariate, revealed a significant post-training improvement in pHR (up an average of 19.4 bpm, $p<0.002$) for the experimental group. Changes in power output (up 31 watt, $p<0.02$), $\dot{V}_E/\dot{V}O_2$ ratio (up 1.3 L, $p<0.03$) and $\dot{V}_E$ (up 9 L/min, $p<0.04$) were observed but did not reach the significance required for the Bonferroni adjustment (Ottenbacher, 1991). Of the pre-training variables that were significantly higher in the control group, only the post-training value for $\dot{V}_E/\dot{V}O_2$ ratio remained significantly higher ($p<0.003$) for this group. This was due to a 15 L/min greater post-training $\dot{V}_E$ for the control group ($p<0.04$). The increase in $A\dot{V}O_2$ (330

ml/min) in the experimental group and the decrease (100 ml/min) in the control group were not significant changes for either group.

The percent change between group mean pre- and post-training values for the peak physiological responses are also presented in table 14. The means, standard deviations and ranges of individual subject change (%diff) between pre- and post-training values are shown in table 15. The improvement between pre- and post-training values, measured as the % difference between pre- and post-training mean values, for HR, $A\dot{V}O_2$ and $R\dot{V}O_2$ was significantly ($p<0.006$) greater for the experimental group than for the control group. Improved cardiorespiratory fitness for subjects in the experimental group was demonstrated by positive changes in these variables, whereas in the control group, $\dot{V}O_2$ declined between pre- and post-testing.

It can be seen from tables 14 and 15, that a significant increase in pHR occurred in the experimental group. While there was no significant change in the actual values for oxygen consumption for either group (table 14), participation in the exercise program resulted in significantly greater improvement in the percent change in $A\dot{V}O_2$ and $R\dot{V}O_2$ for the subjects in the experimental group (17 % & 21 %, respectively) than was observed for the control group subjects (-4 & -6 %, respectively). Although, power output ($p<0.02$), $\dot{V}_E$ ($p<0.04$), TV ($p<0.02$) and $\dot{V}_E/\dot{V}O_2$ ratio ($p<0.03$) did not demonstrate statistically significant changes following the training program when the Bonferroni adjustment was applied, a physiologically significant improvement was observed. From table 15, it can be seen in the ranges of these variables, that all experimental subjects demonstrated an increase where as some subjects in the control group demonstrated decreases.

Submaximal Physiological Responses

The pre-training and post-training means and standard deviations for the selected submaximal responses to exercise for the two groups are presented in table 16. Initial differences between the groups were observed for HR and $(a\text{-}v)O_2$ diff. A significantly

($p<0.05$) higher (16 bpm) heart rate was required by the control group subjects to achieve an oxygen uptake level of 50 % of $pA\dot{V}O_2$. Control subjects also demonstrated a significantly ($p<0.05$) greater $(a\text{-}v)O_2$ diff than experimental subjects at this intensity level.

Within each group, there was little difference (2 bpm) between pre- and post-training heart rates that accompanied exercising at 50 % of $pA\dot{V}O_2$. There continued to be a 16 bpm higher mean HR for the control group during post-training Q maneuvers ($P<0.02$). Although the mean HR required for subjects to exercise at 50 % of their $p\dot{V}O_2$ did not change between pre- and post-training testing for either group, the two-way ANCOVA revealed a significant ($p<0.003$) reduction in the % pHR for the experimental group.

The percent difference between pre- and post-training group mean values are also displayed in table 16. The means, standard deviations and ranges of individual % difference between pre- and post-training values are displayed in table 17. There were no significant differences between the groups for the % difference between pre- and post-training submaximal responses to exercise for any of the selected variables. Some subjects in both groups showed decreases in these variables, while others demonstrated improvement.

The results in tables 16 and 17 with respect to $A\dot{V}O_2$ were expected, as $A\dot{V}O_2$ was the variable that was monitored and adjusted to remain close to 50 % of each subject's $p\dot{V}O_2$, as determined by the preceding incremental cycle tests to volitional fatigue. There was a small decrease of 80 ml/min (-6.1 %) in the value for $A\dot{V}O_2$ for the control group and a small increase of 40 ml/min (3.7 %) in the experimental group. The % $pA\dot{V}O_2$ decreased by a similar amount (5.3 %) in the control subjects and in the experimental group (-8.8 %), despite the small increase in $A\dot{V}O_2$ for this group.

Power output was virtually unchanged at 50 % $p\dot{V}O_2$ in control subjects, but increased by an average of 7.5 watts (13.6 %) in the experimental group. At this exercise intensity, the value for Q increased slightly by 0.22 liters (1.6 %) in the control subjects ,

whereas the experimental subjects showed a 1.3 liter (9.8 %) increase in Q. As the mean HR varied by less than 2 bpm in both groups , changes in SV would appear to be the major contributor to changes in Q observed. The small increase (8 ml/beat, 7.3%) in SV observed in the experimental group would appear to have made the greater contribution to the increase in Q. There was essentially no difference between pre- and post-training values for (a-v)O_2 diff for the experimental group. However, the control group demonstrated a 1 ml/100ml decrease (-10.3 %) .

Body Composition

The means and standard deviations for pre- and post-training values for weight and % fat are presented in table 14. There was no significant change in these values between testing times. The means, standard deviations and range of individual change in weight and body composition are presented in table 15. There was no significant difference in the percent change between pre- and post-training values between the groups.

Validity of Wireless Heart Rate Monitors

The means of the pHRs obtained simultaneously using an ECG and wireless monitor for each subject during the three incremental cycle tests are presented in table 18, along with the percent difference between the mean peak HRs. The % difference between instruments varied between 0.0 % and 3.0 %. The mean peak HRs also showed very high criterion-related validity (Pearson 'r' = 0.99, $p<0.05$) between instruments. The means of a minimum of six submaximum HRs are shown for each subject in table 19. The Pearson product-moment correlation coefficients, average % difference and maximum % difference between individual sets of HRs are also shown in table 19. The correlation coefficients were high and significant (0.95 or greater, $p<0.05$). The average difference between instruments varied by less than 4 % and the maximum difference between the wireless monitor and the ECG ranged from 4.0 % to 9.8 %. As noted by Leger and Thivierge (1988), there was greater variability between the instruments at lower HRs.

DISCUSSION

Peak Physiological Responses

The reliability of peak physiological responses to cycle exercise has been established using data from the pre-training trials (Rowland, 1995). The reliability between the two post-training incremental cycle tests was examined using the same techniques. The means, standard deviations, % difference between the means, ranges and means of individual differences between trials, Pearson (Pagano, 1990) and intraclass correlation coefficients (Bartko, 1976) are presented in Appendix 10. The results were very similar to those reported by Rowland (1995) and give added support to the conclusion that peak physiological responses can be reliably measured during cycle exercise in persons with TBI. With reliability established for obtaining peak physiological responses during cycle exercise, the use of the means of multiple trials increased the accuracy of the pre- and post-training values used to evaluate the effects of the exercise training program.

Criteria for obtaining a measurement of $\dot{V}O_2$ max include; obtaining a plateau or decrease in $\dot{V}O_2$ with increasing power output (Astrand & Rodahl, 1986), attainment of the age-adjusted predicted maximum HR, achieving an RER value above 1.1 (Bhambhani, Norris & Bell, 1994) and reaching volitional exhaustion. Few subjects in this study could achieve the first three of these criteria at either the pre-training or post-training stage. $\dot{V}O_2$ max was based on volitional exhaustion and has been considered to represent a peak rather than maximum value.

The difference between the pre- and post-training values for RER, although not significant may help to explain the initial differences between the groups for the variables HR, $\dot{V}O_2$, $\dot{V}_E$, $\dot{V}_E/\dot{V}O_2$ ratio, TV and power output. The low pre-training RER for the experimental group suggests that, in addition to cardiorespiratory factors, localized muscle fatigue may have contributed to cessation of exercise. Subsequently, the increased

metabolism associated with the increase in post-training RER may represent peripheral adaptations.

Obtaining an RER significantly above 1.0 during exercise is indicative that a significant portion of the energy being consumed is coming from anaerobic sources. The increased lactic acid that is produced anaerobically, is buffered by sodium bicarbonate in the blood so that the acid-base balance is maintained. The excess CO_2 from this buffering system causes the RER ($\dot{V}CO_2/\dot{V}O_2$ ratio) to increase (McArdle, Katch, & Katch, 1991). Excess CO_2 production and a lowering of blood pH, resulting from the production of lactic acid in the muscles, have strong stimulatory effect on breathing. The post-training values for RER increased by about the same amount (0.09, 8.5 % & 0.08, 8.0%) respectively, for the control and experimental groups. However, cardiorespiratory responses varied between the groups.

Subjects in the experimental group who participated for 10 to 12 weeks in an exercise program to improve their cardiorespiratory fitness, demonstrated an improvement in power output of 29 watts (26.6 %). Little improvement (5 watts, 3.8 %) was observed for subjects in the control group. As a result of their increased power output, experimental subjects demonstrated a 14.7 % increase in mean pHR (up 19 bpm), while this mean value was virtually unchanged (up 1.5 bpm) in the control group. Although the raw change in values for $A\dot{V}O_2$ were not significant for either group, the almost 18 % improvement in $A\dot{V}O_2$ (up an average of 320 ml/min) for the experimental group versus the 4.2 % decrease (down by 100 ml/min) in the control group demonstrates a physiologically significant positive effect of exercise training. A similar effect was observed for $R\dot{V}O_2$, increased by 22.2 % (5 ml/kg/min) for the experimental group and decreased by 5.7 % (1.7 ml/kg/min) for the control group. Oxygen pulse ($\dot{V}O_2$/HR) improved marginally, up 2.1 % (0.3 ml/beat) in the experimental group but decreased by 6.1 % (0.9 ml/beat) in the control subjects. Oxygen pulse is accepted as a relative measure of the SV (Astrand & Rodahl, 1986). The direction of

change in pO_2 pulse is the same as that found for SV obtained at a submaximal intensity, which increased by 7.3 % in the experimental group and decreased by 1.2 % in the control group. In addition to increases in SV, an increase in the $(a\text{-}v)O_2$ difference would also contribute to improvement in the O_2 pulse. The small improvement in O_2 pulse in the experimental group suggests that the efficiency of obtaining and consuming O_2 had not substantially improved as a result of participating in the exercise program.

A mean increase in TV of 260 ml/breath (17.4 %) was observed for experimental subjects, whereas a decrease of 60 ml/breath (-3.2 %) was noted for control subjects. A mean increase in $\dot{V}_E$ of 9.21 l/min (15.9 %) for the experimental group was due primarily to the increase in TV, as F showed little increase (1.5 breaths/min, 4.1 %). An increase in F over the pre-training rate, as a result of training and improved $p\dot{V}O_2$ was expected but did not occur. The greater increase in $\dot{V}CO_2$, than $\dot{V}O_2$, suggested by the increase in RER, did not appear to be enough to stimulate ventilation in the experimental group. Conversely, in the control group, although a smaller increase in $\dot{V}_E$ (4.15 l/min, 5.3 %) was observed, an increase in F of 4.6 breaths per minute (11.4 %) was required to compensate for the decrease in TV that occurred.

Despite participating in the exercise program, the mean post-training value for peak $\dot{V}_E$ for the experimental subjects (67.0 l/min) remained below 75 l/min, which is the expected average value for this age group (Astrand & Rodahl, 1986). This could be due to the fact that although the mean duration of coma was similar between groups, all subjects in the experimental group experienced coma for at least 14 days, while coma duration for two subjects in the control group was three days or less. Given that the period of bedrest would also have been longer for the experimental group, deconditioning affects, including weakened respiratory muscles and reduced compliance of the chest wall (Becker, et al., 1978) could be expected. It is not known whether damage to the brainstem and affects to the

respiratory centers could be a factor, but there were three subjects with brainstem damage in the experimental group versus two in the control group.

The $\dot{V}_E/\dot{V}O_2$ ratio increased by 1.3 (10.9 %) and 3.7 (4.4 %) respectively, for the experimental and control groups suggesting that pulmonary efficiency had decreased from pre-training levels in both groups. The $\dot{V}_E/\dot{V}O_2$ ratio is an index of how economically subjects can obtain O_2. Higher $\dot{V}_E/\dot{V}O_2$ ratios indicate that a large volume of air must be ventilated per unit volume of O_2 consumed (Bhambhani & Singh, 1985). A lower ratio indicates that O_2 is obtained more efficiently. The small increase in $\dot{V}E/\dot{V}O_2$ ratio for the experimental subject indicates that the increase in $\dot{V}O_2$ was accompanied by an increase in $\dot{V}_E$, that was accomplished primarily via an increased TV. The larger increase in the $\dot{V}_E/\dot{V}O_2$ ratio for the control subjects was due to a decrease in $\dot{V}O_2$ and an increase in $\dot{V}_E$, brought about primarily by an increase in F.

The improvement in cardiorespiratory fitness in the experimental group, following the exercise training program supports the findings reported by Jankowski and Sullivan (1990) and Hunter et al. (1990) that participation in vigorous exercise can improve cardiorespiratory fitness in adults with TBI. A comparison of the percent change in oxygen consumption found in this study with that reported in these two studies is presented in Table 20. The mean percent change was slightly higher in this study, but the range of individual change was smaller. None of the exercise group subjects demonstrated a decrease in $R\dot{V}O_2$, as was seen in the study by Jankowski and Sullivan (1990).

Another comparison is provided by a study on the effects of aerobic exercise in individuals with physical disabilities, excluding those with cerebral vascular accidents or infarction, cardiovascular dysfunction, diabetes or neuromuscular disorder that prevented testing (Santiago, Coyle, & Kinney, 1993). A comparison of the percent change in the variables $A\dot{V}O_2$, $R\dot{V}O_2$, $\dot{V}_E$, and power output between the exercise and control groups for subjects from this study and the Santiago, Coyle and Kinney study (1993) is presented in

table 21. The improvement in these variables was similar in both exercise groups and represents "an improved cardiorespiratory/vascular and metabolic capacity to meet the physical demands of everyday living and ambulation" (Santiago et al, 1993, p. 1195).

A slight increase in V_E and power output and smaller declines in $A\dot{V}O_2$ and $R\dot{V}O_2$ were observed in the control subjects with TBI when compared to the Santiago et al. (1993) control subjects with physical disabilities who were no longer involved in formal rehabilitation. This suggests that the brain injured control subjects have benefited from participation in rehabilitation. Even without an emphasis on improving aerobic fitness, rehabilitation activities can marginally improve or at least slow the rate of decline in cardiorespiratory fitness that would occur if these individuals were not engaged in supervised physical activities such as physical, occupational and recreational therapy programs. Still, the greater improvement in cardiorespiratory fitness and power output observed in the experimental subjects with TBI following the supervised aerobic training program are emphasized in light of the declines in $\dot{V}O_2$, TV, and SV in the control subjects, who continued to regularly participate in these types of rehabilitation. Alternatively, these results also suggests that a decline in cardiorespiratory fitness will follow discharge from formal rehabilitation programs unless supports have been put in place to insure individuals can and want to participate in exercise programs.

Submaximal Physiological Responses

The reliability of submaximal (50 % of $p\dot{V}O_2$) physiological responses during cycle exercise has been established using data from the four pre-training maneuvers performed at this intensity (Rowland, 1995). The reliability of submaximal exercise responses between the two post-training incremental cycle tests was examined using the same techniques. The means, standard deviations, % difference between the means, ranges and means of individual differences between trials, Pearson (Pagano, 1990) and intraclass correlation coefficients (Bartko, 1976) are presented in Appendix 11. With the exception of $(a\text{-}v)O_2$ diff

which was lower (Pearson 'r's of 0.63 Vs 0.76), the reliability coefficients were high and similar to those determined between pre-training maneuvers. These results substantiate that submaximal exercise responses can be reliably measured in persons with TBI.

An examination of the data presented in tables 16 and 17, indicates that with the exception of %pHR, there were no significant differences between pre- and post-training submaximal values. The pre-training difference between the two groups in peak HR and $A\dot{V}O_2$ is reflected in the higher $A\dot{V}O_2$ and HR for the control group at the submaximal exercise intensity of 50 % $p\dot{V}O_2$. Although, the value for $A\dot{V}O_2$ was closely monitored and the resistance modified when required to maintain a level close to 50 % of $pA\dot{V}O_2$, there was a slight decrease from 56.4 % and 57.8 % to 53.4% and 52.7 %, respectively for the control and experimental groups. The mean pre- and post-training HRs for both groups increased by less than two beats per minute. However, as the pHR for the experimental group had increased from 132 bpm to 151 bpm, this resulted in the HR intensity, reflected by the % pHR decreasing. The 26.6 % increase in peak power output in the experimental group was mirrored by an increase in power output of 7.5 watts (13.6 %) at approximately 50 % $p\dot{V}O_2$. As a result of this increase in submaximal power output, there was no bradycardia for the experimental group at this submaximal level despite the decrease in %pHR.

There was no significant increase in mean SV as a result of participating in the exercise program. Given the low mean pre-training peak HRs for the experimental subjects shown in table 12, which equated to 65 %, 80 %, 78 %, 71 % and 55 % of their age-adjusted maximum heart rates, it is reasonable that the most significant improvement in cardiac function was in HR and not SV.

Within the experimental group, two subjects (1 and 5) who showed decreases in SV of 12 ml/beat (12.2 %) and 23.8 ml/beat (16.7 %) respectively, also had the highest % change in peak HR, with increases of 20 bpm (15.7 %) and 28.5 bpm (27.1 %). Subjects 2, 3 and 5 displayed increases in SV of 37.1, 22.7 and 21.6 ml/beat, which equates to increases of 27.2 %,

17.8 %, and 19.7 %, respectively. For these three subjects, smaller post-training increases in HR were observed, 15.8 bpm (10.4 %), 19 bpm (12.6 %), and 13.5 bpm (11.0 %), respectively. Three of the control subjects showed increases in SV, but in each case the difference was less than 6 %.

Neither $\dot{V}_E/\dot{V}O_2$ ratio or (a-v)O_2 diff showed any significant change between pre- and post-training for the experimental group. However, $\dot{V}_E/\dot{V}O_2$ ratio increased and (a-v)O_2 diff decreased by 10 % in the control group. This would suggest a decline in both pulmonary efficiency and O_2 uptake at the cellular level in the control group. The lack of a significant decrease in the $\dot{V}_E/\dot{V}O_2$ ratio and increase in (a-v)O_2 diff in the experimental group reinforces that there was little change in the pulmonary efficiency or peripheral adaptations, and that increased cardiovascular function mainly contributed to the significantly improved aerobic fitness in the experimental group.

Given the small number of subjects in the experimental and control groups, it is not possible to provide a reason for the lack of a significant improvement in SV and $\dot{V}_E/\dot{V}O_2$ ratio at the submaximal exercise level of 50 % of $p\dot{V}O_2$. Had the exercise program continued, attendance been more faithful, and the intensity at which these subjects could maintain their training HRs increased, improvement in SV and $\dot{V}_E/\dot{V}O_2$ ratio for subjects 1 and 5 might also have occurred. The results of this examination of training effects on SV and other submaximal responses to exercise are inconclusive. However, there appears to be a physiologically significant positive affect, as there was a mean improvement in SV for the experimental group of 7.3 %, while the group mean for the control group declined by 1.2 %. Further research involving a longer duration (in terms of months) of aerobic training is warranted, as SV is considered to be the primary factor associated with increases in Q and $\dot{V}O_2$ associated with endurance training.

Body Composition

Statistics showing high test-retest reliability of between post-training estimates of body composition (%fat) obtained by bioelectrical impedance analysis (BIA) are shown in Appendix 11. The reliability between post-training trials was the same as in the pre-training trials (Pearson 'r' and ICC = 0.99). Although the validity of BIA estimates body composition has been demonstrated in healthy adults (Segal, Gutin, Presta, Wang, & Van Itallie, 1985) and children and adolescents (Wu, Nielson, Cassady, Cooks, Janz, & Hansen, 1990), it has only recently been investigated in persons with TBI (Rowland, 1995).

The lack of a significant weight loss or reduction in body fat in the experimental group was likely due to several factors. In order to lose weight there has to be more calories expended by metabolic processes than are taken in by consumption of food and drinks. Adding the exercise training program to the schedules of the experimental group increased their caloric expenditure, but not to the degree that the group as a whole lost weight. Although nutritional status and body weight are routinely monitored by the physicians, clinical dietitians and nursing staff in the rehabilitation program, diet was not monitored or controlled as part of this study. Most patients in this program have access to a snack bar and cafeteria where they can purchase foods and drinks that are not monitored. A review of the exercise training literature has shown that only a small reduction in body weight results from exercise unless diet is monitored (Wilmore, 1983). Energy is derived almost equally from carbohydrates and fat during short periods of relatively moderate exercise (McArdle, Katch, & Katch, 1991). When the duration of the exercise exceeds one hour, carbohydrates become depleted and there is a gradual increase in the quantity of fat that is utilized as an energy source. Oxygen is essential for the oxidation of free fatty acids and the contribution of fat to energy metabolism at a given workload increases with higher $\dot{V}O_2max$ values (McArdle, Katch, & Katch, 1991). It was estimated using a formula provided by Jones, Makrides, Hitchcock, Chypchar, and McCartney (1985) that the pre-training $pA\dot{V}O_2$ values for three of

the five experimental subjects were below 65 % of their ideal $\dot{V}O_2$max values (44 %, 58 % & 63 %. Post-training p$\dot{V}O_2$s improved but remained low; respectfully, 53 %, 70 % and 75 % of the ideal values. Therefore, when exercising at submaximal levels, subjects were not increasing their rate of fat metabolism. Subjects averaged only 2.5 sessions per week and typically exercised for approximately 45 minutes per session. It is likely that subjects did not exercise often enough, long enough or at an O_2 consumption level high enough to promote increased fat metabolism.

Because overeating may be a common consequence of TBI (Henson, De Castro, Stringers, & Johnson, 1990), estimation of body composition may become an important tool in controlling the caloric intake and educating individuals with TBI and their caregivers about the need to balance food intake with physical activity. BIA has the potential to be a quick and relatively comfortable procedure for subjects to endure compared to skin fold measurements. Validation against body composition determined by skin-fold measurement in the field and against hydrostatic weighing in the laboratory are areas of future research.

Validity of Wireless Heart Rate Monitors

The high and significant Pearson 'r's and small % difference between the wireless monitor and the ECG, established that the intensity of the exercise training programs can be accurately monitored using wireless monitors. Therefore, the exercise HRs shown in table 2 are an accurate representation of the intensity at which subjects in the experimental group trained. Although the fourth and fifth experimental group subjects could not maintain an initial HR that approximated that recorded when they were exercising at 50 % of their pAVO_2, all experimental group subjects exercised above the intensity of 60 % of their pHR as recommended by the ACSM (1990) for the development and maintenance of cardiorespiratory fitness.

Persons with TBI appear to have difficulty monitoring the intensity at which they are exercising. The reliability of the rating of perceived exertion (RPE) at peak exercise

intensity was shown to be poor amongst the three pre-training incremental cycle tests. The reliability of peak RPE values from the two post-training trials was examined using the same statistics used by Rowland (1995). These statistics are shown in Appendix 10. Post-training RPE values showed a higher and significant degree of reliability (Pearson 'r' = 0.70, $p<0.05$). However, the ICC for RPE remained low, although it improved (0.48 Vs 0.23) over the pre-training value. The additional practice by the experimental group in using the RPE scale during the exercise training program may have contributed to the increase in the reliability correlation coefficients obtained post-training. However, the use of the RPE scale continues to remain suspect as a reliable measure in persons with TBI.

More research is needed into the ability of persons with TBI to perceive the rate at which they are exerting themselves. An accurate perception of exertion in persons with TBI would assist them to monitor intensity when exercising and perhaps just as important, when performing activities of daily living and worker roles. This would help them to perform these activities within safe limits that could lead to improvement in their physical work capacity.

The wireless HR monitors used in this study demonstrated high validity and reliability. Therefore, similar monitors could be used by persons with TBI to monitor exercise intensity to ensure that they exercised at a level sufficient to promote training effects (ACSM, 1990; Astrand, 1987) and avoided risks of over exertion. However, as these instruments cost several hundred dollars they may not be affordable to many individuals with TBI. Another possibility is that persons with TBI can learn to accurately take their own pulse rate as an indication of the intensity at which they are exerting themselves. Given the cognitive and neuropsychological deficits that typically accompany moderate to severe TBI, such as decreased learning of new skills, attention deficits, and memory impairments, the ability to monitor exertion and training intensity are areas that warrant further investigation in this population.

IMPLICATIONS FOR REHABILITATION

The issue of return to work for individuals with TBI is often examined from an economic perspective. The cost to society of lost wages and taxes for survivors of TBI and family caregivers, if they must give up work in order to provide care, are compared the costs of vocational rehabilitation programs (Abrams, Barker, Haffey, & Nelson, 1993). Another factor that should be considered is the possible future costs of health care for secondary health problems should the opportunity to return to some form of work be replaced by adoption of a sedentary lifestyle. The ability to work at least three hours without a break has been identified as an important factor in successfully obtaining and maintaining employment for persons with TBI (Wehman et al., 1993). Therefore, improving aerobic conditioning could help secure employment for persons with TBI. Persons with TBI, who are capable of securing employment, will also likely benefit from the increased work load on their cardiorespiratory and musculoskeletal systems. For example, the subject who was discharged from the rehabilitation program but returned for post-training testing demonstrated improvement in cardiorespiratory fitness. This individual had been working at a manual labour job and riding a mountain bike as his primary mode of transportation since completing the pre-testing. Using the same comparisons of the means of the pre- and post-test trials as was used for the control and experimental group subjects, this individual showed the following increases in measures of fitness; pHR (7.4 %), $A\dot{V}O_2$ (8.0 %), $R\dot{V}O_2$ (12.1 %), TV (20.9 %) and SV (16.1 %). A reduction in weight (-2.6 %) and in % body fat (-17.5 %) was also recorded for this subject.

The results of this study and the studies by Hunter et al. (1990) and Jankowski and Sullivan (1990) demonstrate that individuals with TBI can improve their aerobic fitness (often referred to as physical work capacity) with exercise programs designed to place an overload on their cardiorespiratory systems. A greater emphasis on improving cardiorespiratory fitness during rehabilitation for persons with TBI could result in greater success at

subsequent vocational rehabilitation. Individuals with improved $p\dot{V}O_2$, as a result of aerobic training, should demonstrate improved endurance in work activities. It has been suggested that improvement in functional capacity resulting from physical conditioning can improve a person with TBI's chances in maintaining employment (Bray, Carlson, Humphrey, Mastrilli, & Valko, 1987). This is an important area in which further research is warranted.

During the rehabilitation phase, improvement in aerobic fitness is thought to contribute to increased alertness and the recovery of attention and memory function in persons with TBI (Smigielski, Malec, Thompson, & DePompolo, 1992). Aerobic exercise has been suggested as a treatment for reversing the decreased blood flow that occurs during the acute phase following TBI (Gummow, Miller, & Dustman, 1983). It has been shown in healthy subjects that during exercise, blood flow to the brain can increase by as much as 30 % over the resting rate (Thomas, Schroeder, Secher, & Mitchell, 1989). Although, the brain utilizes a substantial amount (20%) of the body's available supply of oxygen and calories (Kalat, 1984), mental work requires only a slight increase in oxygen consumption (Astrand & Rodahl, 1886). However, mental work associated with increased muscle tension or emotional stress can increase the oxygen uptake level in the brain by at least 10 % (Astrand & Rodahl, 1986). It could well be that regular participation in vigorous exercise results not only in adaptations within the cardiorespiratory and musculoskeletal systems (Astrand & Rodahl, 1986; McArdle, Katch, & Katch, 1991) but within the brain as well. Any increases in the ability of brain cells to obtain and utilize oxygen and nutrients could contribute to a recovery in mental processes. As evidence accumulates that aerobic fitness can be reliably measured and improved through exercise in persons with TBI, future research should examine in more detail what effect improved aerobic fitness might have on cognitive abilities in this population.

There is evidence that physical activity and exercise can have positive effects on psychological functions (Pate, et al., 1995) including: alleviation of symptoms associated with mild to moderate depression, reduction of symptoms of anxiety, improvement of self-concept, confidence and mood (Taylor, Sallis, & Needle. 1985). Increased confidence in the ability to be physically active is related to increased participation in physical activities in the general population (Pate, et al, 1995). Cohadon (1981) has stated that a similar relationship between confidence and activity level exists for persons with TBI.

Much of the evidence associating improvements in psychological health to exercise comes from studies of sedentary middle-aged (King, Taylor, Haskell, & DeBrusk, 1988) and older (King, Taylor, & Haskell, 1993) men and women. Taylor, Sallis and Needle (1985), after examining the literature regarding clinical and non-clinical populations, found conflicting support for the effects of exercise on psychological health. They concluded that there appears to be a positive effect but recommended further scientific investigation. As symptoms of depression and anxiety are commonly found in persons with TBI (Rosenthal & Bond, 1990), research into whether improvement in aerobic and physical fitness has an effect on these psychological symptoms in persons with TBI should be undertaken.

In conclusion, the results of this study indicated that significant improvement in cardiorespiratory fitness in persons with TBI can result from engaging in a program of vigorous exercise. Three of the subjects initiated training at an intensity of approximately 50 % or less of their age-predicted HRmax, which is lower than the intensity (60 % to 90 % of HRmax) recommended by the American College of Sports Medicine (1990) for improvement in cardiorespiratory fitness. At the completion of the training program one subject was still exercising below this level, while two others just met this criteria. The positive training effects demonstrated by the experimental group, which can be readily seen in figure 1, suggests that initial training intensity might not be as important a factor as are the frequency,

duration of sessions and length in terms of weeks or months that a person with TBI participates in an exercise program. Given the initial low level of cardiorespiratory fitness of some individuals with TBI, undue attention to training intensity may result in some persons being exempted from exercise training programs. It would appear that a more appropriate approach would be to start at a training intensity that is tolerated so that motivation to continue is supported and increase the intensity when positive training effects occur. This approach would require the ability to monitor training intensity on a regular if not continuous basis and repeat measurements of cardiorespiratory fitness in order to adjust the training intensity to ensure an overload to promote continued improvement.

Table 12 Characteristics of Subjects

Exercise Group									
Subject	**Age**	**Time post-injury**	**Coma**	**Height**	**Weight**	**Peak VO_2**	**Peak HR**	**% Fat**	**Cause of TBI location of injury**
No., Sex	(years)	(months)	(days)	(cm)	(kg)	L/min	bpm		
1, F	22	19	18	171.5	78.5	1.19	128	34.7	MVA, Lt. cerebral hemis, & brainstem
2, M	28	13	56	181.0	100.5	2.90	153	22.0	MVA, diffuse, global & brainstem
3, M	27	6	21	183.0	88.0	2.28	151	18.7	MVA, Rt. cerebral hemis, mild diffuse cerebellar atrophy
4, F	47	13	14	158.0	56.0	1.16	123	22.7	MVA, anoxia, Lt. frontal & diffuse global,
5, M	29	10	35	167.0	77.3	1.74	105	13.3	IA, Lt. frontal & brainstem
Mean	**30.6**	**12.2**	**28.8**	**172.1**	**80.1**	**1.86[a]**	**132[a]**	**22.3**	
SD	**9.6**	**4.8**	**17.1**	**10.3**	**16.4**	**0.74**	**20**	**7.8**	
Control Group									
Subject	**Age**	**Time post-injury**	**Coma**	**Height**	**Weight**	**Peak VO_2**	**Peak HR**	**% Fat**	**Cause of TBI, location of injury**
No., Sex	(years)	(months)	(days)	(cm)	(kg)	L/min	bpm		
1, F	22	9	42	170.5	74.7	1.32	141	33.0	MVA, diffuse, global & brainstem
2, M	24	7	49	167.0	63.6	2.77	180	8.0	MVA, diffuse, global & brainstem
3, M	34	57	21	171.0	100.0	2.95	139	27.0	Rt. fronto-parietal cerebral abscess
4, F	47	45	1	160.0	70.0	1.42	152	29.3	MVA, diffuse, global
5, M	18	5	3	177.0	87.8	3.30	182	16.7	anoxia, global, diffuse
Mean	***29.0***	***24.6***	**23.2**	**169.1**	**79.2**	**2.35[a]**	***160[a]***	**22.8**	
SD	**11.7**	**24.5**	**21.9**	**6.2**	**14.6**	**0.92**	**21**	**10.2**	

MVA - motor vehicle accident IA - *industrial accident* CVA - cerebral vascular accident

[a] significant pre-training difference between groups

Table13 Exercise Training : Intensity and Frequency

Subject	HR at 50% pVO_2	Frequency of Exercise		Initial Intensity of Training			Final Intensity of Training		
	bpm	Weeks	Sessions	HR[a]	% pHR	% Age Adjusted HRmax	HR[a]	% pHR	% Age Adjusted HRmax
1	105	12	28	101	79	51	116	90	59
2	140	8	19	143	94	74	154	101	80
3	120	8	21	119	79	62	125	83	65
4	100	10	26	85	69	46	97	79	56
5	90	12	34	80	76	53	115	110	60

[a] Hrs averaged over the first and last week of training

Table 14

Comparison of the Mean Pre- and Post-training Values for Selected Peak Physiological Responses

Variable	Group	pre-training mean (n= 3 trials)	(SD)	post-training mean (n=2 trials)	(SD)	% change
HR	C	158.9[b]	20.9	160.4	19.0	0.9[d]
bpm	E	132.0	20.1	151.4[a]	17.2	14.7
$A\dot{V}O_2$	C	2.35[b]	0.92	2.25	0.91	- 4.2[d]
L/min	E	1.86	0.74	2.19	0.94	17.7
$R\dot{V}O_2$	C	29.7[b]	11.0	28.0	11.8	- 5.7[d]
ml/kg/min	E	22.6	5.2	27.6	8.0	22.1
Power output	C	138.3[b]	59.1	143.5	69.6	3.8
watts	E	109.0	46.8	138.0	53.6	26.6
$\dot{V}_E$	C	78.3[b]	27.13	82.5[b]	30.43	5.3
L/min	E	57.8	20.66	67. 0	29.97	15.9
O_2 pulse	C	14.7	5.3	13.8	4.5	- 6.1
ml/beat	E	13.9	4.3	14.2	4.9	2.1
$\dot{V}_E/\dot{V}O_2$ ratio	C	34.0 [b]	4.8	37.7	6.7	10.9
	E	29.2	1.0	30.5	1.8	4.4
RER	C	1.06	0.06	1.15	0.10	8.5
	E	1.00	0.05	1.08	0.06	8.0
TV	C	1.90[b]	0.52	1.84	0.64	- 3.2
L/breath	E	1.49	0.37	1.75	0.45	17.4
F	C	40.6	5.0	45.2	7.5	11.4
breaths/min	E	35.4	7.7	36.9	7.8	4.1
Weight	C	79.2	14.6	80.9	16.7	2.1
Kg	E	80.1	16.4	77.5	15.2	- 3.2
% Fat	C	22.8	10.3	21.5	9.0	- 5.7
	E	22.3	7.8	21.2	8.8	- 4.9

[a] significantly different from pre-training value

[b] significant pre-training difference between groups

[c] significant post-training difference between groups

[d] significant post-pre-training difference between groups

Table 15 ***Comparison of the Individual % Difference Between Pre- and Post-training Values for some Peak Physiological Responses***

	Experimental Group (n=5)		Control Group (n=5)		Significance of Difference Between Groups, % diff
Variable	mean % diff (SD)	range individual % diff	mean % diff (SD)	range individual % diff	(P)
HR beats/min	15.4 (6.9)	+ 10.4 to + 27.1	1.2 (4.0)	- 4.1 to + 6.4	(0.004)[a]
$\dot{V}O_2$ l/min	16.9 (7.0)	+ 4.4 to + 20.9	- 4.4 (10.6)	- 13.6 to + 12.4	(0.006)[a]
$\dot{V}O_2$ ml/kg/min	20.8 (12.2)	+ 4.8 to + 35.0	- 6.1 (10.1)	- 20.3 to + 7.4	(0.006)[a]
Power output watts	30.54 (19.5)	+ 17.6 to + 65.0	1.6 (10.6)	- 10.0 to + 12.5	(0.02)
$\dot{V}_E$ l/min	22.3 (10.8)	+ 5.0 to + 33.6	5.3 (11.7)	- 10.2 to + 19.4	(0.04)
O_2 pulse ml/beat	1.7 (7.2)	- 6.0 to + 9.7	- 5.5 (10.6)	- 11.3 to + 12.9	NS
$\dot{V}_E/\dot{V}O_2$ ratio	4.5 (4.1)	+ 0.5 to + 10.5	10.5 (9.7)	- 0.9 to + 25.5	NS
RER	7.3 (3.7)	+ 4.3 to + 21.5	8.7 (7.9)	+ 0.8 to + 21.5	NS
TV ml/breath	17.4 (12.0)	+ 5.5 to + 30.4	- 4.7 (12.0)	- 24.8 to + 5.1	(0.02)
F breaths/min	4.5 (10.9)	- 7.8 to + 16.7	11.6 (13.7)	- 8.3 to + 15.1	NS
Weight kg	- 2.9 (6.0)	- 10.1 to + 4.9	2.0 (7.0)	- 7.8 to + 11.3	NS
% Fat	- 4.3 (13.4)	- 18.2 to + 15.8	-3.4 (12.2)	- 18.2 to + 14.0	NS

[a] significant at the sharpened Bonferroni adjusted level of $p<0.007$

Table 16 Comparison of the Group Mean Pre- and Post-training Values for Selected Submaximal Responses to Exercise

Variable	Group	pre-training mean (3 trials)	(SD)	post-training mean (2 trials)	(SD)	% change
HR	C	128.2[b]	14.5	129.9	13.5	1.3
bpm	E	112.4	20.0	113.5	15.1	1.0
% pHR	C	81.1	6.5	81.1	3.0	0.0
	E	85.2	6.9	75.0[a]	6.0	- 12.0
$A\dot{V}O_2$	C	1.31	0.47	1.23	0.57	- 6.1
L/min	E	1.08	0.49	1.12	0.40	3.7
% $pA\dot{V}O_2$	C	56.4	3.4	53.4	7.1	- 5.3
	E	57.8	4.0	52.7	5.8	- 8.8
Power output	C	71.5	29.6	72.5	35.1	1.4
watts	E	55.0	30.9	62.5	29.3	13.6
O_2 pulse	C	10.3	4.0	9.3	3.9	- 9.7
ml/beat	E	9.3	2.8	9.7	2.4	4.3
$\dot{V}_E/\dot{V}O_2$ ratio	C	34.1	3.7	37.6	8.4	10.3
	E	31.9	1.3	31.6	2.3	- 0.9
Q	C	13.49	4.36	13.71	4.55	1.6
L/min	E	13.57	3.38	14.90	4.96	9.8
QI	C	7.10	2.10	7.09	2.05	- 0.1
L/min/sq. m	E	6.98	1.06	7.76	1.97	11.2
SV	C	105.5	33.3	104.2	28.4	- 1.2
ml/beat	E	120.9	17.1	129.7	30.3	7.3
SVI	C	55.1	14.3	54.0	11.6	- 2.0
ml/beat/sq. m	E	63.2	9.6	68.4	15.0	8.2
(a-v) O_2 diff	C	9.7 [b]	1.0	8.7	1.5	-10.3
ml/100 ml	E	7.7	1.6	7.6	0.8	- 1.3
RPE	C	14.7	1.3	15.5	0.8	5.4
	E	15.5	1.9	14.8	3.3	- 4.5

[a] significantly different from pre-training

[b] significant pre-training difference between groups

Table 17 Comparison of the Individual % Difference Between Pre- and Post-training Values for Some Submaximal Responses

	Experimental Group (n=5)		Control Group (n=5)		Significance of Difference Between Group % diff
Variable	mean % diff (SD)	range individual % diff	mean % diff (SD)	range individual % diff	F value (P)
HR	1.7 (8.2)	- 5.9 to + 15.0	1.6 (7.7)	- 5.1 to + 13.8	NS
% pHR	- 11.9 (2.0)	- 9.5 to + 14.7	0.5 (8.2)	- 8.0 to + 13.5	0.01[a]
$\dot{V}O_2$ L/min	7.2 (115)	- 8.2 to + 24.3	- 9.4 (13.8)	- 23.7 to + 6.2	NS
% p$\dot{V}O_2$	- 8.0 (16.2)	- 24.2 to + 19.1	- 4.9 (14.0)	- 23.7 to + 13.1	NS
Power output watts	22.0 (22.8)	- 14.3 to + 41.7	- 1.5 23.8)	- 33.3 to + 33.3	NS
O_2 pulse ml/beat	5.6 (13.5)	- 7.5 to + 27.6	- 11.1 (13.7)	- 22.2 to + 11.8	NS
$\dot{V}_E/\dot{V}O_2$ ratio	- 9.8 5.3)	- 4.8 to + 5.6	9.9 (18.0)	13.7 to + 37.0	NS
Q L/min	8.3 (12.6)	- 9.0 to + 18.8	1.5 (3.2)	- 2.2 to + 5.1	NS
QI L/min/sq. m	9.8 (12.7)	- 8.4 to + 23.9	0.4 (2.5)	- 3.9 to + 2.3	NS
SV ml/beat	7.2 (20.1)	- 16.7 to + 27.2	0.0 (5.7)	- 8.8 to + 5.7	NS
SVI ml/beat/sq.m	8.6 (20.4)	- 13.6 to + 32.6	1.0 (7.3)	- 12.8 to + 5.7	NS
(a-v)O_2 diff ml/100 ml	0.2 (15.9)	- 21.0 to + 19.4	- 10.5 (12.6)	- 24.9 to + 6.6	NS
RPE	- 4.9 (19.0)	- 23.5 to + 18.0	1.2 (2.7)	0.0 to + 6.1	NS

[a] not significant at the sharpened Bonferroni adjusted level of $p<0.007$

Table 18 Comparison of Mean Peak HRs Between ECG and Wireless Monitor

SUBJECT	max HR ECG mean of 3 trials	max HR MONITOR mean of 3 trials	% DIFF
1	125.0	122.5	2.00
2	165.0	160.0	3.03
3	178.6	176.7	1.06
4	160.3	158.0	1.43
5	158.3	155.3	1.90
6	148.7	150.0	0.87
7	140.7	139.7	0.71
8	140.0	135.0	3.57
9	118.3	119.0	0.59
10	128.7	132.3	2.80
11	101.7	99.7	1.97
12	181.0	174.0	3.87
13	135.7	135.7	0.00

Pearson 'r' = 0.97

Table 19 Comparison of Mean Submaximal HRs Between ECG and Wireless Monitor

SUBJECT	mean HR (ECG)	mean HR (monitor)	AVE. % DIFF	MAX % DIFF	r
1	115.75	115.50	1.9	4.0	0.995
2	134.43	132.43	1.9	5.8	0.990
3	162.89	159.33	2.4	4.6	0.996
4	137.30	133.60	3.4	9.4	0.971
5	129.10	127.80	2.4	4.5	0.995
6	114.38	115.08	2.4	6.6	0.996
7	124.29	123.29	1.8	3.5	0.995
8	113.00	112.40	3.1	8.3	0.952
9	100.23	99.31	2.6	7.4	0.999
10	114.25	115.50	2.1	6.7	0.961
11	89.40	88.90	3.9	9.8	0.972
12	134.85	133.62	1.7	5.3	0.994
13	102.60	101.80	1.9	6.8	0.990

mean heart rates calculated from at least 6 simultaneously recorded values from each instrument

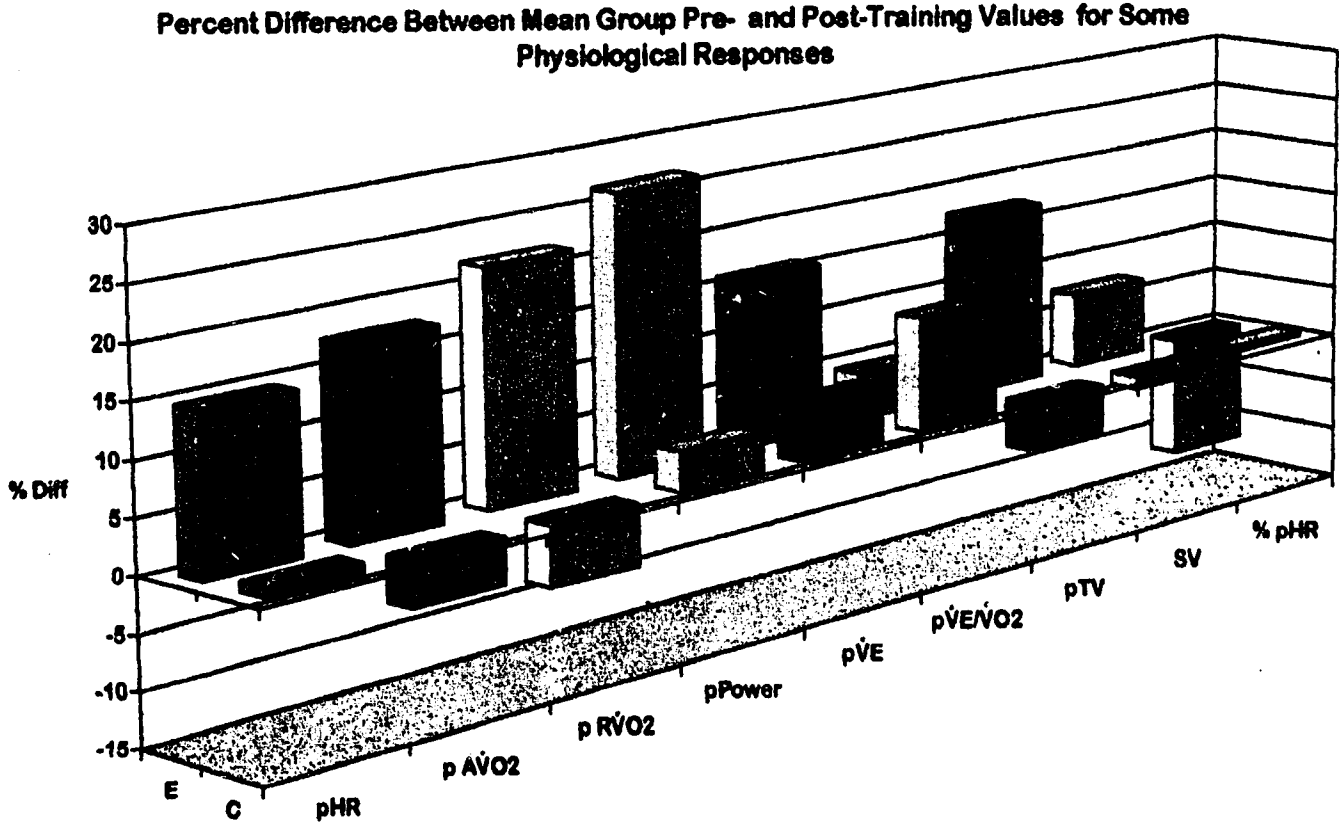
Table 20 Comparison of the Effect of Exercise on Peak Oxygen Uptake in Three Studies in Persons with TBI

Study	Rowland (1995)		Hunter et al. (1990)	Jankowski & Sullivan (1990)
Variable / Statistic	$A\dot{V}O_2$ (l/min)	$R\dot{V}O_2$ (ml/kg/min)	$A\dot{V}O_2$ (ml/min)	$R\dot{V}O_2$ (ml/kg/min)
% change mean T1 Vs T2	18.1	21.6	13.8	15.7
range of individual % change	+ 4.4 to + 20.9	+ 4.8 to + 35.0	+ 1.2 to + 42.1	- 1.6 to + 36.2
mean % individual change (SD)	16.9 (7.0)	20.8 (12.2)	14.4 (12.5)	15.2 (12.1)

Table 21 Comparison of Effects of Training Between Subjects with TBI Vs Physical Disabilities

Variable	Rowland, 1995 - TBI		Santiago, 1993- physical disabilities	
	exercise group	control group	exercise group	control group
$A\dot{V}O_2$	+ 16.9 %	- 4.4 %	+ 22.7 %	- 16.9 %
$R\dot{V}O_2$	+ 20.8 %	- 6.1 %	+ 23.7 %	- 17.1 %
$\dot{V}_E$	+ 22.3 %	+ 5.3 %	+ 16.1 %	- 8.0 %
Power output	+ 30.5 %	+ 1.6 %	+ 23.1 %	- 10.3 %

Figure 1



E - Experimental group

C - Control group

REFERENCES

Abrams, D., Barker, L.T., Haffey, W., & Nelson, H. (1993). The economics of return to work for survivors of traumatic brain injury: vocational services are worth the investment. Journal of Head Trauma Rehabilitation, 8, 59-76.

Ainsworth, B.E., Haskell, W.L., Leon, A.S., Jacobs, Jr., D.R., Montoye, H.J., Sallis, J.F., & Paffenbarger, Jr., R.S. (1993). Compendium of physical activities: classification of energy costs of human physical activities. Medicine and Science in Sports and Exercise, 25, 71-80.

A.C.S.M. American College of Sport Medicine. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness in healthy adults. (ACSM position stand). Medicine and Science in Sports and Exercise, 22, 265-74.

Astrand, P-O. (1987). Exercise physiology and its role in disease prevention and in rehabilitation. Archives of Physical Medicine and Rehabilitation, 68 305-09.

Astrand, P-O. & Rodahl, K. (1986). Textbook of work physiology (3rd ed.). Toronto: McGraw-Hill Book Company.

Bartko, J.J. (1976). On various intraclass correlation reliability coefficients. Psychological Bulletin, 83, 762-65.

Becker, E., Bar-Or, O., Mendelson, L., & Najenson, T. (1978). Pulmonary functions and responses to exercise of patients following cranio cerebral injury. Scandinavian Journal of Rehabilitation Medicine, 10, 47-50.

Bhambhani, Y.N., Eriksson, P., & Steadward, R.D. (1991). Reliability of peak physiological responses during wheelchair ergometry in persons with spinal cord injury. Archives of Physical Medicine and Rehabilitation, 74, 305-11.

Bhambhani, Y.N., Norris, S., & Bell, G. (1994). Prediction of stroke volume from oxygen pulse measurements in untrained and trained men. Canadian Journal of Applied Physiology, 19, 49-59.

Bhambhani, Y.N. & Singh, M. (1985). The effects of three training intensities on VO_2 max and V_E/VO_2 ratio. Canadian Journal of Applied Science, 10, 44-51.

Borg, G. (1970). Perceived exertion as an indicator of somatic stress. Scandinavian Journal of Rehabilitation Medicine, 2, 92-98.

Borg, G. (1982). Psychophysical bases of perceived exertion. Medicine and Science in Sports and Exercise, 14, 377-381.

Bray, L.J., Carlson, F., Humphrey, R., Mastrilli, J.P., & Valko, A.S. (1987). Physical rehabilitation. In M. Ylvisaker & E.M.R. Gobble (Eds.). Community Re-entry for Head Injured Adults (pp.25-85). Boston: Little, Brown and Company.

Cohadon, F. (1981). The importance of rehabilitation programs in the prevention and alleviation of head injury sequelae. Progress in Neurological Surgery, 10, 344-384.

Cooper, M.A. & Sherill, C. (1986). Attitudes toward physical activity of elite cerebral palsied athletes. Adapted Physical Activities Quarterly, 3, 14-21.

Cope, N. & O'Lear, J. (1993). A clinical and economic perspective on head injury rehabilitation. Journal of Head Trauma, 8, 1-14.

Davis, G.M. (1991). Exercise capacity following spinal cord injury. In J.R. Sutton & R. Balnave (Eds.). Cardiovascular and Respiratory Responses to Exercise in Health and Disease (pp. 179-92). Sydney: University of Sidney.

Davis, G.M. (1993). Exercise capacity of individuals with paraplegia. Medicine and Science in Sports and Exercise, 25, 423-432.

Davis, V.P., Fillingim, R.B., Doleys, D.M., & Davis, M.P. (1992). Assessment of aerobic power in chronic pain patients before and after a mult-disciplinary treatment program.

DuBois, D. & DuBois, E.F. (1916). A formula to estimate the approximate surface area if height and weight be known. Archives of Internal Medicine, 17, 863-71.

Fernhall, B. (1993). Physical fitness and exercise training of individuals with mental retardation. Medicine and Science in Sports and Exercise, 24, 442-50.

Fernhall, B., Miller, A.L., Tymeson, G.T., & Burkett, L.N. (1990). Maximal exercise testing of mentally retarded adolescents and adults: reliability study. Archives of Physical Medicine and Rehabilitation, 71, 1065-68.

Figoni, S.F. (1993). Exercise responses and quadriplegia. Medicine and Science in Sports and Exercise, 25, 433-441.

Frankowski, R.F. (1986). Trauma treatment in practice: the head injured patient. Advances in Psychosomatic Medicine, 16, 153-72.

Giles, G.M. (1994). Functional assessment and intervention. In M.A.J. Finlayson & S.H. Garner (Eds.), Brain Injury Rehabilitation: Clinical Considerations (pp. 124-56). Philadelphia: Williams & Wilkins.

Gowland, C. & Gambarotto, C.A. (1994). Assessment and treatment of physical impairments leading to disability after brain injury. In M.A.J. Finlayson & S.H. Garner (Eds.), Brain Injury Rehabilitation: Clinical Considerations (pp. 102-23). Philadelphia: Williams & Wilkins.

Gummow, D., Miller, P., & Distman, R.E. (1983). Attention and brain injury: a case for cognitive rehabilitation of attentional deficits. Clinical Psychology Reviews, 3, 255-274.

Haskell, W.L. (1989). Exercise as a means of maximizing human physical performance and productivity. In R.S. Sanders & A.G. Wallace (Eds.), Biological Effects of Physical Activity (pp. 115-26). Champaign, IL.: Human Kinetics Books

Helledie, N. & Shane, S. (1992). Brain injury: the silent epidemic. Research Highlights, 3, (Vocational and Rehabilitation Research Institute).

Henson, M.B., De Castro, J.M. , Stringer, A.Y., & Johnson, C. (1993). Food intake by brain-injured humans who are in the chronic phase of recovery. Brain Injury, 7, 169-178.

Hjeltnes, N. (1982). Capacity for physical work and training after spinal cord injuries and stroke. Scandinavian Journal of Rehabilitation Medicine, 29, 254-51.

Hunter, M., Tomberlin, J., Kirkikis, C., & Kuna, S. (1990). Progressive exercise testing in closed head-injured subjects: comparison of exercise apparatus in assessment of a physical conditioning program. Physical Therapy, 70, 363-371.

Jankowski, L.W. & Sullivan, S.J. (1990). Aerobic and neuromuscular training: effects on the capacity, efficiency, and fatigability of patients with traumatic brain injuries. Archives of Physical Medicine and Rehabilitation, 71, 500-504.

Jennet, B., Snoak, J., Bond, M., & Brooks, N. (1981). Observations on the use of the Glasgow Outcome Scale. Journal of Neurology, Neurosurgery and Psychiatry, 44, 285-293.

Jennet, B. & Teasdale, G. (1981). Management of Head Injuries. Philadelphia: F. A. Davis Company.

Jones, N.L., Makrides, L., Hitchcock, C., Chypchar, T., & McCartney, N. (1985). Normal standards for an incremental progressive cycle ergometer test. American Review of Respiratory Disease, 131, 700-08.

Kalat, J.W. (1984). Biological Psychology (2nd ed.). Belmont, CA: Wadswroth, Inc.

Kalisky, Z., Morrison, D., Meyers, C., & Von Lufan, A. (1985). Medical problems encountered during rehabilitation of patients with head injuries. Archives of Physical Medicine and Rehabilitation, 66, 25-29.

Kalsbeek, W.D., McLaurin, R.L., Harris, B.S., & Miller, J.D. (1980). The National Head and Spinal Cord Injury Survey: major findings. Journal of Neurosurgery, 53, S19-S31.

King, A.C., Taylor, C.B., & Haskell, W.L. (1993). Effects of differing intensities and formats of 12 months of exercise training on psychological outcomes in older adults. Health Psychology, 12, 292-300.

King, A.C., Taylor, C.B., Haskell, W.L., & DeBusk, R.F. (1989).. Influence of regular aerobic exercise on psychological health: a randomized, controlled trial of healthy middle-aged adults. Health Psychology, 8, 305-24.

Leger, L. & Thivierge, M. (1988). Heart rate monitors: validity, stablity, and functionality. The Physician and Sportsmedicine, 16, 143-151.

Levin, H., Benton, A., & Grossman, R. (1982). Neurobiological Consequences of Closed Head Injury. Oxford: Oxford University Press.

Lindsley, D.F. & Holmes, J.E. (1984). Basic Human Neurophysiology. Amsterdam: Elsevier Science Publishing Co., Inc.

McArdle, W.D. Katch, F.I., & Katch, V.L. (1991). Exercise Physiology: Energy, Nutrition, and Human Performance (3rd. ed.).. Philadelphia: Lea & Febiger.

Miller, A.L., Fernhall, B., & Burkett, L.E. (1993). Effects of aerobic training in adolescents with Down Syndrome. Medicine and Science in Sports and Exercise, 25, 270-274.

Novack, T.A., Roth, D.L., & Boll, T.J. (1988). Treatment alternatives following mild head injury. Rehabilitation Counseling Bulletin, 31, 313-24.

Oddy, M., Coughlan, T., Tyerman, A., & Jenkins, D. (1985). Social adjustment after closed head injury: a further follow-up seven years after injury. Journal of Neurology, Neurosurgery, and Psychiatry, 48, 564-68.

Oddy, M., Humphrey, M., & Utley, D. (1978). Subjective impairment and social recovery after closed head injury. Journal of Neurology, Neurosurgery and Psychiatry, 41, 611-16.

Ottenbacher, K.J. (1991). Statistical conclusion validity: multiple inferences in rehabilitation research. American Journal of Physical Medicine and Rehabilitation, 70, 317-22.

Pagano, R.R. (1990). Understanding Statistics in The Behavioral Sciences (3rd ed.). St. Paul: West Publishing Company.

Pate, R.R., Pratt, M., Blair, S.N., Haskell, W.L., Macera, C.A., Bouchard, C., Buchner, D., Ettinger, W., Heath, G.W., King, A.C., Kriska, A., Leon, A.S., Marcus, B.H., Morris, J., Paffenbarger, Jr., R.S., Patrick, K., Pollock, M.L., Rippe, J.M., Sallis, J., Wilmore, J.H. (1995). Physical activity and public health: A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. Journal of the American Medical Association, 273, 402-07.

Patterson, R.P., Pearson, J., & Fisher, S.V. (1985). Work-rest periods: their effect on normal physiologic response to isometric and dynamic work. Archives of Physical Medicine and Rehabilitation, 66, 348-52.

Perry, J. (1983). Rehabilitation of the neurologically disabled patient: principles, practice and scientific basis. Journal of Neurosurgery, 58, 799-816

Ponichtera-Mulcare, J.A. (1990). Exercise and multiple sclerosis. Medicine and Science in Sports and Exercise, 25, 451-65.

Prigatano, G. and others. (1986). Neuropsychological Rehabilitation After Brain Injury. Baltimore: John Hopkins University Press Ltd.

Prigatano, G.P. & Altman, I.M. (1990). Impaired awareness of behavioral limitations after traumatic brain injury. Archives of Physical Medicine and Rehabilitation, 71, 1058-64.

Rintala, P., Dunn, J.M., McCubbin, J.A., & Quinn, C. (1992). Validity of a cardiorespiratory fitness test for men with mental retardation. Medicine and Science in Sports and Exercise, 24, 941-45.

Rosenthal, M. & Bond, M.R. (1990). Behavioral and psychiatric sequelae. In M. Rosenthal, E.R. Griffith, M.R. Bond, & J.D. Miller (Eds)., Rehabilitation of the Adult and Child with Traumatic Brain Injury (2 ed.). (pp. 179-92). Philadelphia: F.A. Davis Company.

Rowland, G.J. (1995). Reliability of physiological responses during cycle exercise in persons with traumatic brain injury. Unpublished mater's thesis, University of Alberta.

Saltin, B., Blomqvist, G., Mitchell, J.H., Johnson Jr., R.L. Wildenthal, K., & Chapman, C.B. (1968). Response to exercise after bedrest and training. Circulation, Supplement VII, 1-78.

Santiago, M.C., Coyle, C.P., & Kinney, W.B. (1993). Aerobic exercise effect on individuals with physical disabilities. Archives of Physical Medicine and Rehabilitation, 74, 1192-98.

Scherzer, B.P. (1986). Rehabilitation following severe head trauma: results of a three year program. Archives of Physical Medicine and Rehabilitation, 67, 366-74.

Segal, K.S., Gutin, B., Presta, E., Wang, J., & Van Itallie, T.B. (1985). Estimation of human body composition by electrical impedance methods: a comparative study. Journal of Applied Physiology, 58, 1565-71.

Smigielski, J.S., Malec, J., Thompson, J.M., & DePompolo, R.W.(1992). Mayo Medical Center brain injury outpatient program: treatment procedures and early outcome. Mayo Clinic Proceedings, 67, 767-774.

SPSS, Inc. (1988). SPSS-X (Computer software, VAX/VMS release 3.0). Chicago: Author

Statistica (1991). (Statistics Software, release 3.1). Tulsa: Author

Sullivan, S.J. Richer, E., & Laurent, F. (1990). The role of and possibilities for physical conditioning programmes in the rehabilitation of traumatically brain-injured persons. Brain Injury, 4, 407-14.

Taylor, C.B., Sallis, J.F., & Needle, R. (1985).. The relation of physical activity and exercise to mental health. Public Health Reports, 100, 195-202.

Thomas, S.N., Schroeder, T., Secher, N.H., & Mitchell, J.H. (1989). Cerebral blood flow during submaximal and maximal dynamic exercise in humans. Journal of Applied Physiology, 67, 744-48.

Varney, N.R. & Menefee, L. (1993). Psychosocial and executive deficits following closed head injury: implications for orbital frontal cortex. Journal of Head Trauma, 8, 32-44.

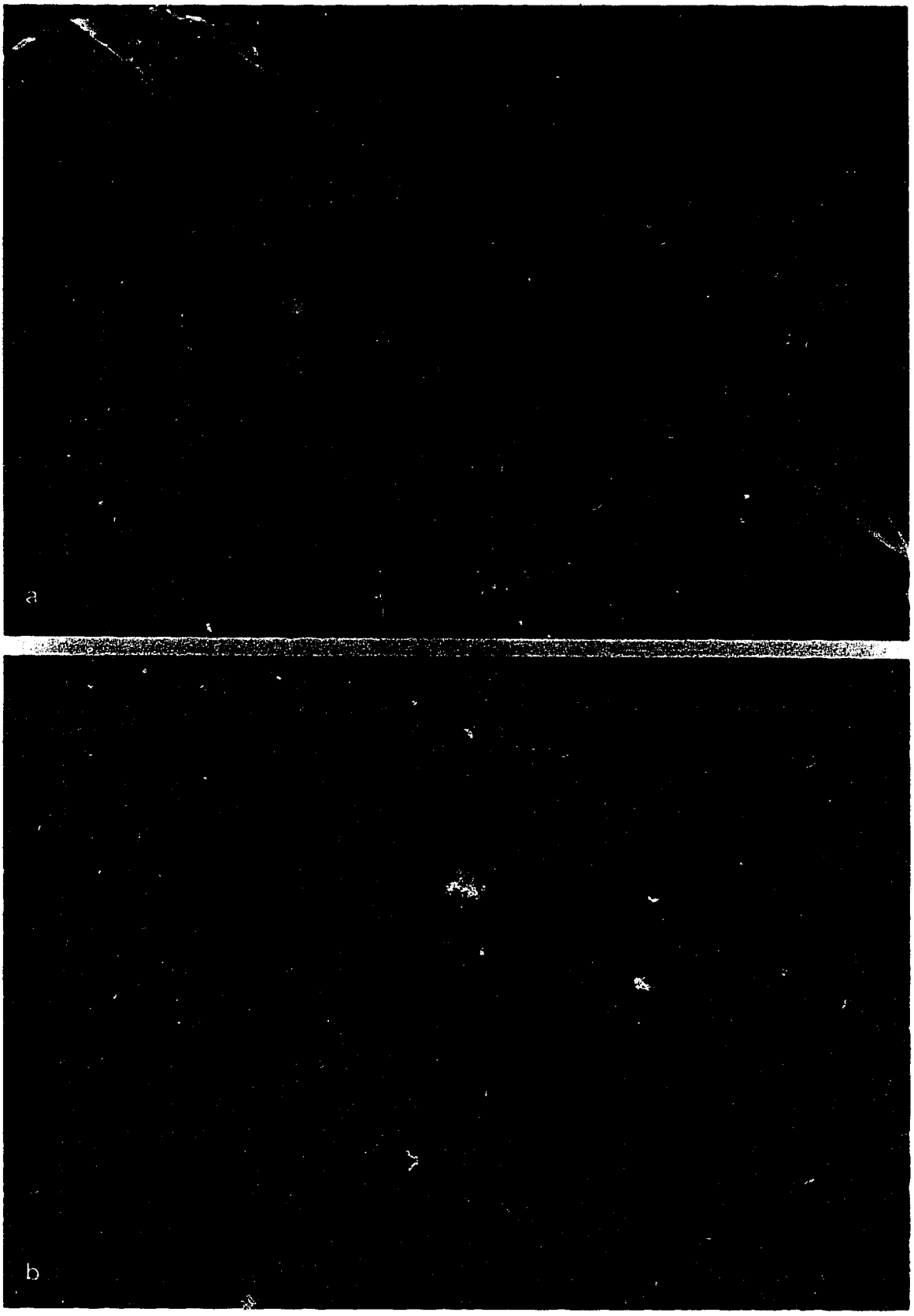
Wehman, P., Kregel, J., Sherron, P., Nguyen, S., Kreutzer, J., Fry, R., & Zasler, N. (1993). Critical factors associated with the successful supported employment placement of patients with severe traumatic brain injury. Brain Injury, 7, 31-44.

Whipp, B.J. & Wasserman, K. (Eds.). (1991). Exercise: Pulmonary Physiology and Pathophysiology. New York: Marcel Dekker, Inc.

Wilmore, J.H. (1983). Appetite and body composition consequent to physical activity. Research Quarterly Exercise and Sports, 54, 415-25.

Wilmore, J.H., Farrell, P.A., Norton, A.C., Cote III, R.W., Coyle, E.F., Ewy, G.A., Temkin, L.P., & Billing, J.E. (1982). An automated, indirect assessment of cardiac output during rest and exercise. Journal of Applied Physiology, 52, 1493-1497.

Wong, P.P., Dornan, J., Schentage, C.T., Ip, R., & Keating, A.M. (1993). Statistical profile of traumatic brain injury: a Canadian rehabilitation population. Brain Injury, 7, 283-94.


a
b

SUPPLIERS

a) Sports Tester, Model PE3000, Potential Training Products Co., Inc., 2410 Speers Road, Oakville, Ontario L6L 5M2 (also sold as the AMF Quantum XL.)

b) R.J.L. Body Composition Analyzer BIA-101, R.J.L. Systems Inc., 9930 Whittier, Detroit, MI. 48224

c) Corival 400, Quinton Instruments Co., 2121 Terry Avenue, Seattle, WA. 98121

d) Model MMC Horizon, Sensormedics, 1630 South State College Boulevard, Anaheim, CA. 98206

e) Model 1500B, Hewlett Packard, 6877 Goreway Drive, Mississauga, Ontario, Canada L4V-1M8

Appendix 1

Table A1

INDIVIDUAL VALUES OF SOME PEAK PHYSIOLOGICAL RESPONSES DURING THE THREE GRADED EXERCISE TRIALS

Variable		HR max	$A\dot{V}O_2$	$R\dot{V}O_2$	$\dot{V}CO_2$	$\dot{V}_E$	TV	F	O_2 PULSE	RER
Subject & trial no.		beats/ min	l/min	ml/kg/ min	l/min	l/min	ml/breath	breaths/ min	ml/beat	
1	trial 1	118	0.96	12.23	1.05	31.9	1.11	28.8	8.14	1.09
	trial 2	127	1.25	15.91	1.15	37.8	1.20	31.5	9.84	0.92
	trial 3	140	1.36	17.32	1.23	40.9	1.27	32.1	9.71	0.90
2	trial 1	147	2.51	25.00	2.25	65.3	1.55	42.3	17.10	0.89
	trial 2	146	2.96	29.48	3.06	86.0	1.74	49.3	20.29	1.03
	trial 3	165	3.24	32.20	3.24	99.5	1.96	50.7	19.61	1.00
3	trial 1	179	2.65	41.71	2.77	76.6	1.92	40.0	14.82	1.04
	trial 2	181	2.70	42.52	2.67	81.7	1.87	43.7	14.94	0.99
	trial 3	181	2.95	46.38	3.20	91.5	2.07	44.3	16.30	1.09
4	trial 1	152	2.38	35.16	2.31	79.8	1.53	52.1	15.66	0.97
	trial 2	160	2.37	35.01	2.54	86.9	1.75	49.7	14.81	1.07
	trial 3	158	2.18	32.20	2.45	80.7	1.74	46.3	13.80	1.12
5	trial 1	155	2.96	36.59	3.11	92.8	1.74	53.4	19.10	1.05
	trial 2	171	2.74	33.87	3.53	107.6	1.95	55.3	16.02	1.29
	trial 3	153	3.32	41.04	3.18	93.7	1.76	53.8	21.70	0.96
6	trial 1	144	2.12	24.09	2.10	54.6	1.74	31.4	14.72	0.99
	trial 2	145	2.15	24.43	2.35	64.3	2.25	28.6	14.83	1.09
	trial 3	164	2.58	29.32	2.62	74.3	2.06	36.0	15.73	1.02
7	trial 1	142	1.26	16.87	1.17	38.5	1.23	31.2	8.87	0.93
	trial 2	149	1.47	19.61	1.47	48.3	1.34	36.0	9.83	1.00
	trial 3	132	1.25	16.68	1.16	39.3	1.23	32.0	9.44	0.93

table continues

Variable	HR max	$A\dot{V}O_2$	$R\dot{V}O_2$	$\dot{V}CO_2$	$\dot{V}_E$	TV	F	O_2 PULSE	RER
Subject & trial no.	beats/ min	l/min	ml/kg/ min	l/min	l/min	ml/breath	breaths/ min	ml/beat	
8 trial 1	137	2.91	29.10	3.07	90.1	2.44	37.0	21.24	1.05
trial 2	148	3.06	30.64	3.36	106.3	2.53	42.0	20.70	1.10
trial 3	133	2.87	28.70	3.16	97.5	2.46	39.6	21.58	1.10
9 trial 1	133	1.22	21.79	1.28	36.6	1.14	32.0	9.17	1.05
trial 2	121	1.09	19.41	1.15	27.8	1.22	22.8	8.98	1.06
trial 3	115	1.18	21.07	1.32	36.3	1.20	30.2	10.26	1.12
10 trial 1	164	1.56	22.21	1.73	69.3	1.51	46.0	9.48	1.11
trial 2	149	1.40	19.99	1.54	60.9	1.47	41.4	9.39	1.10
trial 3	142	1.31	18.71	1.51	50.4	1.45	34.7	9.23	1.15
11 trial 1	96	1.64	21.22	1.56	48.7	1.29	37.7	17.08	0.95
trial 2	112	1.79	23.16	1.75	52.7	1.19	44.3	15.98	0.98
trial 3	107	1.78	23.03	1.79	50.2	1.49	33.8	16.64	1.01
12 trial 1	187	3.33	37.93	3.46	103.9	2.29	45.4	17.81	1.04
trial 2	180	3.23	36.73	3.77	117.0	2.36	49.7	17.92	1.17
trial 3	179	3.35	38.14	3.50	103.5	2.29	45.2	18.71	1.04
13 trial 1	132	1.92	19.51	2.03	59.1	2.01	29.4	14.55	1.06
trail 2	141	2.08	21.12	2.09	59.5	2.01	29.6	14.74	1.01
trail 3	138	1.87	19.00	1.91	54.2	1.96	27.6	13.55	1.02

Appendix 2

Table A2

Individual Subject Differences Between Trials for Some Peak Physiological Responses

Variable		HR	$A\dot{V}O_2$	$R\dot{V}O_2$	$\dot{V}CO_2$	$\dot{V}_E$	TV	F	O2 PULSE	RER
Subject & trial no.		beats/ min	l/min	ml/kg /min	l/min	l/min	l/breath	breaths/ min	ml/beat	
1	%diff 1-2	7.6	30.2	30.2	9.5	18.5	8.3	9.4	21.0	-15.9
	%diff 2-3	10.2	8.8	8.8	7.0	8.2	6.2	1.9	- 1.3	- 1.7
	%diff 1-3	18.6	41.7	41.7	17.1	28.2	15.0	11.5	19.4	-17.3
2	%diff 1-2	- 0.7	17.9	17.9	36.1	31.7	12.8	16.6	18.7	15.4
	%diff 2-3	13.0	9.2	9.2	5.8	15.7	12.6	2.8	- 3.4	- 3.1
	%diff 1-3	12.2	28.8	28.8	44.0	52.4	27.1	19.9	14.7	11.8
3	%diff 1-2	1.1	1.9	1.9	- 3.5	6.7	- 2.4	9.2	0.8	- 5.4
	%diff 2-3	0.0	9.1	9.1	19.8	12.0	10.5	1.4	9.1	9.8
	%diff 1-3	1.1	11.2	11.2	15.6	19.4	7.8	10.8	10.0	3.9
4	%diff 1-2	5.3	- 0.4	- 0.4	10.1	8.9	14.2	- 4.6	- 5.4	10.6
	%diff 2-3	- 1.2	- 8.0	- 8.0	- 3.7	- 7.1	- 0.4	- 6.8	- 6.8	4.7
	%diff 1-3	4.0	- 8.4	- 8.4	6.1	1.1	13.7	-11.1	- 11.9	15.8
5	%diff 1-2	10.3	- 7.4	- 7.4	13.4	16.0	12.1	3.6	- 16.1	22.5
	%diff 2-3	-10.5	21.2	21.2	- 9.9	-12.9	- 9.9	- 2.7	35.4	-25.6
	%diff 1-3	- 1.3	12.2	12.2	2.1	1.0	1.0	0.8	13.6	- 8.9
6	%diff 1-2	0.7	1.4	1.4	11.8	17.8	29.3	- 8.9	0.7	10.2
	%diff 2-3	13.1	20.0	20.0	11.5	15.6	- 8.3	25.9	6.1	- 7.1
	%diff 1-3	13.9	21.7	21.7	24.6	36.1	18.6	14.6	6.9	2.4
7	%diff 1-2	4.9	16.3	16.3	25.6	25.4	8.6	15.4	10.8	8.1
	%diff 2-3	-11.4	-15.0	-15.0	-21.0	-18.6	- 8.2	-11.1	- 4.0	- 7.1
	%diff 1-3	- 7.0	- 1.1	- 1.1	- 0.8	2.1	- 0.3	2.6	6.9	0.4

table continues

Variable	HR	$A\dot{V}O_2$	$R\dot{V}O_2$	$\dot{V}CO_2$	$\dot{V}_E$	TV	F	O2 PULSE	RER
Subject & trial no.	beats/ min	l/min	ml/kg /min	l/min	l/min	l/breath	breaths/ min	ml/beat	
8 %diff 1-2	8.0	5.3	5.3	9.3	18.0	3.8	13.5	- 2.5	3.8
%diff 2-3	- 10.1	- 6.3	- 6.3	- 5.8	- 8.3	- 2.8	- 5.7	4.2	0.5
%diff 1-3	- 2.9	- 1.4	- 1.4	2.9	8.2	0.8	7.0	1.6	4.4
9 %diff 1-2	- 9.0	- 10.9	- 10.9	- 10.2	- 24.0	6.6	-28.8	- 2.1	0.8
%diff 2-3	- 5.0	8.6	8.6	14.8	30.6	- 1.4	32.5	14.2	5.7
%diff 1-3	- 13.5	- 3.3	- 3.3	3.1	- 0.8	5.2	- 5.6	11.9	6.6
10 %diff 1-2	- 9.2	- 10.0	- 10.0	- 10.9	- 12.1	- 2.6	-10.0	- 1.0	- 0.9
%diff 2-3	- 4.7	- 6.4	- 6.4	- 2.1	- 17.2	- 1.4	-16.2	- 1.8	4.6
%diff 1-3	- 14.4	- 15.8	- 15.8	- 12.7	- 27.3	- 4.0	-24.6	- 2.7	3.6
11 %diff 1-2	16.7	9.2	9.2	12.2	8.2	- 7.8	17.5	- 6.4	2.8
%diff 2-3	- 4.5	- 0.6	- 0.6	2.3	- 4.7	25.2	-23.7	4.1	2.9
%diff 1-3	11.5	8.5	8.5	14.7	3.1	15.5	-10.3	- 2.6	5.7
12 %diff 1-2	- 3.7	- 3.2	- 3.2	9.1	12.6	3.1	9.5	0.6	12.6
%diff 2-3	- 0.6	3.8	3.8	- 7.3	- 11.5	- 3.0	- 9.0	4.4	- 10.8
%diff 1-3	- 4.3	0.6	0.6	1.1	- 0.4	- 0.4	- 0.4	5.1	0.5
13 %diff 1-2	6.8	8.2	8.2	3.0	0.7	0.0	0.7	1.3	- 4.9
%diff 2-3	- 2.1	- 10.0	- 10.0	- 8.6	- 8.9	- 2.5	- 6.8	- 8.0	1.6
%diff 1-3	4.6	- 2.6	- 2.6	- 5.9	- 8.3	- 2.5	- 6.1	- 6.8	- 3.4

Appendix 3

Table A3 Individual Mean Values and Percent Difference for Consecutive Trials for some Peak Physiological Responses during the Three GXT Trials

Variable	HR	$\dot{A}VO_2$	$\dot{R}VO_2$	$\dot{V}CO_2$	$\dot{V}_E$	TV	F	O_2 PULSE	RER
Subject & trial no.	beats/ min	l/min	ml/kg/ min	l/min	l/min	l/breath	breaths/ min	ml/beat	
1 m1 (1-2)	122.5	1.10	14.08	1.10	34.85	1.15	30.15	8.99	1.00
m2 (2-3)	133.5	1.30	16.62	1.19	39.35	1.24	31.80	9.78	0.91
%diff m1-m2	9.0	18.2	18.1	8.2	12.9	7.2	5.5	8.8	- 9.0
2 m1 (1-2)	146.5	2.74	27.24	2.66	75.65	1.64	45.80	18.69	0.96
m2 (2-3)	155.5	3.10	30.84	3.15	92.75	1.85	50.00	19.95	1.02
%diff m1-m2	6.1	13.1	13.2	18.4	22.6	12.7	9.2	6.7	6.2
3 m1 (1-2)	180.0	2.68	42.11	2.72	79.15	1.89	41.85	14.88	1.02
m2 (2-3)	181.0	2.82	44.45	2.94	86.60	1.97	44.00	15.62	1.04
%diff m1-m2	0.6	5.2	5.5	8.1	9.4	4.0	5.1	5.0	2.0
4 m1 (1-2)	156.0	2.38	35.08	2.42	83.35	1.64	50.90	15.24	1.02
m2 (2-3)	159.0	2.28	33.60	2.49	83.80	1.75	48.00	14.30	1.09
%diff m1-m2	1.9	- 4.2	- 4.2	2.9	0.5	6.4	- 5.7	- 6.1	7.4
5 m1 (1-2)	163.0	2.85	35.23	3.32	100.20	1.85	54.35	17.56	1.17
m2 (2-3)	162.0	3.03	37.45	3.36	100.65	1.85	54.55	18.86	1.12
%diff m1-m2	- 0.6	6.3	6.3	1.1	0.4	0.5	0.4	7.4	- 4.2
6 m1 (1-2)	144.5	2.14	24.26	2.22	59.45	2.00	30.00	14.77	1.04
m2 (2-3)	154.5	2.36	26.88	2.48	69.30	2.16	32.30	15.28	1.06
%diff m1-m2	- 6.9	10.3	10.8	11.7	16.6	8.1	7.7	3.4	1.9
7 m1 (1-2)	145.5	1.36	18.24	1.32	43.40	1.29	33.60	9.35	0.96
m2 (2-3)	140.5	1.36	18.15	1.32	43.80	1.29	34.00	9.64	0.96
%diff m1-m2	- 3.4	0.0	- 0.5	0.0	1.2	- 0.2	1.2	3.0	0.0

table continues

Variable	HR	$A\dot{V}O_2$	$R\dot{V}O_2$	$\dot{V}CO_2$	$\dot{V}_E$	TV	F	O_2 PULSE	RER
Subject & trial no.	beats/ min	l/min	ml/kg/ min	l/min	l/min	l/breath	breaths/ min	ml/beat	
8 m1 (1-2)	142.5	2.98	29.87	3.22	98.20	2.49	39.50	20.97	1.08
m2 (2-3)	140.5	2.96	29.67	3.26	101.90	2.50	40.80	21.14	1.10
%diff m1-m2	- 1.4	- 0.7	- 0.7	1.2	3.8	0.4	3.3	0.8	1.8
9 m1 (1-2)	127.0	1.16	20.60	1.22	32.20	1.18	27.40	9.08	1.06
m2 (2-3)	118.0	1.14	20.24	1.24	32.05	1.21	26.50	9.62	1.09
%diff m1-m2	- 7.1	- 1.7	- 1.7	1.6	- 0.5	2.5	- 3.3	6.0	2.8
10 m1 (1-2)	156.5	1.48	21.10	1.64	65.10	1.49	43.70	9.44	1.10
m2 (2-3)	145.5	1.36	19.35	1.53	55.65	1.46	38.05	9.31	1.12
%diff m1-m2	- 7.0	- 8.1	- 8.3	- 6.7	- 14.5	- 2.0	-12.9	- 1.4	1.8
11 m1 (1-2)	104.0	1.72	22.19	1.66	50.70	1.24	41.00	16.53	0.96
m2 (2-3)	109.5	1.78	23.09	1.77	51.45	1.34	39.05	16.31	0.99
%diff m1-m2	5.3	3.5	4.1	6.6	3.6	8.1	- 4.8	- 1.4	3.1
12 m1 (1-2)	183.5	3.28	37.33	3.62	110.45	2.33	47.55	17.86	1.10
m2 (2-3)	179.5	3.29	37.44	3.64	110.25	2.32	47.45	18.31	1.10
%diff m1-m2	- 2.2	0.3	0.3	0.6	- 0.2	- 0.0	- 0.2	2.5	0.0
13 m1 (1-2)	136.5	2.00	20.32	2.06	59.30	2.01	29.50	14.64	1.04
m2 (2-3)	139.5	1.98	20.06	2.00	56.85	1.99	28.60	14.14	1.02
%diff m1-m2	2.2	- 1.0	- 1.2	- 2.9	- 4.1	- 1.2	- 3.0	- 3.4	- 1.9

APPENDIX 4

Scatterplots of Selected Peak Physiological Responses

Pages 110-120

Figure 2
Heart Rate, beats/min
Trial 1 vs 2, r = 0.91, p<0.001

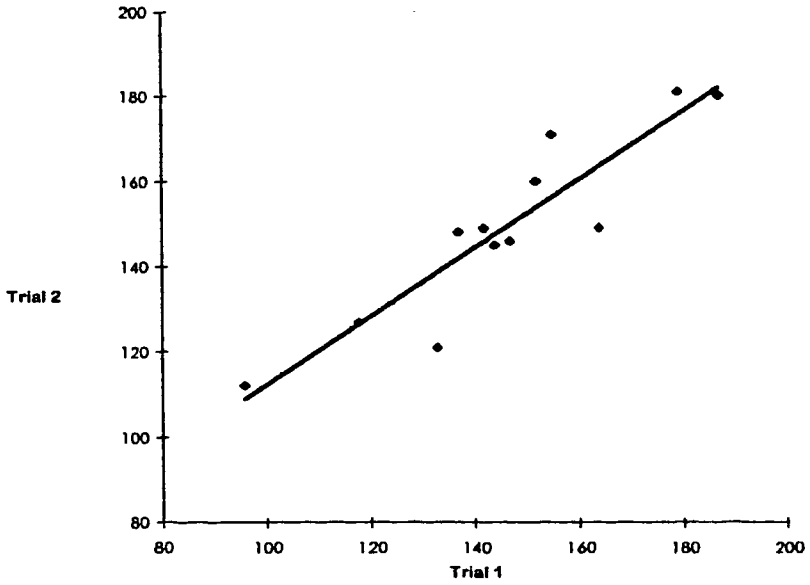


Figure 3
Heart Rate, beats/min
Trial 2 vs 3, r=0.86, p<0.001

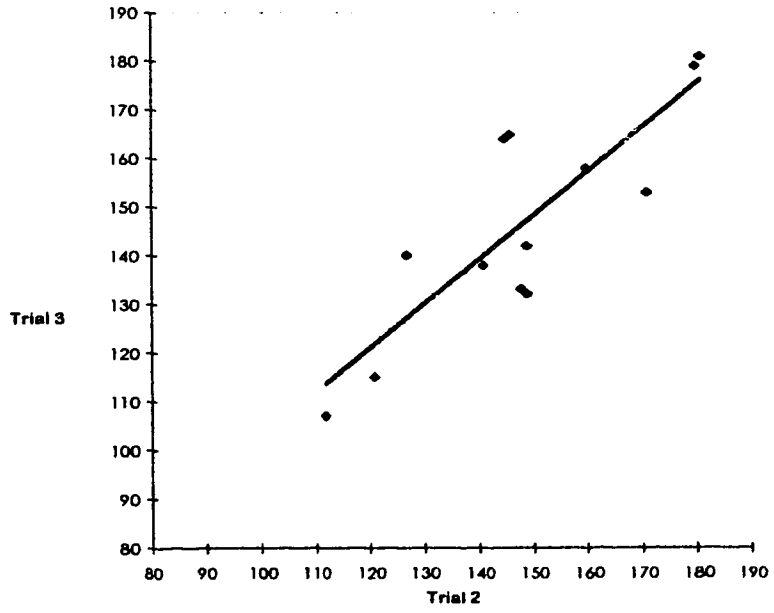


Figure 4
Heart Rate, beats/min
Trials 1 vs 3, r=0.90, p<0.001

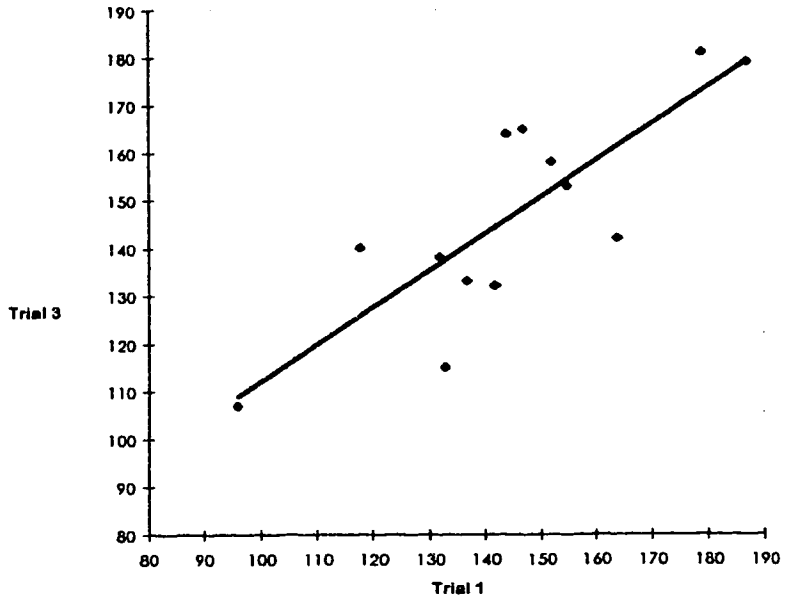


Figure 5
Oxygen Uptake ($\dot{V}O_2$), L/min
Trial 1 vs 2, r=0.97, p<0.001

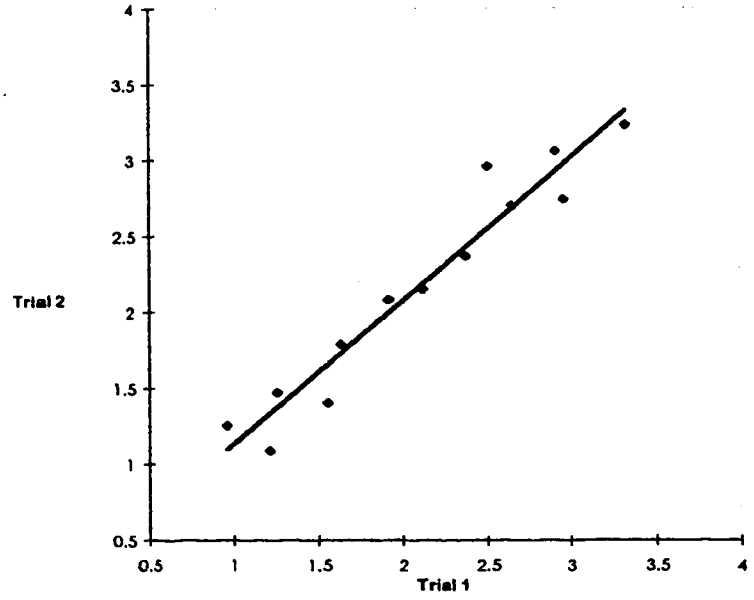


Figure 6
Oxygen Uptake ($\dot{V}O2$), L/min
Trial 2 vs 3, r=0.96, p<0.001

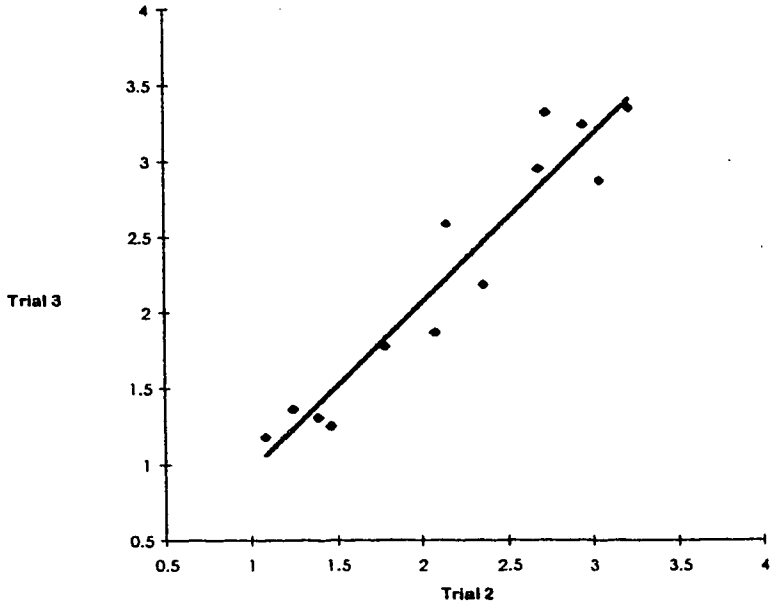


Figure 7
Oxygen Uptake ($\dot{V}O2$), L/min
Trial 1 vs 3, r=0.94, p<0.001

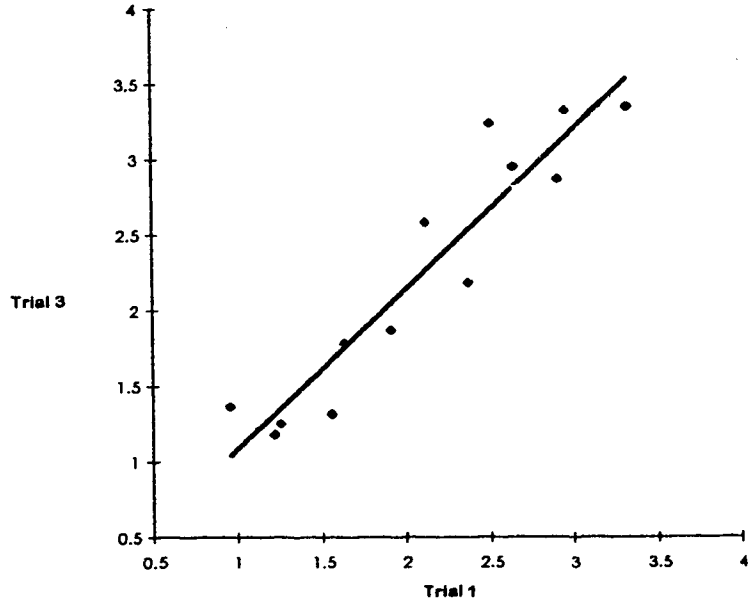


Figure 8
Carbon Dioxide Production ($\dot{V}CO2$), L/min
Trials 1 vs 2, r=0.96, p<0.001

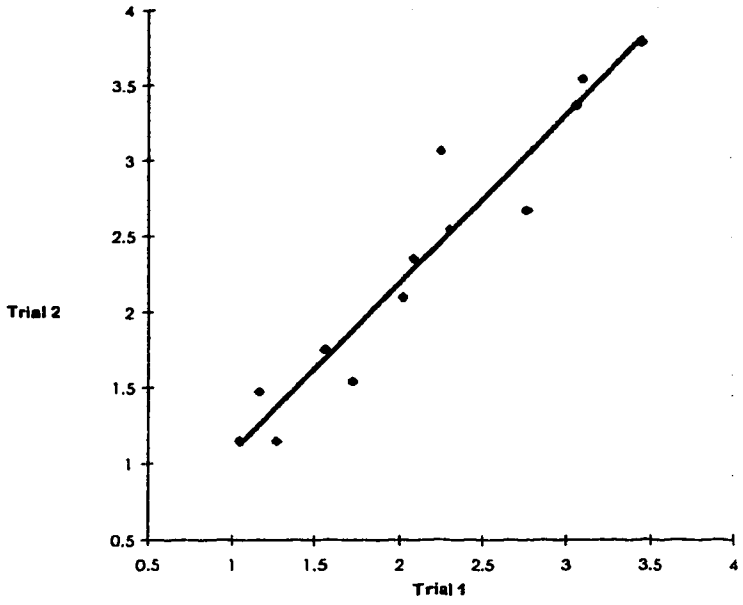


Figure 9
Carbon Dioxide Production ($\dot{V}CO2$), L/min
Trial 2 vs 3, r=0.93, p<0.001

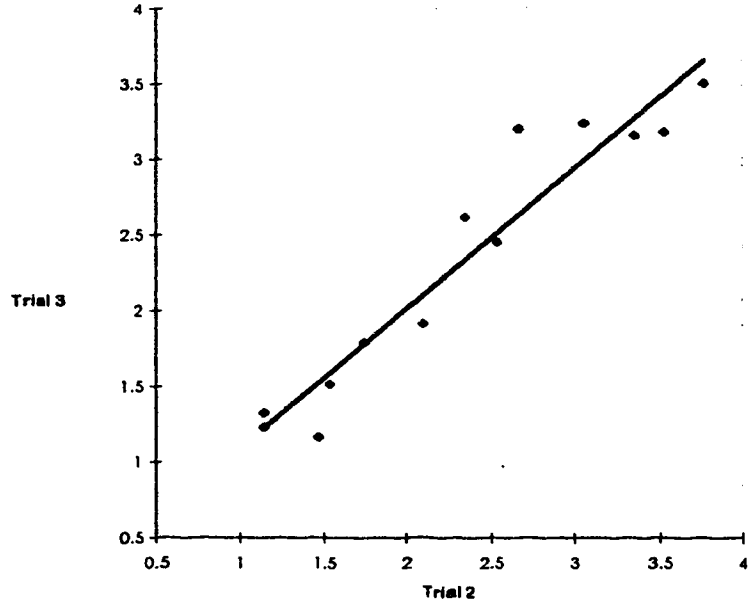


Figure 10
Carbon Dioxide Production ($\dot{V}CO2$), L/min
Trial 1 vs 3, r=0.93, p<0.001

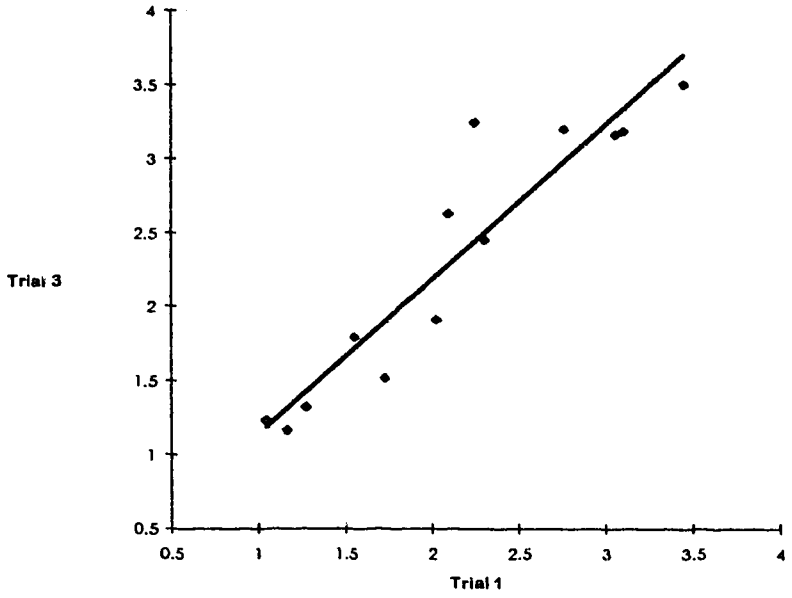


Figure 11
Minute Ventilation ($\dot{V}E$), L/min
Trial 1 vs 2, r=0.96, r<0.001

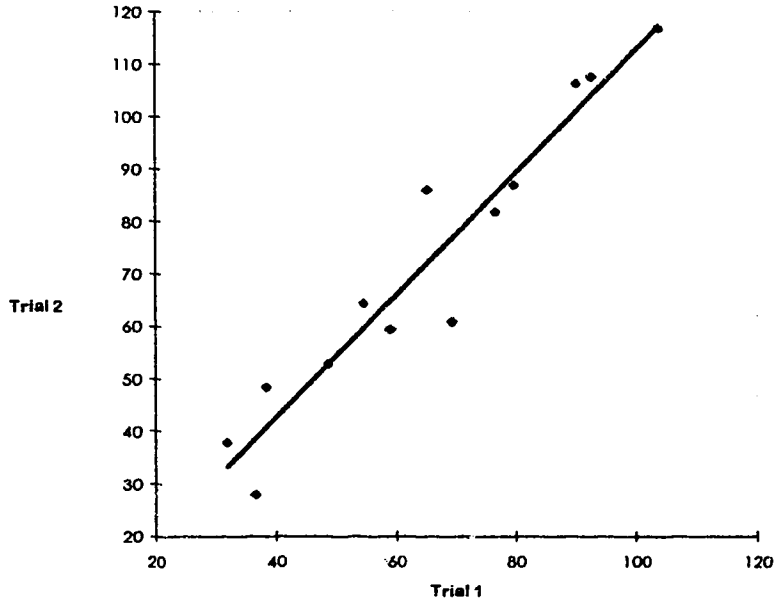


Figure 12
Minute Ventilation ($\dot{V}E$), L/min
Trial 2 vs 3, r=0.94, p<0.001

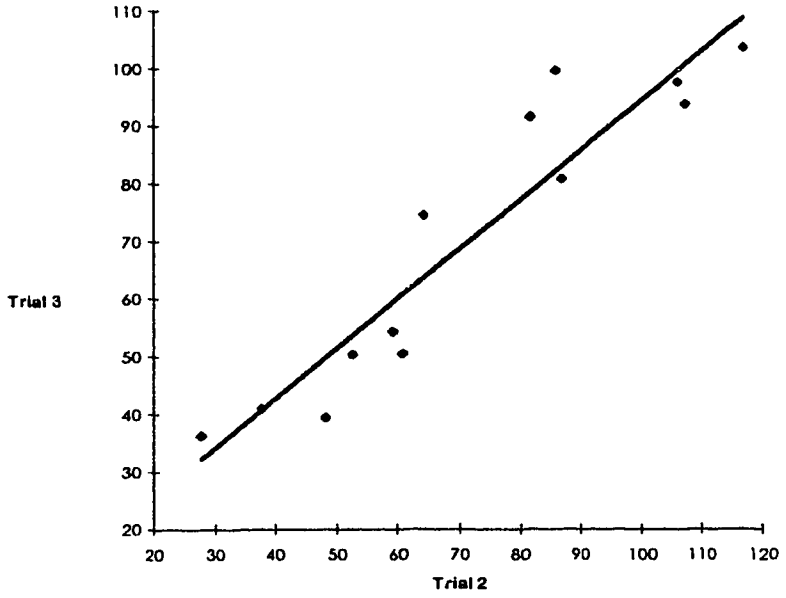


Figure 13
Minute Ventilation ($\dot{V}E$), L/min
Trial 1 vs 3, r=0.87, p<0.001

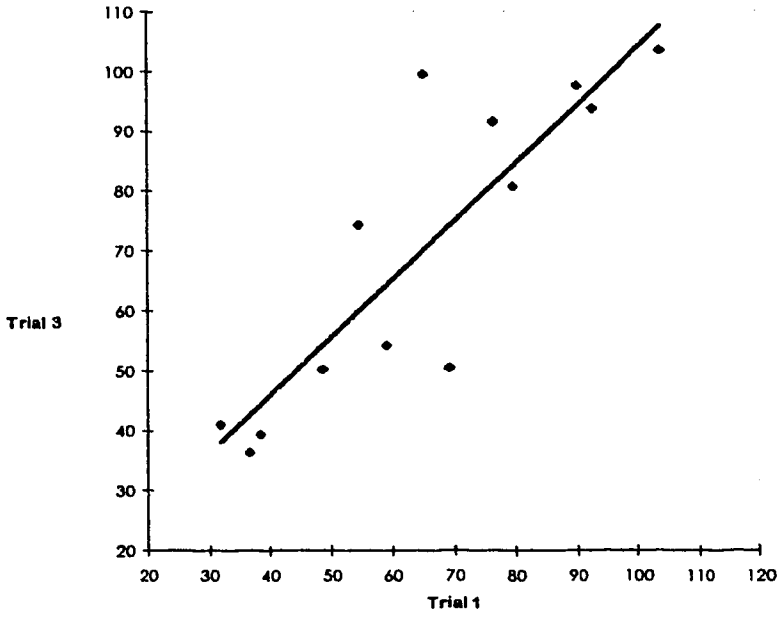


Figure 14
Tidal Volume (TV), L/breath
Trial 1 vs 2, r=0.94, p<0.001

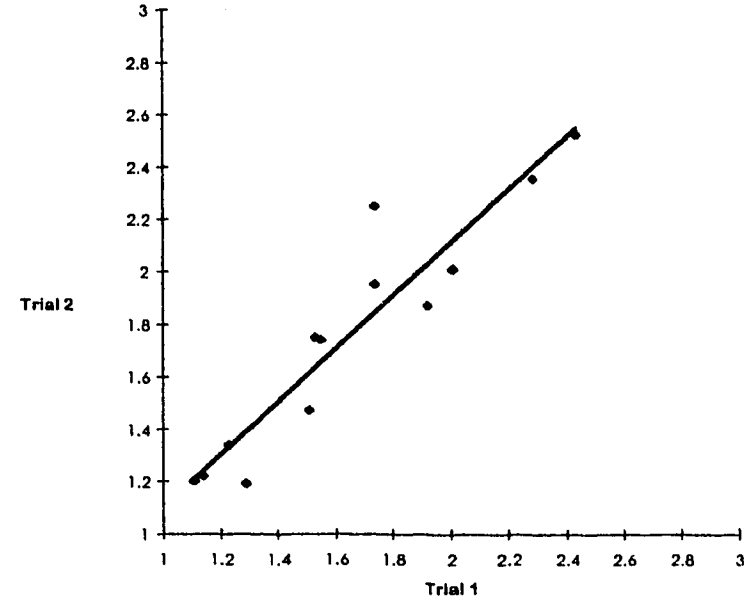


Figure 15
Tidal Volume (TV), L/breath
Trial 2 vs 3, r=0.94, p<0.001

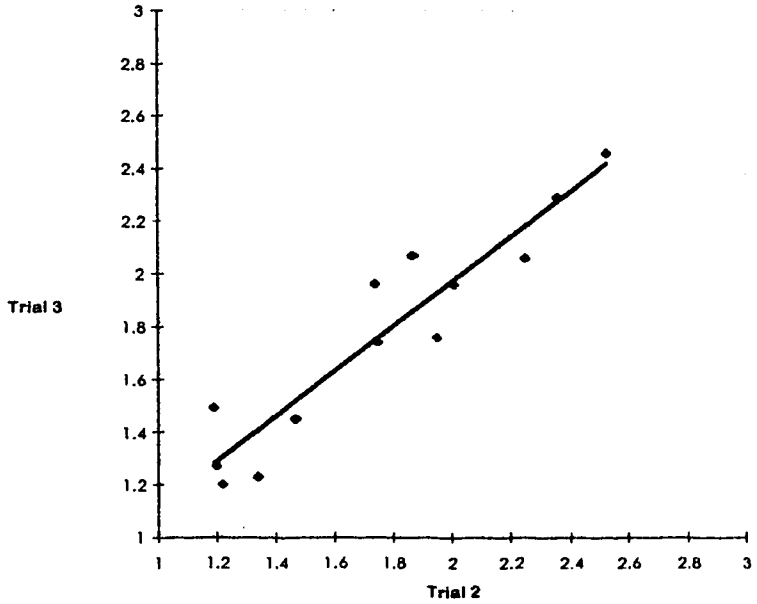


Figure 16
Tidal Volume (TV), L/breath
Trial 1 vs 3, r=0.94, p<0.001

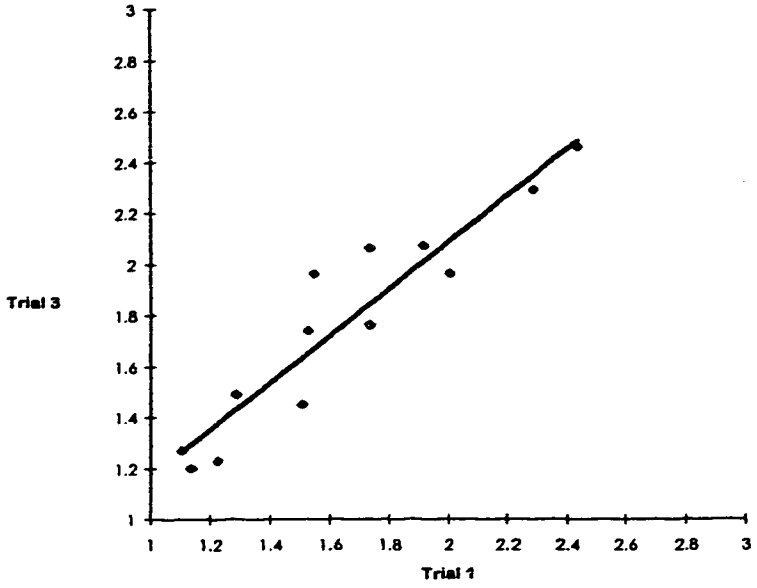


Figure 17
Breathing Rate (F), breaths/min
Trial 1 vs 2, r=0.87, p<0.001

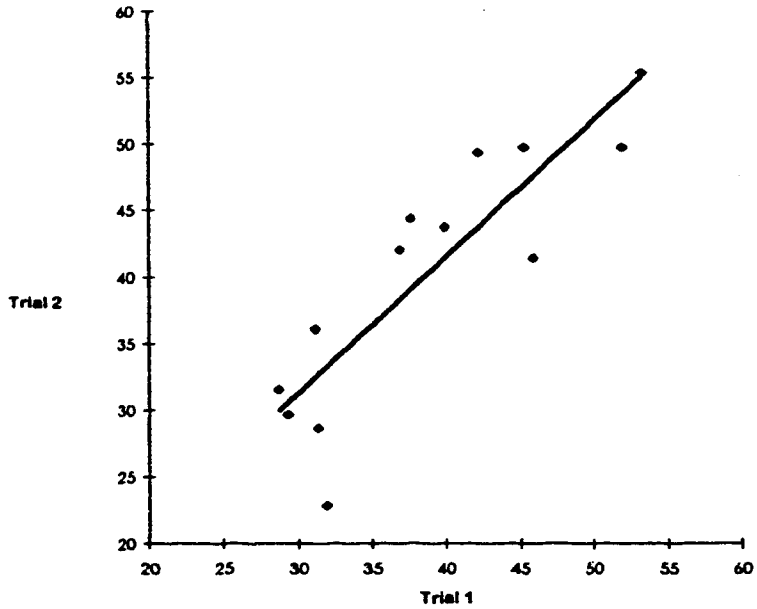


Figure 18
Breathing Rate (F), breaths/min
Trial 2 vs 3, r=0.86, p<0.001

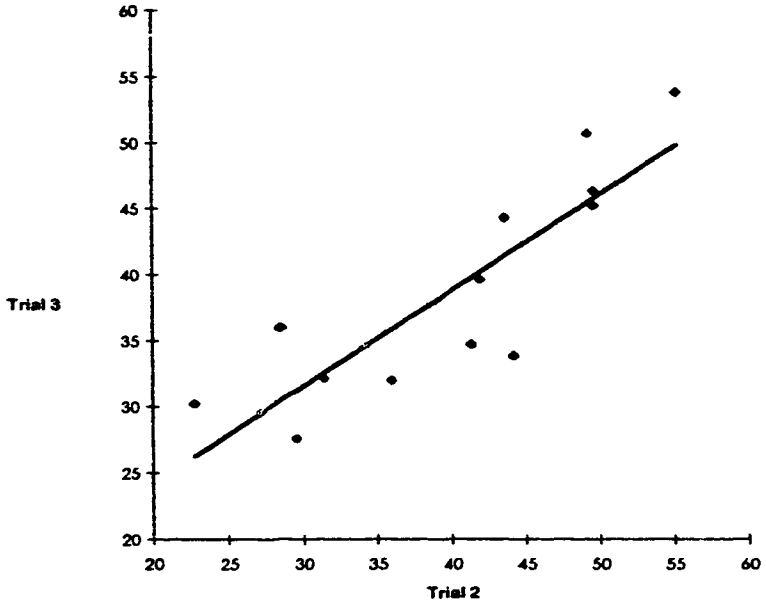


Figure 19
Breathing Rate (F), breaths/min
Trial 1 vs 3, r=0.82, p<0.001

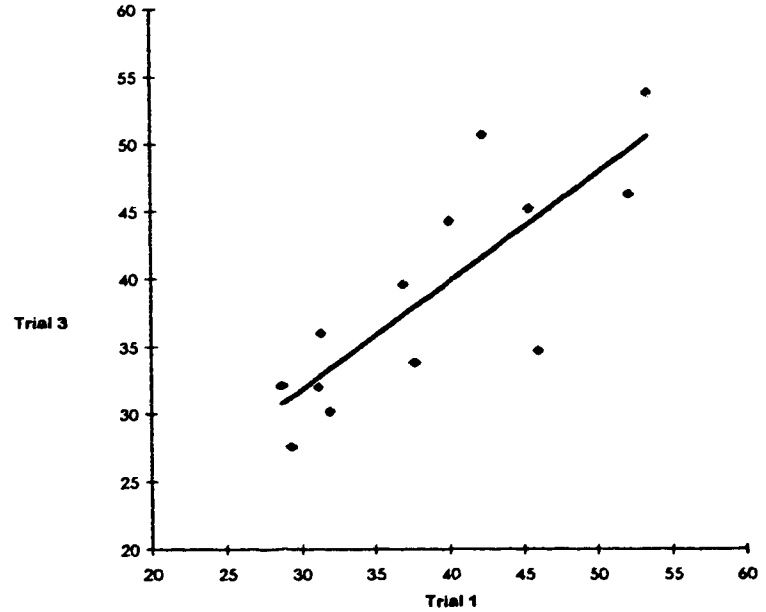


Figure 20
Oxygen Pulse, ml/beat
Trial 1 vs 2, r=0.94, p<0.001

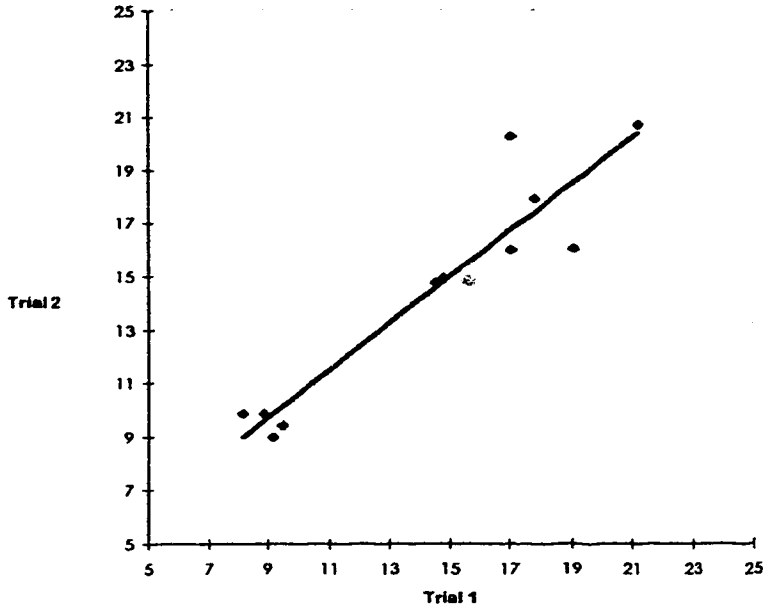


Figure 21
Oxygen Pulse, ml/beat
Trial 2 vs 3, r=0.92, p<0.001

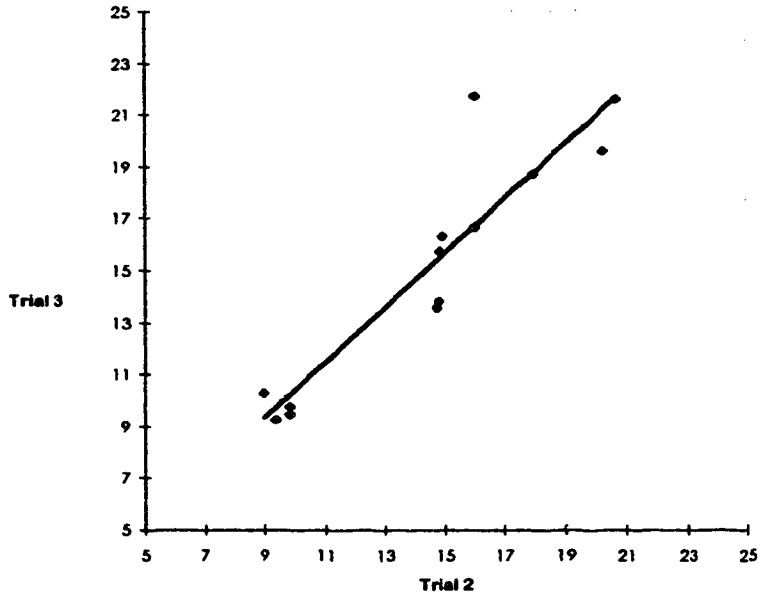
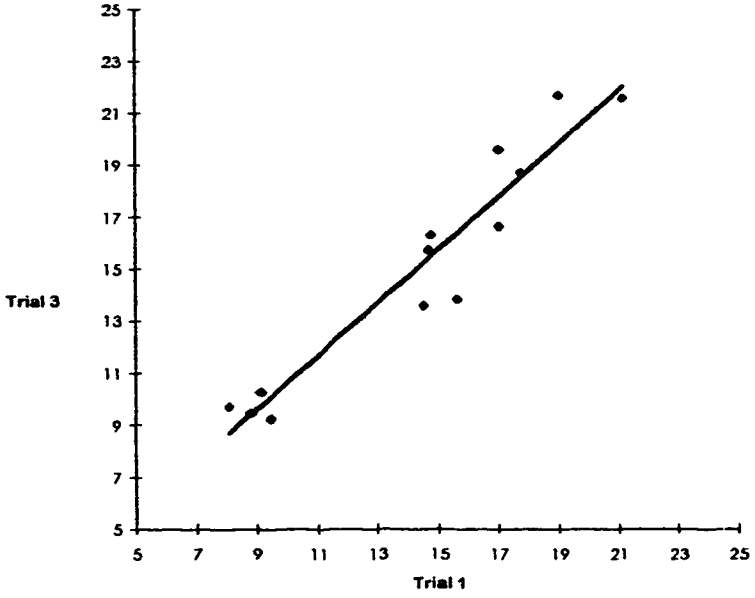


Figure 22
Oxygen Pulse, ml/beat
Trial 1 vs 3, r=0.96, p<0.001



Appendix 5

Table A5 Individual Values for Some Physiological Responses During Determination of Cardiac Output

Variable	HR	$\dot{V}O_2$	$\dot{V}CO_2$	O_2 PULSE	$\dot{V}_E$	Q	QI	SV	SVI	$(a\text{-}v)O_2$ diff	TV
Subject & trial	beats/ min	l/min	ml/min	ml/beat	l/min	l/min	l/min/ sq.m	ml/ beat	ml/beat/ sq.m	ml / 100 ml	ml/ breath
1 trial 1a	104	0.66	627	6.36	19.8	9.7	5.05	93	48.58	6.84	995
trial 1b	111	0.[illegible]	728	6.98	24.1	11.76	6.13	106	55.19	6.59	998
trial 2a	104	0.73	712	7.02	23.0	10.52	5.48	101	52.70	6.94	1017
trial 2b	111	0.72	683	6.52	22.2	10.16	5.29	92	47.67	7.13	970
2 trial 1a	143	1.77	1960	12.38	57.2	18.79	8.54	131	59.72	9.42	1420
trial 1b	148	1.89	2127	12.74	65.6	19.72	8.96	133	60.56	9.57	1599
trial 2a	142	1.80	1756	12.63	51.5	18.10	8.23	127	57.93	9.91	1336
trial 2b	143	1.75	1667	12.22	50.3	17.79	8.09	124	56.55	9.82	1250
3 trial 1a	150	1.58	1742	10.51	51.5	15.18	8.88	101	59.17	10.38	1489
trial 1b	158	1.58	1751	10.03	52.0	15.63	9.14	99	57.84	10.14	1637
trial 2a	148	1.29	1656	8.71	49.4	17.03	9.96	115	67.30	7.57	1432
trial 2b	155	1.34	1829	8.63	54.7	18.17	10.63	117	68.56	7.36	1518
4 trial 1a	129	1.36	1469	10.50	44.7	16.24	9.02	126	69.95	8.34	1551
trial 1b	130	1.46	1602	11.26	51.9	13.70	7.61	105	58.53	10.69	1392
trial 2a	120	1.28	1412	10.63	39.9	13.55	7.53	113	62.75	9.41	1416
trial 2b	133	1.35	1383	10.14	42.3	12.60	7.00	95	52.65	10.69	1324
5 trial 1a	114	1.01	970	8.86	28.7	11.12	5.64	98	49.51	9.08	1368
trial 1b	109	1.33	1293	12.17	35.7	13.91	7.02	128	64.77	9.53	1400
trial 2a	108	1.50	1330	13.93	36.1	15.47	7.85	143	72.70	9.72	1663
trial 2b	112	1.40	1391	12.53	37.8	16.68	8.47	149	75.58	8.41	1955
6 trial 1a	120	1.50	1833	12.49	50.9	17.48	8.33	146	69.38	8.57	2009
trial 1b	118	1.40	1569	11.83	46.0	15.81	7.53	134	63.81	8.83	2043
trial 2a	115	1.18	1210	10.22	35.7	12.90	6.14	112	53.42	9.11	1535
trial 2b	121	1.30	1329	10.75	35.8	14.14	6.73	117	55.66	9.20	1895
7 trial 1a	116	0.79	776	6.80	27.9	8.59	4.62	74	39.81	9.19	982
trial 1b	122	0.81	812	6.62	29.5	8.49	4.57	70	37.43	9.51	1020
trial 2a	118	0.78	670	6.57	28.5	7.49	4.02	63	34.11	10.35	875
trial 2b	120	0.65	569	5.42	23.9	6.73	3.62	56	30.15	9.66	814

table continues

Variable	HR	$\dot{V}O_2$	$\dot{V}CO_2$	O_2 PULSE	$\dot{V}_E$	Q	QI	$S\dot{V}$	SVI	$(a\text{-}v)O_2$ diff	TV
Subject & trial	beats/ min	l/min	ml/min	ml/beat	l/min	l/min	l/min/ sq.m	ml/ beat	ml/beat/ sq.m	ml / 100 ml	ml/ breath
8 trial 1a	121	1.90	1969	15.71	60.0	17.44	8.26	144	68.30	10.90	1862
trial 1b	124	1.82	1799	14.67	58.7	15.96	7.56	129	61.00	11.40	1723
trial 2a	84	1.59	1573	18.95	47.6	14.60	6.92	174	82.35	10.91	1520
trial 2b	106	1.52	1455	14.39	50.1	13.56	6.43	128	60.62	11.25	1481
9 trial 1a	92	0.56	577	5.82	18.1	9.77	6.27	106	68.14	5.47	884
trial 1b	95	0.53	612	5.56	19.0	8.92	5.72	94	60.17	5.92	914
trial 2a	101	0.66	677	6.53	19.5	13.92	9.92	138	88.35	4.74	986
trial 2b	101	0.68	661	6.71	20.4	10.04	6.44	99	63.72	6.75	917
10 trial 1a	122	0.84	896	6.84	29.3	9.75	5.60	80	45.91	8.57	1139
trial 1b	130	0.90	963	6.93	33.8	10.22	5.88	79	45.19	8.81	1216
trial 2a	124	0.87	947	7.00	32.6	10.50	6.04	85	48.69	8.26	1092
trial 2b	132	0.85	925	6.47	34.1	9.23	5.31	70	40.20	9.25	1094
11 trial 1a	89	0.95	908	10.64	26.6	16.31	8.72	183	98.02	5.81	1550
trial 1b	95	0.97	979	10.17	31.3	14.18	7.58	149	79.82	6.81	1445
trial 2a	91	0.97	932	10.67	35.3	10.02	5.36	110	58.87	9.69	1467
trial 2b	93	0.84	811	9.08	32.8	11.30	6.04	121	64.95	7.47	1470
12 trial 1a	101	1.69	1762	16.62	50.1	15.31	7.43	152	73.61	10.96	1558
trial 1b	127	1.79	1768	14.09	49.4	16.66	8.09	131	63.68	10.74	1683
trial 2a	133	1.77	1929	13.29	54.1	20.16	9.79	152	73.59	8.77	1972
trial 2b	141	1.87	1863	13.26	51.8	19.06	9.25	135	65.61	9.81	2029
13 trial 1a	96	0.87	861	9.03	26.3	9.45	4.39	98	45.78	9.18	1400
trial 1b	99	0.92	916	9.25	28.4	11.07	5.15	112	52.00	8.28	1453
trial 2a	99	1.08	995	10.90	32.4	10.30	4.79	104	48.40	10.47	1460
trial 2b	99	1.09	1014	11.04	32.6	10.55	4.91	107	49.58	10.36	1466

Appendix 6

Table A6
Percent Difference for Some Variables Between Trials to Determine Cardiac Output

Variable		HR	$\dot{V}O_2$	$\dot{V}CO_2$	O_2 PULSE	$\dot{V}_E$	Q	QI	SV	SVI	$(a\text{-}v)O_2$ diff	TV
Subject & trials		beats/ min	l/min	ml/min	ml/beat	l/min	l/min	l/min/ sq.m	ml/ beat	ml/beat /sq.m	ml / 100 ml	ml/ breath
1	%diff 1a-1b	6.7	18.2	16.1	9.7	21.7	21.2	21.4	14.0	13.6	- 3.6	0.3
	%diff 2a-2b	6.7	- 1.4	- 4.1	- 7.1	- 3.5	- 3.4	- 3.5	- 8.9	- 9.5	2.7	- 4.6
	%diff 1a-2a	0.0	10.6	13.6	0.6	16.2	8.4	8.5	8.6	8.5	1.5	2.2
	%diff 1b-2b	0.0	- 7.7	- 6.2	- 6.2	- 7.9	- 13.6	- 13.7	- 13.2	- 13.6	8.2	- 2.8
2	%diff 1a-1b	3.5	6.8	8.5	2.9	14.7	4.9	4.9	1.5	1.4	1.6	12.6
	%diff 2a-2b	0.7	- 2.8	- 5.1	- 3.2	- 2.3	- 1.7	- 1.7	- 2.4	- 2.4	- 0.9	- 6.4
	%diff 1a-2a	- 0.7	1.7	- 10.4	2.0	- 10.0	- 3.7	- 3.6	- 3.0	- 3.0	5.2	- 5.9
	%diff 1b-2b	- 3.4	- 7.4	- 21.6	- 4.1	- 23.3	- 9.8	- 9.7	- 6.8	- 6.6	2.6	- 21.8
3	%diff 1a-1b	5.3	0.0	0.5	- 4.6	1.0	3.0	2.9	2.0	- 2.2	- 2.3	9.9
	%diff 2a-2b	0.7	3.9	10.4	- 0.9	10.7	6.7	6.7	1.7	1.9	- 2.8	6.0
	%diff 1a-2a	- 1.3	- 18.4	- 4.9	-17.1	- 4.1	12.2	12.2	13.9	13.7	-27.1	- 3.8
	%diff 1b-2b	- 1.9	- 15.2	4.5	-14.0	5.2	16.2	16.3	18.2	18.5	-27.4	- 7.3
4	%diff 1a-1b	0.8	7.4	9.1	7.2	16.1	- 15.6	- 15.6	16.7	- 16.3	28.2	- 10.2
	%diff 2a-2b	10.8	5.5	- 2.1	- 4.6	6.0	- 7.0	- 7.0	- 15.9	- 16.1	13.6	- 6.5
	%diff 1a-2a	- 7.0	- 5.9	- 3.9	1.2	- 10.7	- 16.6	- 16.5	- 10.3	- 10.3	12.8	- 8.7
	%diff 1b-2b	2.3	- 7.5	- 13.7	- 9.9	- 18.5	- 8.0	- 8.0	- 9.5	- 10.0	0.0	- 4.9
5	%diff 1a-1b	- 4.4	31.7	33.3	37.4	24.4	25.1	24.5	31.6	30.8	5.0	2.3
	%diff 2a-2b	3.7	- 6.7	4.6	-10.0	4.7	7.8	7.9	4.2	4.0	- 13.5	17.6
	%diff 1a-2a	- 5.3	48.5	37.1	14.5	25.8	39.1	39.2	45.9	46.8	7.0	21.6
	%diff 1b-2b	2.8	5.3	7.6	3.0	5.9	19.9	20.6	16.4	16.7	- 11.8	39.6
6	%diff 1a-1b	- 1.7	- 6.7	- 14.4	- 5.3	- 9.6	- 9.6	- 9.6	- 8.2	- 8.0	3.0	1.7
	%diff 2a-2b	5.2	10.2	9.8	4.9	0.3	9.6	9.6	4.5	4.2	1.0	23.4
	%diff 1a-2a	- 4.2	- 21.3	- 34.0	-18.2	- 29.9	- 26.2	- 26.3	- 23.3	- 23.0	6.3	- 23.6
	%diff 1b-2b	2.5	- 7.1	- 15.3	- 9.1	- 22.2	- 10.6	- 10.6	- 12.7	- 12.8	4.2	- 7.2

table continues

Variable		HR	$\dot{V}O_2$	$\dot{V}CO_2$	O_2 PULSE	$\dot{V}_E$	Q	QI	SV	SVI	$(a\text{-}v)O_2$ diff	TV
Subject & trials		beats/ min	l/min	ml/min	ml/beat	l/min	l/min	l/min /sq.m	ml/ beat	ml/beat /sq.m	ml / 100 ml	ml/ breath
7	%diff 1a-1b	5.2	2.5	4.6	2.6	5.7	- 1.2	- 1.1	5.4	- 6.0	3.5	3.9
	%diff 2a-2b	1.7	- 16.7	- 15.1	-17.5	- 16.1	- 10.1	- 10.0	- 11.1	- 11.6	- 6.7	- 7.0
	%diff 1a-2a	1.7	- 1.3	- 13.7	- 3.4	2.2	- 12.8	- 13.0	- 14.9	- 14.3	12.6	- 10.9
	%diff 1b-2b	- 1.6	- 19.8	- 29.9	-18.1	- 19.0	- 20.7	- 20.8	- 20.0	- 19.4	1.6	- 20.2
8	%diff 1a-1b	2.5	- 4.2	- 8.6	- 6.6	- 2.2	- 8.5	- 8.5	- 10.4	- 10.7	4.6	- 7.5
	%diff 2a-2b	26.2	- 4.4	- 7.5	- 24.1	5.2	- 7.1	- 7.1	- 26.4	- 26.4	3.1	- 2.6
	%diff 1a-2a	-30.6	- 16.3	- 20.1	20.6	- 20.7	- 16.3	- 16.2	20.8	20.6	0.1	- 18.4
	%diff 1b-2b	-14.5	- 18.1	- 19.1	- 1.9	- 14.6	- 15.0	- 14.9	- 0.8	- 0.6	- 1.3	- 14.0
9	%diff 1a-1b	3.3	- 5.4	6.1	- 4.5	5.0	- 8.7	- 8.8	- 11.3	- 11.7	8.2	3.4
	%diff 2a-2b	0.0	3.0	- 2.4	2.8	4.6	- 27.9	- 35.1	- 28.3	- 27.9	42.4	- 7.0
	%diff 1a-2a	9.8	17.9	17.3	12.2	7.7	42.5	58.2	30.2	29.7	- 13.3	11.5
	%diff 1b-2b	6.3	28.3	8.0	20.7	7.4	12.6	12.6	5.3	5.9	14.0	0.3
10	%diff 1a-1b	6.6	7.1	7.5	1.3	15.4	4.8	5.0	- 1.2	- 1.6	2.8	6.8
	%diff 2a-2b	6.4	- 2.3	- 2.3	- 7.6	4.6	- 12.1	- 12.1	- 17.6	- 17.4	12.2	0.2
	%diff 1a-2a	1.6	3.6	5.7	2.3	11.3	7.7	7.9	6.2	6.1	- 3.6	- 4.1
	%diff 1b-2b	1.5	- 5.6	- 3.9	- 6.6	0.9	- 9.7	- 9.7	- 11.4	- 11.0	5.0	- 10.0
11	%diff 1a-1b	6.7	2.1	7.8	- 4.4	17.7	- 13.1	- 13.1	- 18.6	- 18.6	17.2	- 6.8
	%diff 2a-2b	2.2	- 13.4	- 13.0	- 14.9	- 7.1	12.7	12.7	10.0	10.3	- 22.9	0.2
	%diff 1a-2a	2.2	2.1	2.6	0.3	32.7	- 38.6	- 38.5	- 39.9	- 39.9	66.8	- 5.4
	%diff 1b-2b	- 2.1	- 13.1	17.2	- 10.7	4.8	- 20.3	- 20.3	- 18.8	- 18.6	9.7	1.7
12	%diff 1a-1b	25.7	5.9	0.3	- 15.2	- 1.4	8.8	8.9	- 13.8	- 13.5	- 2.0	8.0
	%diff 2a-2b	6.0	5.6	- 3.4	- 0.2	- 4.2	- 5.5	- 5.5	- 11.2	- 10.8	11.9	2.9
	%diff 1a-2a	31.7	4.7	9.5	- 20.0	8.0	31.7	31.8	0.0	0.1	- 20.0	26.6
	%diff 1b-2b	11.0	4.5	5.4	- 5.9	4.9	14.4	14.3	3.0	3.0	- 8.7	20.6
13	%diff 1a-1b	3.1	5.7	6.4	2.4	8.0	17.1	17.3	14.3	13.6	- 9.8	3.8
	%diff 2a-2b	0.0	0.9	1.9	1.3	0.6	2.4	2.5	2.9	2.4	- 1.0	0.4
	%diff 1a-2a	3.1	24.1	15.6	20.7	23.2	9.0	9.1	6.1	5.7	14.0	4.3
	%diff 1b-2b	0.0	18.5	10.7	19.4	17.2	- 4.7	- 4.7	- 4.5	- 4.6	25.1	0.9

Appendix 7

Table A7
Percent Difference Between the Means of Each Days Two Maneuvers for Some Variables During Determination of Cardiac Output

Variable	HR	$\dot{V}O_2$	$\dot{V}CO_2$	O_2 PULSE	$\dot{V}_E$	Q	QI	SV	SVI	(a-v) O_2 diff	TV
Subject & trial	beats/ min	l/min	ml/min	ml/beat	l/min	l/min	l/min/ sq.m	ml/beat	ml/beat / sq.m	ml / 100 ml	ml/ breath
1 mean 1a-1b	107.5	0.72	677.5	6.67	21.95	10.73	5.59	99.5	51.88	6.72	996.5
mean 2a-2b	107.7	0.72	697.5	6.77	22.60	10.34	5.38	96.5	50.18	7.04	993.5
%diff X - Y	0.0	0.7	3.7	1.5	3.0	- 3.6	- 3.7	- 3.0	- 3.3	4.8	- 0.3
2 mean 1a-1b	145.5	1.83	2043.5	12.56	61.40	19.26	8.75	132.0	60.14	9.5	1509.5
mean 2a-2b	142.5	1.78	1711.5	12.42	50.90	17.94	8.16	125.5	57.24	9.86	1293.0
%diff X - Y	- 2.1	- 3.0	- 16.2	1.1	- 17.1	- 6.8	- 6.7	- 4.9	- 4.8	3.9	- 14.3
3 mean 1a-1b	154.0	1.58	1746.5	10.27	51.75	15.40	9.01	100.0	58.50	10.26	1563.0
mean 2a-2b	151.5	1.32	1742.5	8.67	52.05	17.60	10.30	116.0	67.93	7.46	1475.0
%diff X - Y	- 1.6	- 16.8	- 0.2	- 15.6	0.6	14.2	14.26	16.0	16.1	- 27.2	- 5.6
4 mean 1a-1b	129.5	1.41	1535.5	10.88	[illegible]	14.97	8.32	115.5	64.24	9.52	1471.5
mean 2a-2b	126.5	1.32	1397.5	10.38	[illegible]	13.08	7.26	104.0	57.70	10.05	1370.0
%diff X - Y	- 2.3	- 6.7	- 9.0	- 4.6	- 14.9	- 12.7	- 12.6	- 10.0	- 10.2	5.6	- 6.9
5 mean 1a-1b	111.5	1.17	1131.5	10.52	32.20	12.52	6.33	113.0	57.14	9.30	1384.0
mean 2a-2b	110.0	1.45	1360.5	13.23	36.95	16.08	8.16	146.0	74.14	9.56	1809.0
%diff X - Y	1.4	23.9	20.2	25.8	14.8	28.4	28.9	29.2	29.8	2.8	30.7
6 mean 1a-1b	119.0	1.45	1701.0	12.16	48.45	16.64	7.93	140.0	66.60	8.70	2026.0
mean 2a-2b	118.0	1.24	1269.5	10.48	35.75	13.52	6.44	114.5	54.54	9.16	1715.0
%diff X - Y	- 0.8	-14.5	- 25.4	- 13.8	- 26.2	- 18.8	- 18.8	- 18.2	- 18.1	5.2	- 15.4
7 mean 1a-1b	119.0	0.80	794.0	6.71	28.7	8.54	4.60	72.0	38.62	9.35	1001.0
mean 2a-2b	119.0	0.72	619.5	6.00	26.2	7.11	3.82	59.5	32.13	10.00	884.5
%diff X - Y	0.0	-10.6	- 22.0	- 10.7	- 8.7	- 16.7	- 16.9	- 17.4	- 16.8	7.0	- 15.6

table continues

Variable	HR	$\dot{V}O_2$	$\dot{V}CO_2$	O_2 PULSE	$\dot{V}_E$	Q	QI	SV	SVI	(a-v) O_2 diff	TV
Subject & trial	beats/ min	l/min	ml/min	ml/beat	l/min	l/min	l/min/ sq.m	ml/beat	ml/beat/ sq.m	ml / 100 ml	ml/ breath
8 mean 1a-1b	122.5	1.86	1884.0	15.19	59.35	16.70	7.91	136.5	64.65	11.15	1792.5
mean 2a-2b	95.0	1.56	1514.0	16.67	48.85	14.08	6.68	151.0	71.48	11.08	1500.5
%diff X - Y	- 22.4	- 16.4	- 19.6	9.7	- 17.7	- 15.7	- 15.6	10.6	10.6	- 0.6	- 16.3
9 mean 1a-1b	93.5	0.54	594.5	5.69	18.55	9.34	6.00	100.0	64.16	5.70	899.0
mean 2a-2b	101.0	0.67	669.0	6.62	19.95	11.98	8.18	118.5	76.04	5.75	951.5
%diff X - Y	8.0	22.9	12.5	16.3	7.5	28.2	36.4	18.5	18.5	0.9	5.8
10 mean 1a-1b	126.0	0.87	929.5	6.88	31.55	9.98	5.74	79.5	45.55	8.69	1177.5
mean 2a-2b	128.0	0.86	936.0	6.74	33.35	9.86	5.68	77.5	44.44	8.76	1093.0
%diff X - Y	1.6	- 1.1	0.7	- 2.2	5.7	- 1.2	- 1.1	- 2.5	- 2.4	0.7	- 7.2
11 mean 1a-1b	92.0	0.96	943.5	10.40	28.95	15.24	8.15	166.0	88.92	6.31	1497.5
mean 2a-2b	92.0	0.90	871.5	9.88	34.05	10.66	5.70	115.5	61.91	8.58	1468.5
%diff X - Y	0.0	- 5.7	- 7.6	- 5.1	17.6	- 30.1	- 30.1	- 30.4	- 30.4	36.0	- 1.4
12 mean 1a-1b	114.0	1.74	1765.0	15.36	49.75	15.98	7.76	141.0	68.64	10.85	1602.5
mean 2a-2b	137.0	1.82	1896.5	13.28	52.95	19.61	9.52	143.5	69.60	9.29	2000.5
%diff X - Y	20.2	4.9	7.4	-13.6	6.4	22.7	22.7	1.8	1.4	- 14.4	23.4
13 mean 1a-1b	97.5	0.90	888.5	9.14	27.35	10.26	4.77	105.0	48.89	9.83	1426.5
mean 2a-2b	99.0	1.09	1004.5	10.97	32.50	10.42	4.85	105.5	48.99	10.42	1463.0
%diff X - Y	1.5	21.2	13.0	20.0	18.8	1.6	1.7	0.5	0.2	6.0	2.6

X = mean of maneuvers 1a & 1b, Y = mean of maneuvers 2a & 2b

APPENDIX 8

Scatterplots of Selected Submaximal Responses

Pages 128 - 137

Figure 23
Heart Rate, beats/ min
Maneuver 1a vs 1b, r= 0.93, p< 0.01

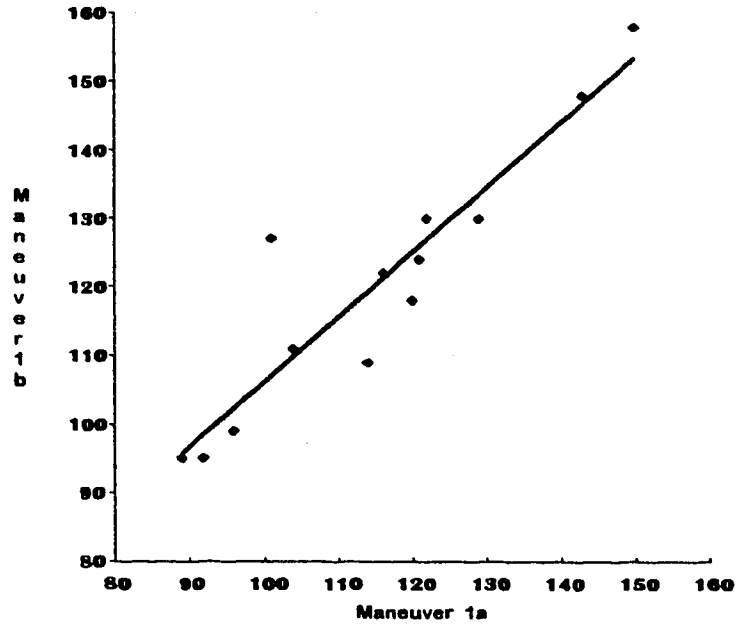


Figure 24
Heart Rate, beats/min
Maneuver 2a vs 2b, r= 0.95, p<0.01

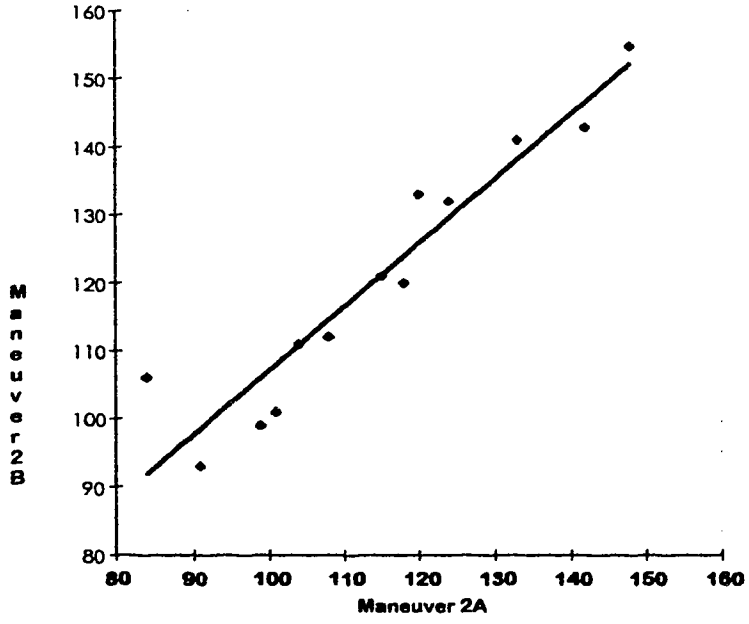


Figure 25
Heart Rate, beats/min
Maneuver 1a vs 2a, r= 0.69, p<0.01

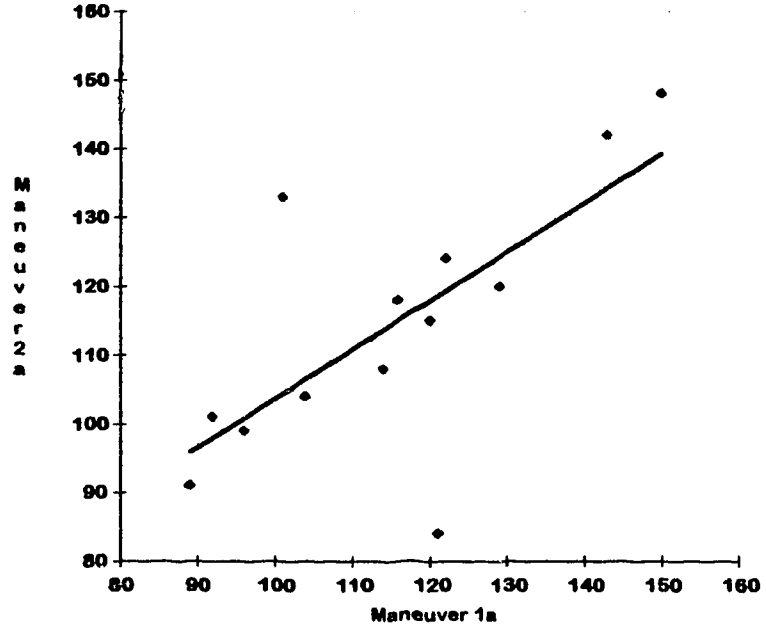


Figure 26
Heart Rate, beats/min
Maneuver 1b vs 2b, r=0.93, p<0.01

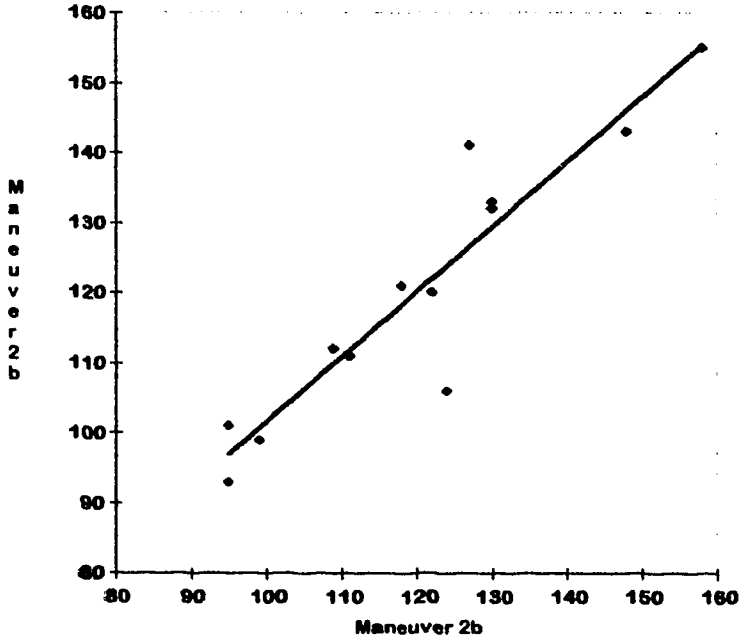


Figure 27
Oxygen Uptake, L/min
Maneuver 1a vs 1b, r=0.97, P< 0.01

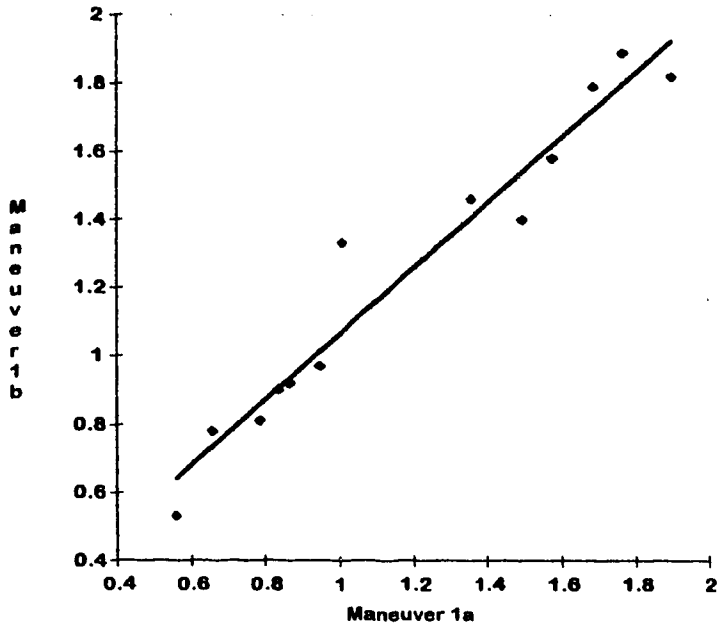


Figure 28
Oxygen Uptake, L/min
Maneuver 2a vs 2b, r=0.98, p<0.01

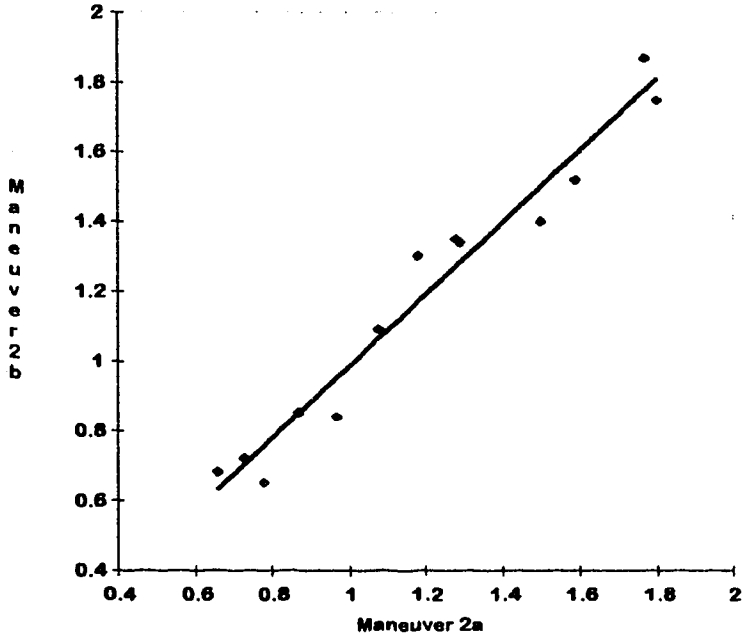


Figure 29
Oxygen Uptake, L/min
Maneuver 1a vs 2a, r =0.87, p<0.01

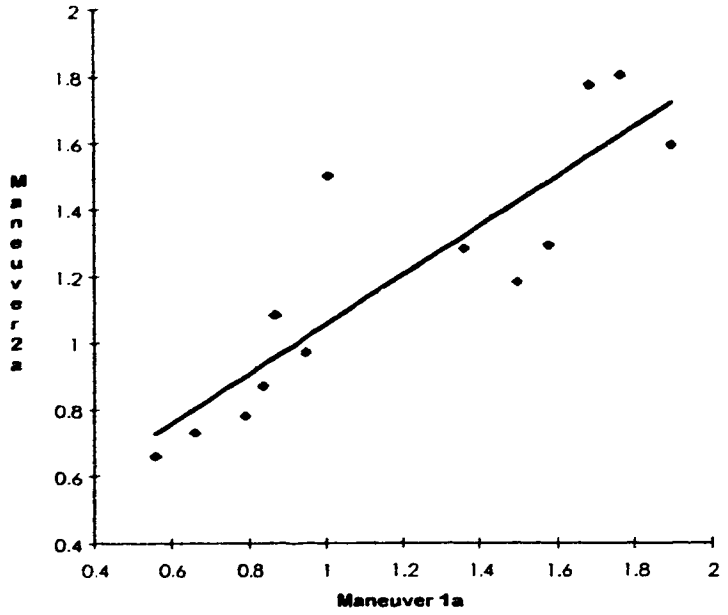


Figure 30
Oxygen Uptake, L/min
Maneuver 1b vs 2b, r= 0.95, p<0.01

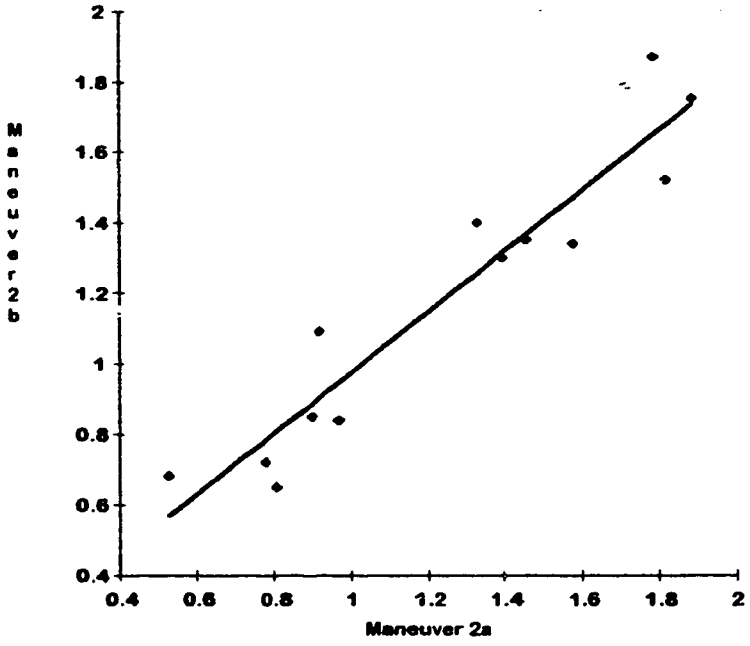


Figure 31
Cardiac Output, L/min
Maneuver 1a vs 1b, r=0.89, p<0.01

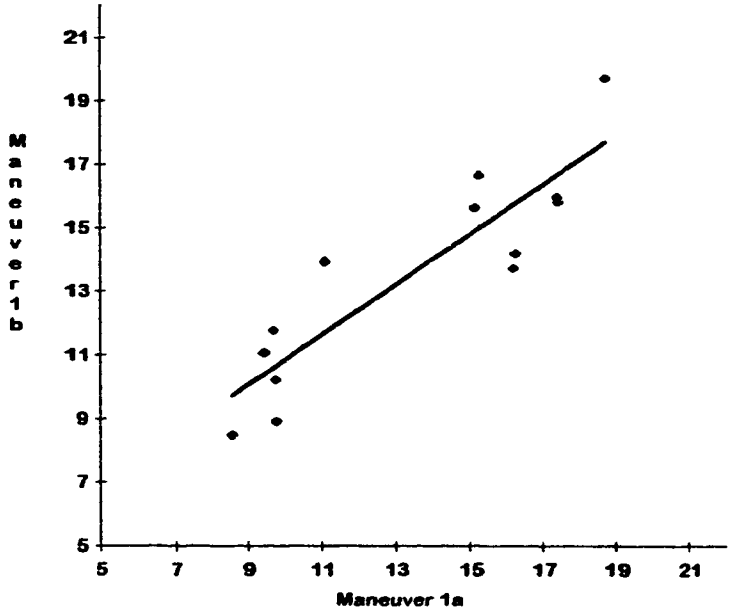


Figure 32
Cardiac Output, L/min
Maneuver 2a vs 2b, r=0 93, p<0.01

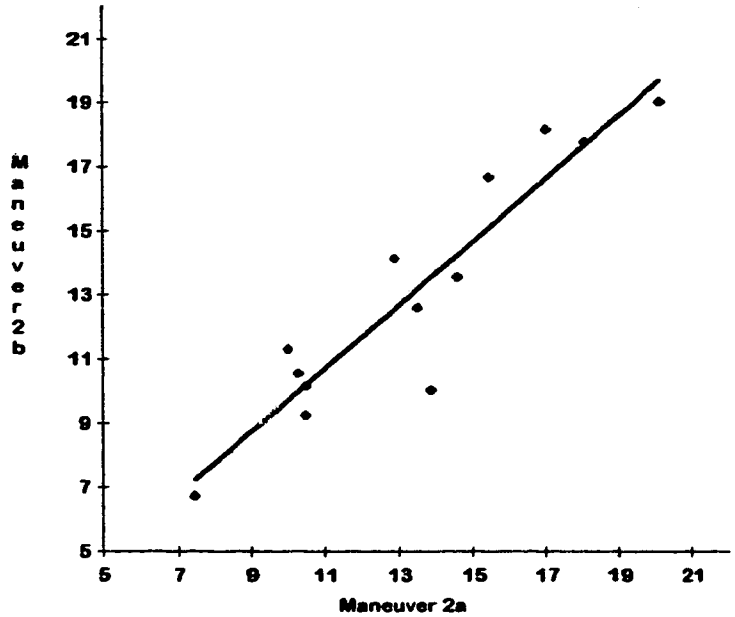


Figure 33
Cardiac Output, L/min
Maneuver 1a vs 2a, r=0.57, p=0.04

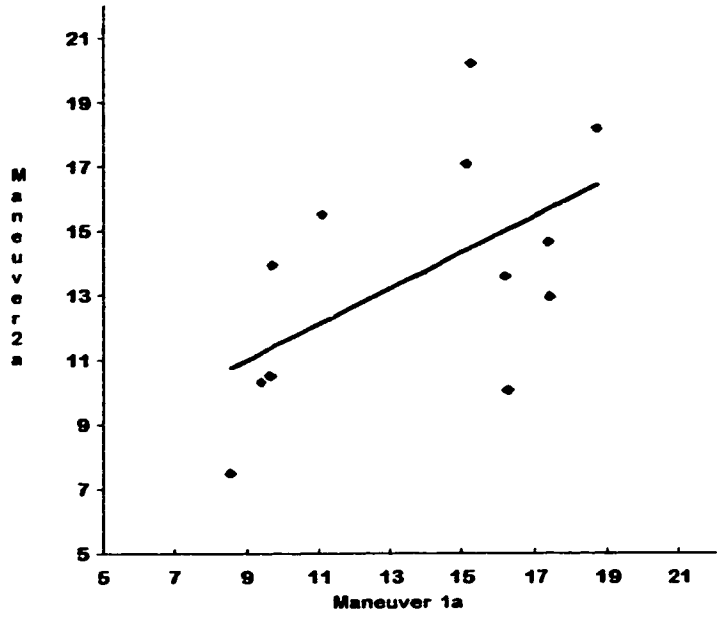


Figure 34
Cardiac Output, L/min
Maneuver 1b vs 2b, r=0.86, p<0.01

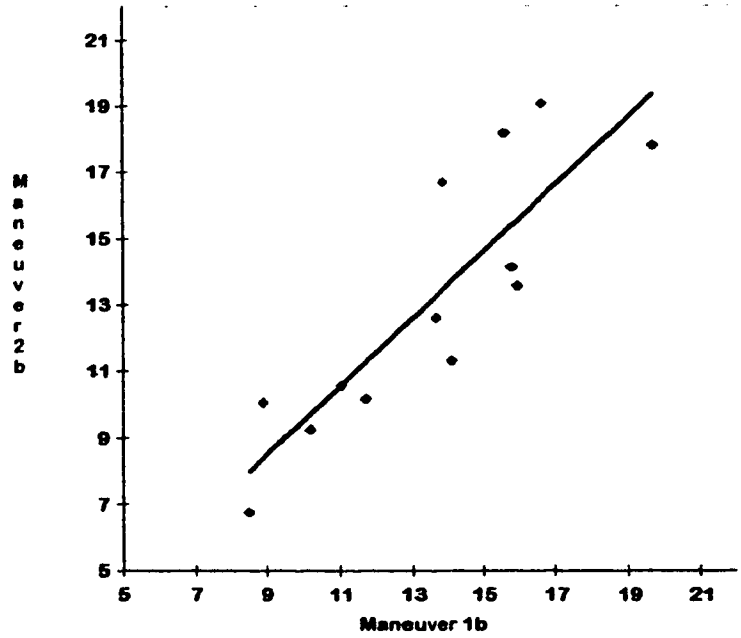


Figure 35
Stroke Volume, ml/heart beat
Maneuver 1a vs 1b, r=0.85, p<0.01

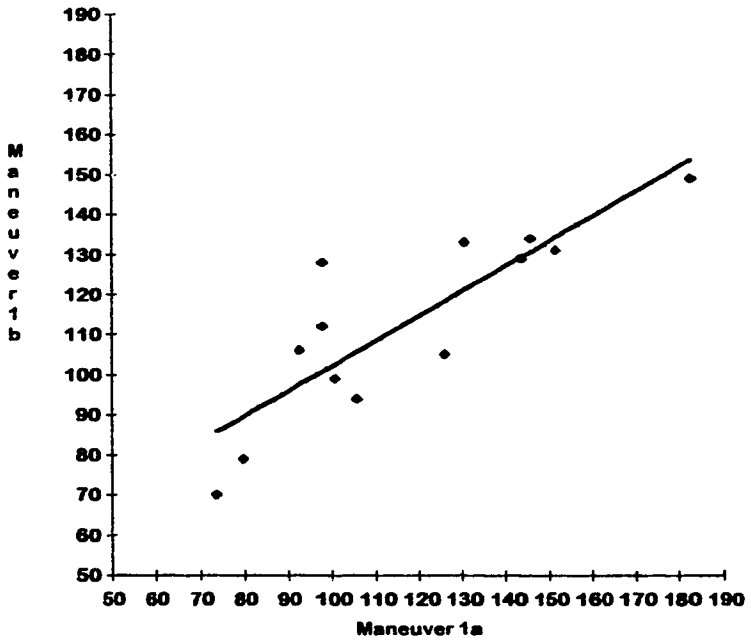


Figure 36
Stroke Volume, ml/heart beat
Maneuver 2a vs 2b, r=0.81, p<0.01

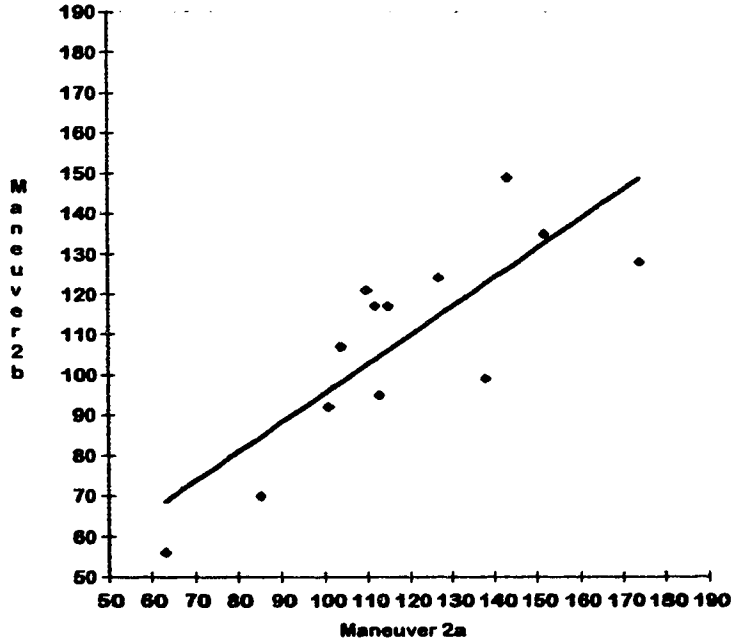


Figure 37
Stroke Volume, ml/heart beat
Maneuver 1a vs 2a, r=0.51, p=0.8, N.S.

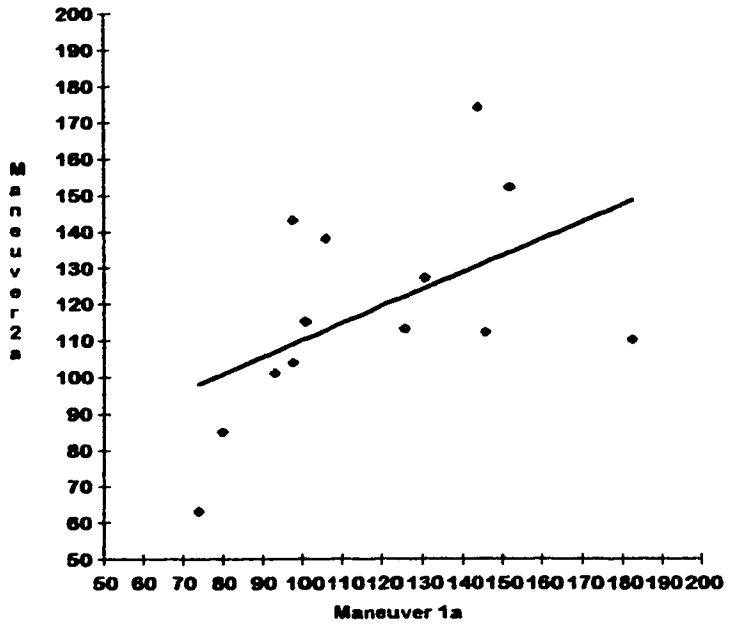


Figure 38
Stroke Volume, ml/heart beat
Maneuver 1b vs 2b, r=0.85, p<0.01

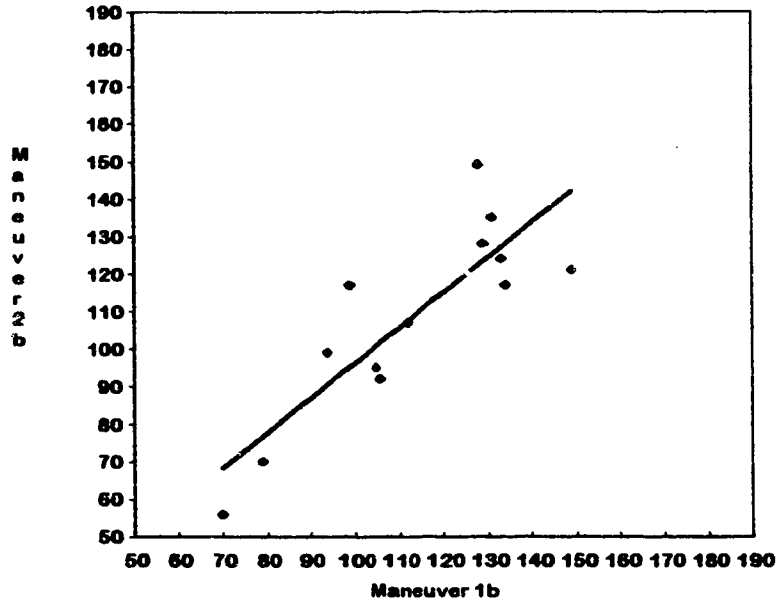


Figure 39
(a-v)O2 diff, ml/100ml
Maneuver 1a vs 1b, r=0.90, p<0.01

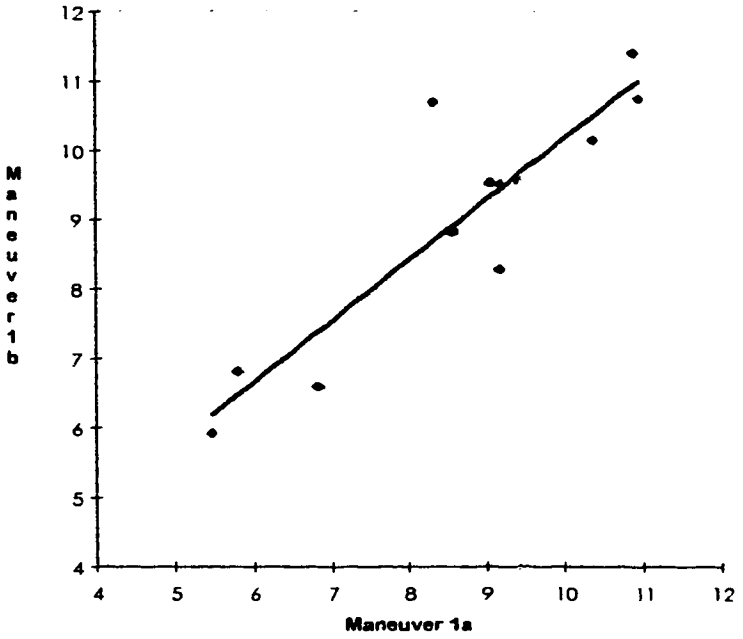


Figure 40
(a-v)O2 diff, ml/100ml
Maneuver 2a vs 2b, r=0.76, p<0.01

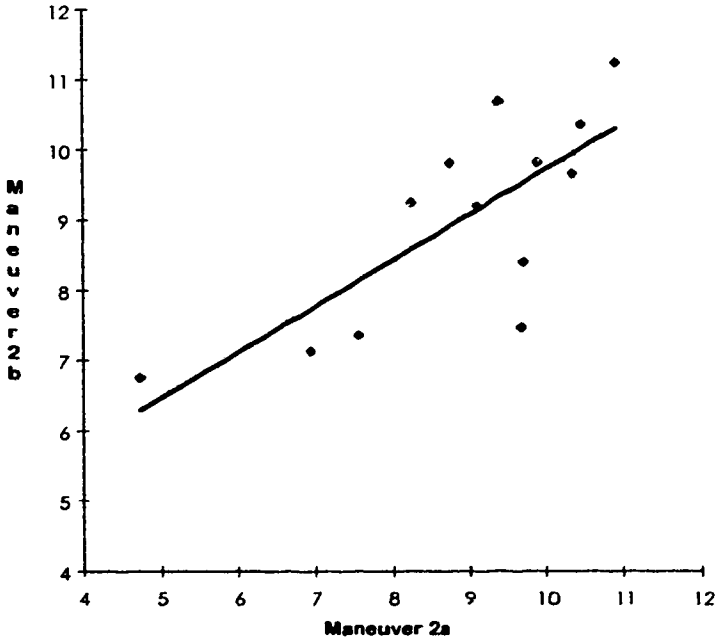


Figure 41
(a-v)O2 diff, ml/100ml
Maneuver 1a vs 2a, r=0.50, p=0.06,NS

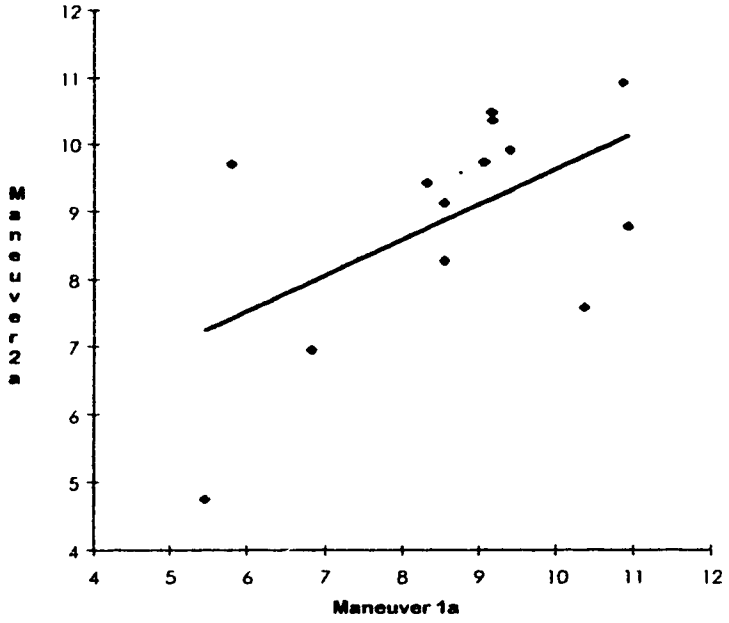


Figure 42
(a-v)O2 diff, ml/100ml
Maneuver 1b vs 2b, r=0.74, p< 0.01