# THE EFFECT OF ACTIVE RECOVERY ON THE POST-EXERCISE DIFFUSION CAPACITY

by

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B.Sc., The University of British Columbia, 1995

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF

THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE

in

THE FACULTY OF GRADUATE STUDIES

SCHOOL OF HUMAN KINETICS

We accept this thesis as confirming
to the required standard

THE UNIVERSITY OF BRITISH COLUMBIA

October 1997

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## ABSTRACT

The purpose of this study was to investigate the effect of active recovery on the post-exercise pulmonary diffusion capacity (DL) and its two components, alveolar-capillary membrane diffusion capacity (DM) and pulmonary capillary blood volume (Vc). Ten trained non-smoking male cyclists ( age = 22 ± 2 yrs; ht = 177.4 ± 7.1 cm; mass = 70.3 ± 9.1 kg; $VO_{2max}$ = 61.0 ± 4.4 ml/kg/min) were recruited for this study. All subjects demonstrated normal pulmonary function with no history of respiratory disease. All spirometry and diffusion measurements were administered using the Collins PLUS DS II pulmonary function testing unit. Subjects cycled to exhaustion to determine maximal oxygen consumption ($VO_{2max}$) and ventilatory threshold (VT) on an electronically-braked cycle ergometer in their first visit. In the following two experimental trials labeled active recovery (AR) and inactive recovery (IR), all pulmonary diffusion measurements were performed. In both sessions, pre-exercise baseline values for DLco, DM and Vc were first obtained. Subjects then performed 45 minutes of cycling exercise at the individual's VT with maximal effort near the end. In only the AR trial, subjects performed an additional 30 minutes of cycling at 10% of individual's maximal power output immediately following the 45-minute exercise bout. Two additional pulmonary diffusion capacity measurements were made at 1 and 2 hours following the 45-minute submaximal exercise test. DM and Vc were calculated by measuring DLco at two inspired $O_2$ concentrations using the technique of Roughton and Forster (1957).

DLco was significantly reduced 1 hour post-exercise ($p<0.05$) and further reduced during the second hour of seated recovery in both AR and IR conditions ($p<0.01$). A significant reduction in DM following exercise was only observed in IR condition ($p<0.05$), while post-exercise DM remained at pre-exercise baseline level in AR condition. Vc was significantly decreased at 1 and 2 hours post-exercise in both conditions ($p<0.05$ and 0.01, respectively).

Mean heart rate at 1 hour post-exercise was found to be higher than resting baseline ($p<0.05$), indicating that some of the decrease in DL, DM and Vc might have been masked by the elevated cardiac output. The most significant finding was that the depressed post-exercise DM was recovered by an active recovery, giving stronger support for the presence of pulmonary edema during and after the sustained effort which was partially responsible for the reduction in DM following exercise. Changes of Vc were in identical pattern and similar magnitude in both AR and IR conditions, suggesting that the distribution of central blood volume due to gravity might have greater effect on post-exercise Vc than the shunting mechanism. This study represents the first attempt to examine the effect of active recovery on the post-exercise pulmonary diffusion capacity.

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# LIST OF ABBREVIATIONS AND SYMBOLS

| | |
|---|---|
| A-aDO2 | Alveolar-arterial oxygen difference |
| AR | Active recovery |
| BHT | Breath hold time |
| BTPS | Body temperature and pressure saturated |
| bpm | Beats per minute |
| CO exp | Carbon monoxide expired |
| DL | Diffusion capacity of the lung |
| DLco | Diffusion capacity of the lung for carbon monoxide |
| DM | Diffusion capacity for alveolar-capillary membrane |
| EIH | Exercise-induced arterial hypoxemia |
| FVC | Forced vital capacity |
| FEV1.0 | Forced expired volume in first second |
| Hb | Hemoglobin |
| [Hb] | Hemoglobin concentration |
| He insp | Fraction of helium inspired |
| He exp | Fraction of helium expired |
| HR | Heart rate |
| HR@VT | Heart rate at ventilatory threshold |
| HRmax | Maximal heart rate |

| | |
|---|---|
| IR | Inactive recovery |
| Ln | Natural logarithm |
| PACO2 | Alveolar partial pressure of carbon dioxide |
| PaCO2 | Arterial partial pressure of carbon dioxide |
| PAO2 | Alveolar partial pressure of oxygen |
| PaO2 | Arterial partial pressure of oxygen |
| PB | Barometric pressure |
| PcapO2 | Capillary partial pressure of oxygen |
| Power@VT | Power output at ventilatory threshold |
| Q | Cardiac output |
| Qc | Capillary perfusion |
| RER | Respiratory exchange ratio |
| %SaO2 | Percentage of arterial oxyhemoglobin saturation |
| %SaO2min | Minimal percentage of arterial oxyhemoglobin saturation during exercise |
| sb | Single breath |
| STPD | Standard temperature and pressure dry |
| $\theta$ | Reaction rate of hemoglobin and carbon monoxide |
| VA | Alveolar ventilation |
| VA | Alveolar volume |
| VA/Qc | Ventilation-perfusion ratio |
| Vc | Pulmonary capillary blood volume |

| | |
|---|---|
| $VCO_2$ | Rate of carbon dioxide production |
| Ve | Expired ventilation per minute |
| $Ve/VCO_2$ | Ventilatory equivalent for carbon dioxide |
| $Ve/VO_2$ | Ventilatory equivalent for oxygen |
| VI | Volume inspired |
| $VO_2$ | Rate of oxygen uptake |
| $VO_2max$ | Maximal rate of oxygen uptake |
| VT | Ventilatory threshold |

## ACKNOWLEDGMENTS

I would like to acknowledge my supervisor Dr. Ken Coutts for his outstanding guidance and support along the way that had helped me go through many obstacles. I am also thankful to Dr. Don McKenzie for the inspiration and ideas he provided. Very special thanks to Dr. Jim Potts for his support and friendship that had made my master work a very enjoyable experience.

Many thanks to Diana Jesperson, for her patience and assistance that had made my data collection possible.

Finally, I would like to extend my gratitude to my parents and Brenda, for their never-ending love and support.

# INTRODUCTION

Diffusion limitation has been considered as one of the mechanisms responsible for the development of exercise-induced arterial hypoxemia (EIH) which occurs in about 50% of highly trained athletes exercising at oxygen uptakes in excess of 4.0-5.0 L/min ( Dempsey et al, 1984 , Powers et al. 1988, Hopkins et al. 1994). One possible reason for a diffusion limitation is the formation of pulmonary edema. A technique for the direct assessment of subclinical perivascular and/or peribronchial edema has yet to be established, but indirect assessment can be made by measurement of pulmonary diffusion capacity (DL) and the diffusion capacity of the alveolar-capillary membrane (DM). During exercise, DL and its two components, DM and pulmonary capillary blood volume (Vc) have been shown to significantly increase mainly due to the increased cardiac output (Hsia et al. 1995). Therefore, many studies have looked at the post-exercise DL, DM and Vc measurements and inferred from the post-exercise changes what was occurring during exercise.

A number of authors have reported a decrease in DL ,DM and Vc following a period of high intensity exercise (Miles et al. 1983; Manier et al. 1991 and 1993; Hanel et al. 1994; Sheel, 1995; Lama et al. 1995; Stewart et al. 1996). Decreases in post-exercise DL and DM have been interpreted to be the result of subclinical pulmonary edema (Buono et al. 1983; Lewis et al. 1958; Manier et al. 1991; Miles et al. 1983; Rasmussen et al. 1986; Schaffartzik et al. 1992; Staub et al. 1967). Some authors have suggested that the

decrease in Vc is primarily responsible for the decrease in DL (Hanel et al. 1993, Clifford et al. 1991). According to the Roughton-Forster model of pulmonary diffusion :

$$\frac{1}{DL} = \frac{1}{DM} + \frac{1}{\theta \times Vc}$$

the changes in DL are multifactorial. Furthermore, changes in DM and Vc might be interrelated (Prisk et al. 1993 ; Pistelli et al. 1991). Therefore, the interpretation of post-exercise diffusion data requires further investigation.

***(1) Are the decreases in post-exercise DL and Dm a reflection of subclinical pulmonary edema ?***

DM can be considered as the resistance of the surface allowing gas uptake by the pulmonary capillary blood. The value of DM is determined by the surface area available for diffusion and (inversely) by the thickness of the alveolar-capillary membrane (Weibel et al. 1993). Therefore, a decrease in Dm can be due to (1) a decrease in effective surface area for gas exchange as the result of a decrease in Vc and/or the worsening of ventilation to perfusion matching ($V_A/Q_C$) (Pistelli et al. 1991), and/or (2) the accumulation of interstitial fluid which increases the alveolar-capillary membrane thickness and, therefore, decreases the rate of the gas exchange (Miles et al. 1983, Manier et al. 1991 and 1993). Pulmonary edema may result from elevated capillary hydrostatic pressure, increased capillary permeability to plasma proteins, increased capillary surface area, and lymphatic insufficiency during strenuous exercise (Staub et al. 1967 and West ,1977). It is possible that highly trained athletes may accumulate more fluid than the lymphatic system can

clear during strenuous exercise (Sheel, 1995). Many studies have attributed the reduction in DL and DM to the presence of pulmonary edema as the result of strenuous exercise (Buono et al. 1983; Lewis et al. 1958; Manier et al. 1991; Miles et al. 1983; Rasmussen et al. 1986; Schaffartzik et al. 1992; Staub et al. 1967). However, as mentioned above, the changes in DL and DM are multifactorial. Changes in DL are influenced by the changes in both DM and Vc, and the changes in DM are influenced by membrane thickness, as well as effective surface area for gas exchange. Therefore, a straightforward attribution of exercise induced changes in DL and DM to the presence of pulmonary edema is not likely. Studies utilizing exercise of light to moderate intensity have demonstrated the multifactorial properties of the alteration in DL and DM (Hanel et al. 1993 and 1994 ; Sharratt et al. 1996). Hanel and her co-workers (1993) found a significant reduction in DL after subjects performed 6 minutes of rowing at 61% of maximal intensity. She suggested that the fact that a fall in DLco occurs after such mild exercise makes a significant change in pulmonary capillary membrane integrity or subclinical pulmonary edema an unlikely explanation; rather, a fall in central blood volume is more likely to be the mechanism responsible for the decrement in DLco. Sharratt et al. (1996) found significant reduction in both DM and Vc after subjects (N=8) performed 10 minutes of cycling at 25% and 50% $VO_{2max}$, in two separate sessions, clearly suggesting that mechanisms responsible for the reduction in DM are more related to the alteration in the effective surface area for gas exchange, partially as the result of the decrease in Vc.

To date, results from studies using computerized tomography (CT scan) or magnetic resonance imaging (MRI) to detect the increase in lung density and water

content as the direct evidence of subclinical pulmonary edema were mixed (Caillaud et al. 1995; McKenzie et al. 1996; Gallagher et al. 1988). McKenzie et al. (1996) found no changes in lung density using CT scan and MRI despite a significant reduction in DLco, after subjects performed 5 minutes of strenuous exercise. To investigate the role of exercise duration on the development of pulmonary edema, Caillaud et al. (1995) studied 8 triathletes and found a significant increase in mean lung density using CT scanning and a significant decrease in DLco after performing a triathlon that lasted 120±20 minutes, suggesting the existence of mild subclinical pulmonary edema which would partially explain the decrease in DLco .

***(2) Decrease in Vc after exercise: A gravitational or exercise effect ?***

Pulmonary capillary blood volume (Vc) has been shown to decrease one hour after exercise (Sheel, 1995; Lama et al. 1996; Stewart et al. 1996; Sharratt et al. 1996); and two hours post-exercise (Hanel et al. 1993 and 1994; Rasmussen et al. 1986). Sheel (1995) showed that Vc was decreased one hour after a short bout of intense exercise and reached a minimum value at 6 hours post-exercise.

Diffusion measurements taken within one hour after exercise seem to yield some contradictory results. Manier et al.(1991) found elevated Vc (10.7%) in 11 trained runners shortly (28±14 minutes) after running a marathon. Manier et al.(1993) found no change in Vc 30 minutes after 20-22 minutes of progressive cycling to exhaustion. During exercise, DLco, Dm and Vc are substantially elevated due to an increase in cardiac output and

recruitment of pulmonary capillaries ( Ayers et al. 1975 ). Hsia et al. (1995) showed that DL, Dm and Vc increase linearly with respect to cardiac output from rest to near-maximal exercise. Rasmussen et al.(1992) found significant increase in DLco immediately post-exercise. Therefore, it is possible that 30 minutes was not sufficient enough for Vc to return to baseline as shown in the studies by Manier et al. (1991 and 1993). Caillaud et al. (1995) pointed out that the elevated postrace HR observed after endurance races such as triathlon and marathon might mask part of the reduction in Vc and DL.

To date, there has only been a few speculations on the mechanisms responsible for the decrease in Vc following strenuous exercise. Lama (1996) suggested that the depressed post-exercise Vc may reflect a redistribution of blood away from the chest to the periphery in order to clear metabolic waste products from exercised muscles (Lama et al. 1996). Following termination of exercise, blood lactate levels continue to rise, and the maximum concentration in the blood may occur 2 to 5 minutes post-exercise for individual athletes (Gollnick et al. 1986). This might explain the depressed Vc one or two hours after strenuous exercise when the cardiac output has completely returned to resting level, but is unable to explain the results by Sheel (1995) in which they found the lowest Vc value at six hours and the complete recovery of Vc 24 hours after a short bout (2-3 minutes) of intense exercise. Sharratt et al. (1996) found similar reduction in Vc after subjects (N=8) performed a 10 minute cycling at 25%, 50% and 75% of maximal intensity. Since the excess metabolic waste production is likely to be minimal at exercise intensity equivalent of 25% and 50% $VO_{2max}$, the reduction in Vc should be caused by mechanisms other than

shunting as suggested by Lama (1996). The etiology of the depressed Vc several hours after strenuous exercise therefore still remains unclear.

***(3) Effect of post-exercise active warm/cool down:***

Beneficial effects of post-exercise active cool-down have been well documented (Mechelen et al. 1993; Edwards and Hopkins, 1993; Stainsby and Brooks, 1990; McMaster et al. 1989). Cool-down at low to moderate intensity significantly reduces blood lactate concentration over passive rest following strenuous exercise (McMaster et al. 1989 ; Stainsby and Brooks, 1990). The mechanisms for accelerated clearance of lactate during submaximal exercise include efflux of lactate from muscle to blood, uptake by liver, heart and skeletal muscle, and an increase in local blood flow (Gollnick et al. 1986).

**Hypotheses :**

The main purpose of this study was to identify some of the possible causes of the depressed DL, DM and Vc following sustained strenuous exercise by attempting to reverse the situation. The role of active cool-down on the post-exercise pulmonary gas exchange has yet to be investigated. However, since cooling-down at low intensity can facilitate the removal of metabolic waste products (Stainsby and Brooks, 1990 ; McMaster et al. 1989) and increase venous return after a period of strenuous leg exercise such as cycling, we hypothesized that the active cool-down would facilitate the recovery of the depressed post-exercise Vc . Coates et al.(1984) found a significant linear correlation between lymph flow and cardiac output ($r = 0.87$, $p<0.01$) resulted primarily

from an increase in perfused microvascular surface area. A low intensity (10-15% $VO_{2max}$) active cool-down should moderately increase the lymph flow and facilitate the removal of the interstitial fluid accumulated during heavy exercise. We therefore hypothesized that a low intensity active cool-down would facilitate the recovery of the depressed post-exercise Dm by means of removing the interstitial edema and increasing surface area for gas exchange as the result of a higher Vc. The following hypotheses were tested :

One and two hours after 45 minutes of submaximal exercise,

1. DLco , Dm and Vc will be significantly depressed in the inactive recovery (IR) condition when compared to baseline values.
2. Vc, Dm and DLco will decrease less in the active recovery (AR) compared with the IR condition.

# METHODOLOGY

## Subjects

Ten (N=10) non-smoking trained male endurance cyclists between the ages of 20 and 25 years were recruited. All subjects were screened for inclusion in the study, and met the following criteria : (1) $VO_{2max} \geq$ 55 ml/kg/min or 4.5 l/min, and (2) normal resting pulmonary function with no history of respiratory disease. $VO_{2max}$ was determined using a cycle ergometer as described in the experimental protocol. Spirometry measurements to ensure normal pulmonary function included the following: forced vital capacity (FVC), forced expiratory volume in one second ($FEV_{1.0}$), and the ratio of $FEV_{1.0}$/FVC. Prior to any testing, subjects received a verbal description of the experiment and completed a written informed consent form. This study was approved by the Clinical Screening Committee for Research and Other Studies Involving Human Subjects of the University of British Columbia.

## Experimental protocol :

Each subject visited the lab a total of three times. Except for two subjects, the remaining individuals completed the second and third sessions at one week intervals.

**First session :**

Upon arrival at the lab, subjects were familiarized with the experimental protocol and signed the informed consent form. The first session included anthropometric measurements, basic spirometry and a maximal exercise test. Spirometry was done on the Collins Plus DS II pulmonary function testing unit.

*Maximal exercise test ( $VO_{2max}$ test) :*

The main purpose of the maximal exercise was to determine the ventilatory threshold (VT) and the maximal power output at $VO_{2max}$. For each subject, heart rate at VT (HR@VT) and power output at VT (POWER@VT) were calculated to determine the reference intensity for the two 45 minute submaximal exercise sessions. Prior to the $VO_{2max}$ test, subjects warmed up on the electronically-braked cycle ergometer (Lode Excalibur, Groningen, The Netherlands) for 5 minutes at a self-selected intensity (40-120 watts). A ramp protocol (30 W/min) was used in the maximal exercise test. Subjects were given verbal encouragement to continue exercise as long as possible. While cycling, subjects breathed through a two way non-rebreathing value (Hans-Rudolph, #2700B). Expired gases were measured and analyzed continuously by an automated gas analysis system (Rayfield System). Variables analyzed and computed included minute ventilation ($V_E$), oxygen consumption ($VO_2$), carbon dioxide production ($VCO_2$) , respiratory exchange ratio (RER) , ventilatory equivalent of $O_2$ ($V_E$ /$VO_2$) , and ventilatory equivalent of $CO_2$ ($V_E$ /$VCO_2$). Heart rate (HR) was monitored using a Polar heart rate monitor ( Polar Vantage XL, Kempele, Finland) . In addition, arterial oxygen saturation ($SaO_2$) was

measured via ear oximetry (Ohmeda Biox 3740 pulse oximeter, BOC Health Care Inc. Edison, NJ). All the metabolic variables were measured at 15 second intervals. The criteria for $VO_{2max}$ was the attainment of three of the following: (1) a plateau in $VO_2$ with an increase in power output ; (2) RER ≥ 1.20 ; (3) a peak heart rate of ± 10% of the age-predicted maximum ( 220-age); and (4) volitional fatigue.

*Ventilatory threshold (VT) determination :*

The primary criteria used to determine the VT was the abrupt non-linear increase in the excess $CO_2$ as described in previous studies ( Volkov et al. 1975; Rhodes and McKenzie, 1984 ; Anderson and McKenzie, 1990). Estimates of ventilatory threshold were made by two observers independently, without reference to the $VO_2$ or Time axis to reduce the possibility of bias. The mean of the two estimates was used, unless two estimates differed by > 15 seconds in time at which VT took place. In that case a third independent estimate was obtained and the mean of the two closer estimates was used. Once the VT was determined, HR@ VT and POWER@VT were subsequently calculated based on the linear relationships between Time and $VO_2$, HR and POWER.

**Second and third sessions :**

The following two trials labeled active recovery (AR) and inactive recovery (IR) were separated by at least 72 hours and took place at approximately the same time of day for all subjects. Prior to the testing, subjects were required to avoid any exercise for 24 hours

and refrain from consuming alcohol and caffeine for 12 hours. Upon arrival at the laboratory subjects sat and rested for 30 minutes to stabilize their cardiopulmonary system prior to measuring DLco. All the pulmonary diffusion measurements were administered in the upright seated position as recommended by the American Thoracic Society (1995). DLco was assessed and partitioned a total of three times in each session : prior to exercise (Pre-Ex), 60 minutes after completion of the 45 minutes of submaximal exercise (post-1 Hr) and 120 minutes after exercise ( post-2 Hr). Hyperoxic (89.7%$O_2$, 10%He, 0.3%CO) and normoxic (20.9%$O_2$, 9.7%He, 0.3%CO balanced with $N_2$) test gases were breathed to facilitate partitioning DM and Vc. In the active recovery (AR) session, immediately after the 45-minute exercise, subjects performed an additional 30 minutes of cycling at 10 % of maximal power output obtained in the $VO_{2max}$ test. Following the AR each subject remained seated for 30 minutes before the post-1 Hr DLco measurement. In the IR, subjects remained seated for 60 minutes before the post 1-Hr DLco measurement. The order of the two sessions was randomly selected to eliminate any order effect. A schematic representation of the experimental design for AR and IR is as follows :

Active recovery (AR) : DLco(Pre-Ex) → 45-minute exercise bout → 30-minute cycling at 10% POWERmax → 30-minute seated resting → DLco (Post 1-Hr) → Seated resting → DLco (Post 2-Hr).

Inactive recovery (IR) : DLco(Pre-Ex) → 45-minute exercise bout → 60-minute seated resting → DLco (Post 1-Hr) → Seated resting → DLco (Post 2-Hr).

*Submaximal cycle ergometry*

Subjects performed 45 minutes of cycling on the same cycle ergometer as used in the $VO_{2max}$ test. Prior to the exercise protocol, each subject warmed up on the cycle ergometer for 5 minutes at a self-selected intensity (40-100 watts). HR was monitored and recorded every 15 seconds using a portable heart rate monitor. At the end of warm-up, the resistance was increased by approximately 30 watts per minute until subject's HR reached the predicted value for individual HR@VT. Subjects then maintained their HR at or just below HR@VT for the rest of 45 minutes, except for the last 3 minutes during which the intensity was gradually increased to their POWERmax. During the exercise HR was continuously monitored by the author to ensure that the subject was exercising at simulated race pace. Feedback from the subjects was also taken into consideration in the event that an individual HR@VT did not accurately estimate the subject's race pace. Subjects were encouraged to intake sufficient amount of fluid during the exercise to avoid any adverse effects of dehydration on exercise performance and pulmonary diffusion measurements.

*Pulmonary diffusion data collection :*

Pulmonary diffusion capacity (DLco, ml/min/mmHg) was determined by the single breath method of Roughton and Forester (1957) as modified by Ogilvie et al. (1957). This method has been shown to represent true characteristics of the membranes and pulmonary capillary bed of the ventilated parts of lungs. Seated subjects made a maximal inspiration from residual volume of a gas mixture containing 20.9% $O_2$, 9.7% He,

0.3% CO balanced with $N_2$. The breath was held for approximately 10 seconds, and then expired. The rate of disappearance of carbon monoxide (CO) from the alveolar gas during the breath hold was assessed using an infrared analyzer. Duplicate trials were performed in order to ensure that values differed by less than 3 ml/min/mmHg. The average of the two closest values was recorded. In order to calculate diffusion of the membrane (DM) and pulmonary capillary blood volume (Vc), a second DLco test was performed similar to the method of Roughton and Forester (1957). Subjects breathed for 5 minutes through a low resistance valve (Hans Rudolph) attached to a Douglas bag filled with a gas mixture of approximately 90% $O_2$ and 10% $N_2$, followed by a standard DLco test using gas mixture of 90% $O_2$, 10% He, and 0.3% CO. Prior to post-exercise DLco measurements, [Hb] and subject body mass were measured in order to ascertain whether sufficient liquids were consumed to maintain the total blood volume. Capillary blood samples were used to calculate [Hb] (HemoCue, Helsingborg, Sweden). Heart rate was recorded prior to each DLco measurement.

**Calculation of diffusion capacity (DL)** *:*

Diffusion measurements were calculated by the Collins PLUS DS II system using the following equations :

*Alveolar volume (single breath)*

$$VA\,(sb) = \frac{He\ insp}{He\ exp} \times VI \times 1.05 \times BTPS$$

VA = Alveolar Volume

VI = Volume inspired

He insp = Fraction of inspired Helium

He exp = Fraction of expired Helium

BTPS = Body temperature and pressure saturated

1.05= Correction factor for 5% carbon dioxide in expired air removed prior to analysis

*Diffusion of the lung/Alveolar Volume*

$$DL/VA = (60/BHT) \times (1000/PB-47) \times Ln\ [He\ exp/CO\ exp] \times (STPD/BTPS)$$

BHT = breath hold time

Ln = natural logarithm

PB = barometric pressure

CO exp = carbon monoxide expired

STPD = standard temperature and pressure dry

*Diffusion of the lung ( single breath)*

$$DL\ (sb) = VA\ (sb) \times DL/VA$$

**Calculation of DM and VC :**

The reciprocal of DLco ( 1/DLco), or resistance, is the sum of two resistances (membrane and erythrocyte): (1) the resistance to diffusion of CO from the alveoli through the alveolar epithelium, basement membrane and capillary endothelium and then through a plasma layer to the surface of the red blood cell (1/DM) and (2) the resistance dependent on the specific rate of CO uptake by red blood cells and reaction with hemoglobin ($\theta$), and the total pulmonary capillary blood volume (Vc). The overall relationship can be expressed as follows :

$$\frac{1}{DLco} = \frac{1}{DM} + \frac{1}{\theta \times Vc}$$

By measuring DLco at two different inspired $O_2$ concentrations ( 21% and 90%), and plotting each value of 1/DLco against each $1/\theta$ , a linear regression can be formed. The slope of the regression line estimates 1/Vc. Resistance to diffusion offered solely by the membrane component (1/DM) is represented by the Y-intercept. Each value of $1/\theta$ was calculated as described by Forster et al. (1986) when mean capillary oxygen ($PcapO_2$) tension can be estimated by using the alveolar air equation assuming a respiratory exchange ratio (RER) value of 0.8 and the arterial pressure of carbon dioxide ($PaCO_2$) is equal to an alveolar $PCO_2$ ($PACO_2$) of 40 mmHg. Values for $\theta$ pre- and post-exercise were corrected for deviations from normal [Hb] (Cotes et al. 1972).

$$1/\theta = \frac{[0.34 + (0.006 \times PcapO_2)]}{([Hb]/15)}$$

The test-retest correlation for both test gas measures was assessed previously in this lab and was found to be very high ( r = 0.98 and 0.96, for DLco 21%$O_2$ and DLco 90%$O_2$, respectively ) (Sheel, 1995).

**Statistical analyses :**

Data ( DLco, DM, Vc and HR) was examined using a 2 X 3 ( Recovery method X Time) factorial analysis of variance with repeated measures across both factors. Newman-Keuls post-hoc test was used if a significant time effect (3 levels) was found. If a significant interaction occurred, Scheffe's post-hoc procedure was applied for further comparison. The level of significance was set at $p < 0.05$ for all statistical comparisons.

# RESULTS

## Subject Descriptive Data

Subject (N=10) anthropometric and resting pulmonary function data are presented in Table 1. All data are expressed as means ± SD. All resting pulmonary function test results showed no abnormalities.

Table 1. Physical characteristics of subjects.

| AGE (yrs) | HEIGHT (cm) | MASS (kg) | FVC (L) | $FEV_{1.0}$ (L) | $FEV_{1.0}/FVC$ (%) |
|---|---|---|---|---|---|
| 22.4 ± 1.7 | 177.4 ± 7.1 | 70.3 ± 9.1 | 5.7 ± 0.9 | 4.9 ± 0.6 | 86.0 ± 4.0 |

Values are means ± SD

## Maximal Exercise Test Data

The mean physiological measures (± SD) in the maximal exercise test are presented in Table 2. All subjects were competitive male cyclists from the UBC Cycling Club. Using the criteria $\%SaO_{2min} < 91\%$ (Powers et al., 1983), 2 subjects exhibited EIH.

Table 2. Maximal and minimum values for metabolic, ventilatory and power output data during maximal exercise test.

| $VO_{2max}$ (L/min) | $VO_{2max}$ (ml/kg/min) | Peak Power (watts) | $\%SaO_{2min}$ (%) | HRmax (bpm) | HR@VT (bpm) | POWER@VT (watts) |
|---|---|---|---|---|---|---|
| 4.3 ± 0.6 | 61.0 ± 4.4 | 412.1 ± 50.9 | 93.0 ± 1.7 | 191.3 ± 8.4 | 169.1 ± 5.0 | 291.8 ± 43.7 |

Values are means ± SD

**Submaximal Cycle Ergometry**

Table 3 shows a comparison during the 45 minutes of cycling exercise in AR and IR sessions. Each subject exercised at exactly same power output in both conditions (Figure 1). No difference was found in average HR during exercise between conditions (Figure 2). The resistance of the cycle ergometer (POWER) was continuously adjusted to maintain HR at target level ( i.e. HR@VT ), and %POWERmax were calculated based on the HRmax and POWERmax each subject reached in the $VO_{2max}$ test. Figure 3 shows the mean HR during the 30 minutes of active recovery in AR session.

Table 3. Average HR and power output expressed as absolute value or percentage of maximal value during the 45 minutes of submaximal cycle ergometry.

| | Ave. HR (bpm) | %HRmax | Ave. POWER (watts) | %POWERmax |
|---|---|---|---|---|
| Active Recovery | 169.4 ± 5.6 | 88.4 ± 2.9 | 237.7 ± 33.1 | 62.3 ± 8.2 |
| Inactive Recovery | 170.6 ± 6.0 | 89.0 ± 3.2 | 237.7 ± 33.1 | 62.3 ± 8.2 |

Values are means (± SD)

Figure 1. Mean power output during the 45 minute submaximal cycle ergometry

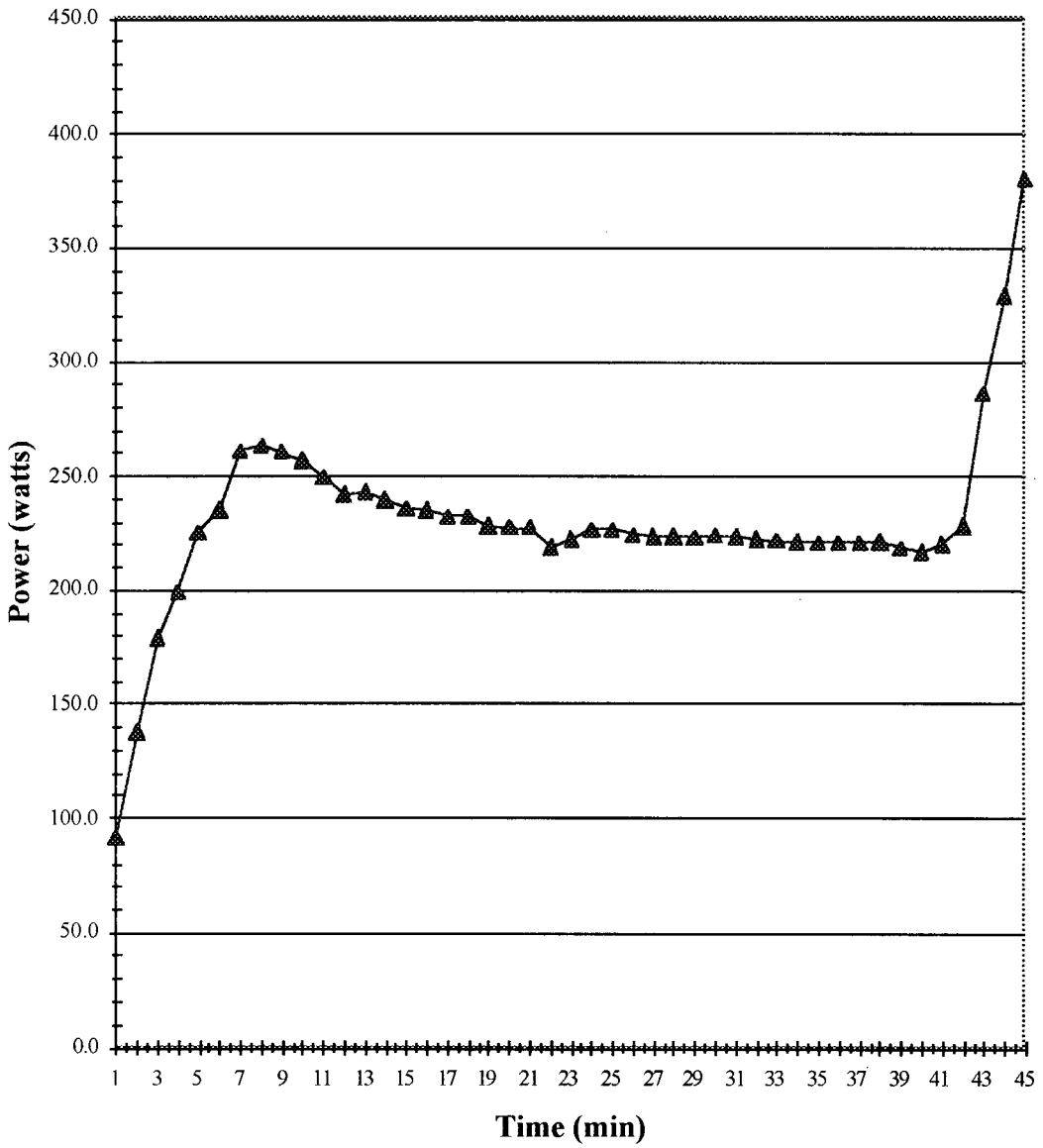


Figure 2. Mean heart rate during the 45 minutes of submaximal cycle ergometry in AR and IR conditions

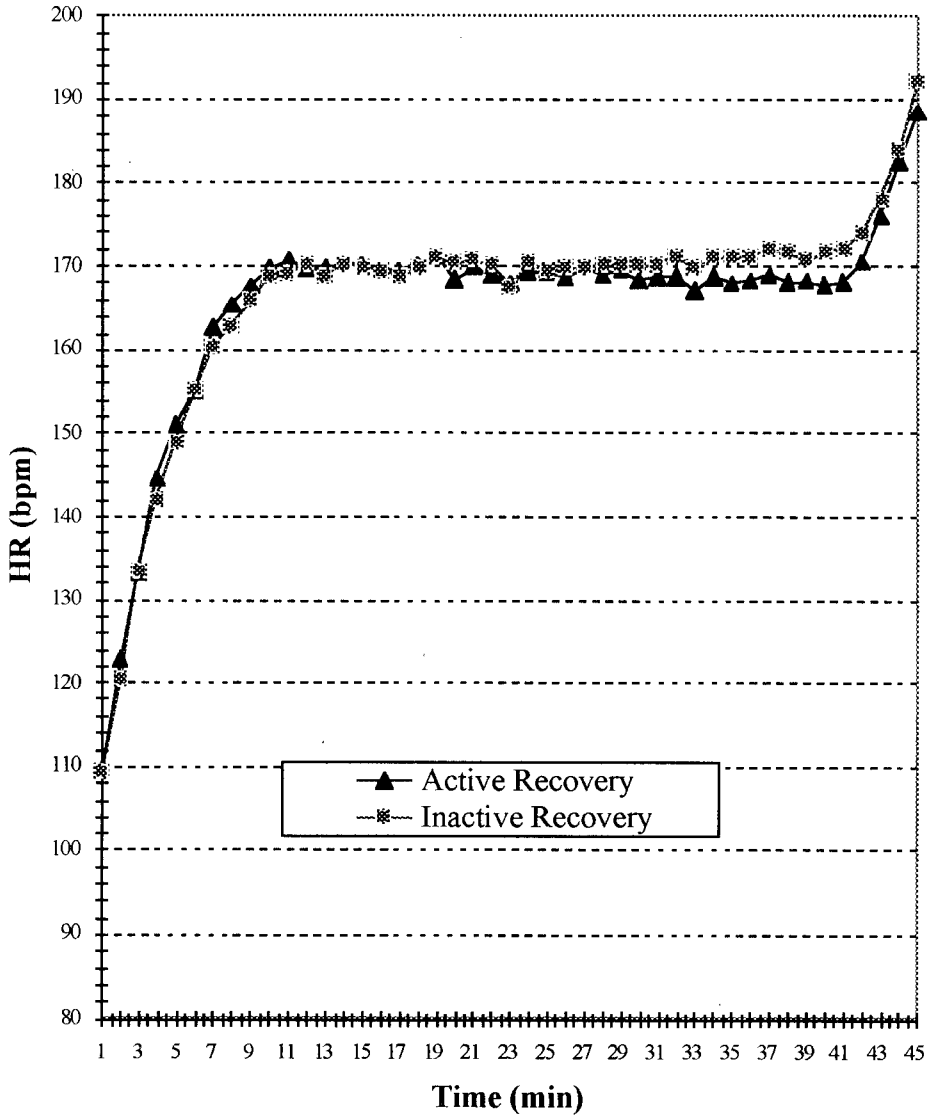
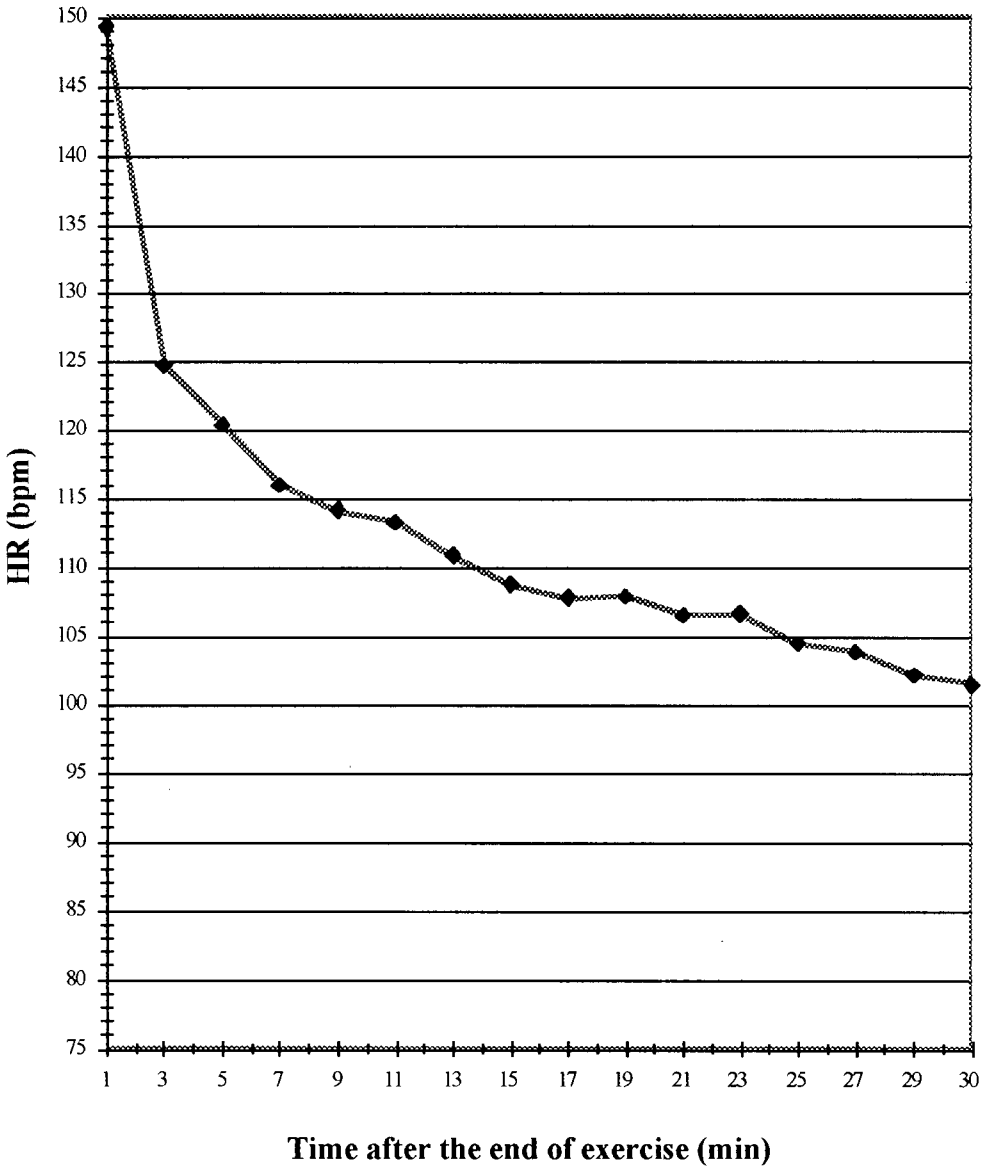


Figure 3. Mean heart rate during the 30 minutes of active recovery in AR condition



**Pulmonary Diffusion capacity for Carbon Monoxide (DLco)**

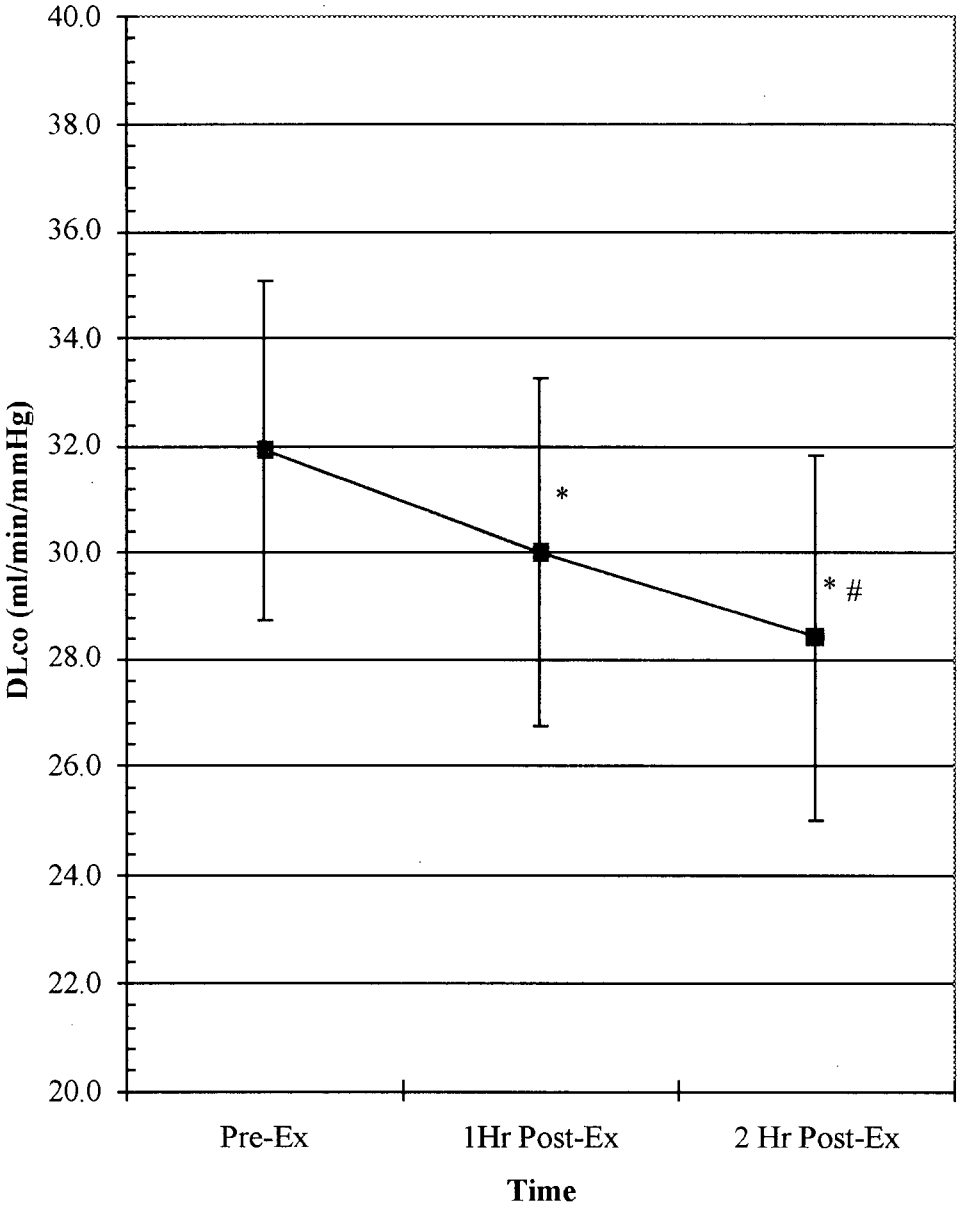
DLco was not significantly different between AR and IR conditions ( $F=0.54$, $P=0.48$). However, a significant time effect was found , indicating that mean DLco averaged over conditions was different between time periods ( $F=17.08$, $P<0.001$). Newman-Keuls test reveals that DLco was significantly decreased 1 hour after exercise, and decreased further during the second hour of recovery period ($p<0.05$, Table 4). Figure 4 displays overall values over time. There was also a significant condition x time interaction effect ( $F=4.13$, $P<0.05$). This interaction indicates that the groups were heterogeneous over time. However, Scheffe's post-hoc procedure revealed no significant difference between AR and IR at any given time. The trend for group DLco over time is visually depicted in Figure 5.

Table 4. Pulmonary diffusion capacity for carbon monoxide (ml/min/mmHg) during rest (Pre-Ex) and following 45 minutes of submaximal exercise in both AR and IR conditions.

| Condition | Pre-Ex | 1 Hr Post-Ex | 2 Hr Post-Ex |
|---|---|---|---|
| AR (N=10) | 31.49 ± 3.15 | 30.25 ± 3.47 | 29.15 ± 3.21 |
| IR (N=10) | 32.33 ± 3.33 | 29.72 ± 3.20 | 27.66 ± 3.63 |
| Mean (± SD) | 31.91 ± 3.18 | 29.98 ± 3.26 * | 28.41 ± 3.42 * # |

Values are means (± SD). AR, active recovery; IR, inactive recovery.
* Significantly different from Pre-Ex, $p<0.05$
# Significantly different from 1 Hr Post-Ex, $p<0.05$

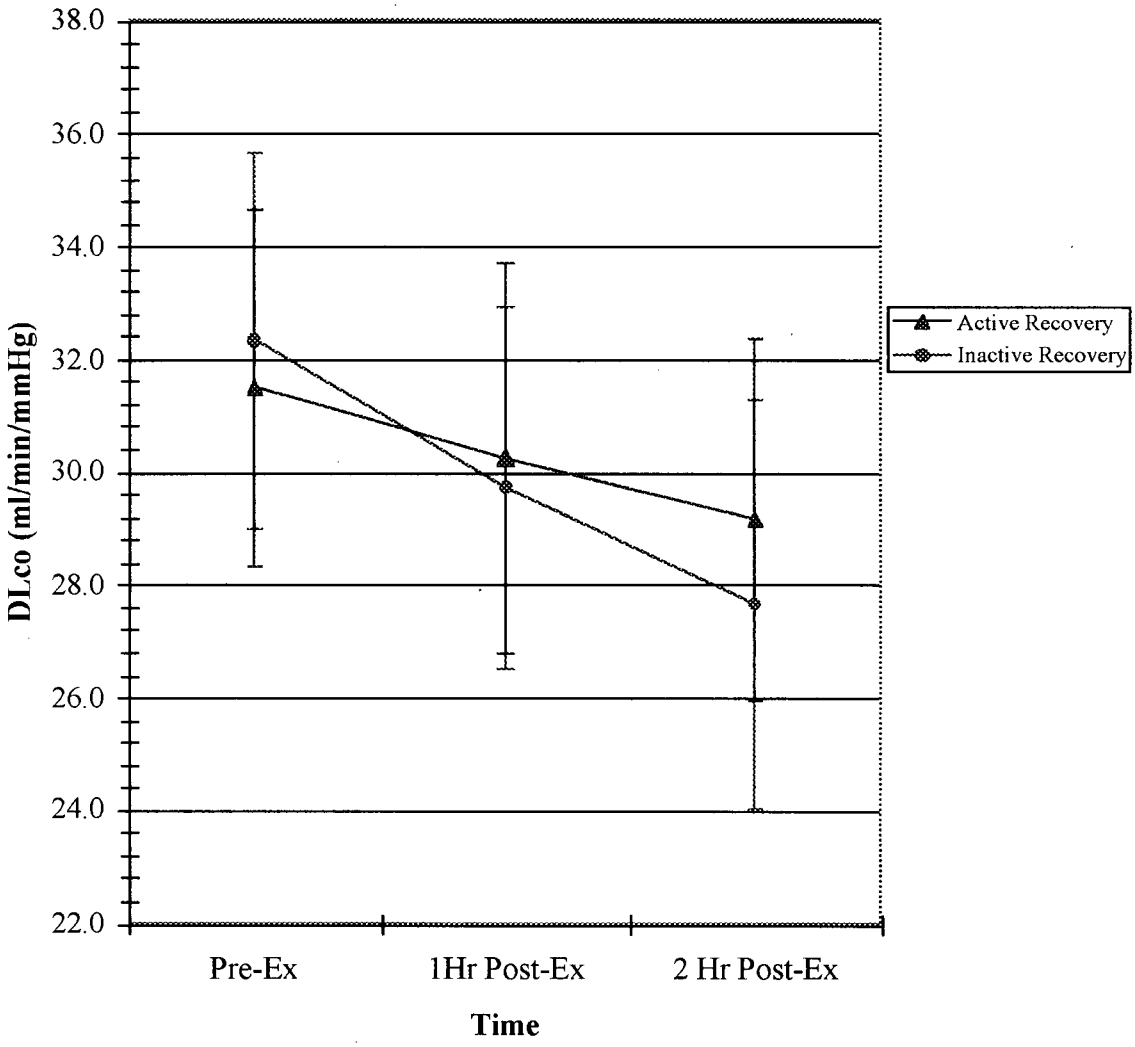
Figure 4. Overall pulmonary diffusion capacity for carbon monoxide during rest (Pre-Ex) and following 45 minutes of submaximal cycle ergometry (Mean ± SD)




* Significantly lower than Pre-Ex, $p < 0.05$

\# Significantly lower than 1 HR Post-Ex, $p < 0.05$

Figure 5. Group pulmonary diffusion capacity for carbon monoxide (DLco) during rest (Pre-Ex) and following 45 minutes of submaximal cycle ergometry (Mean±SD)



**Diffusion Capacity for Alveolar-Capillary Membrane (DM)**

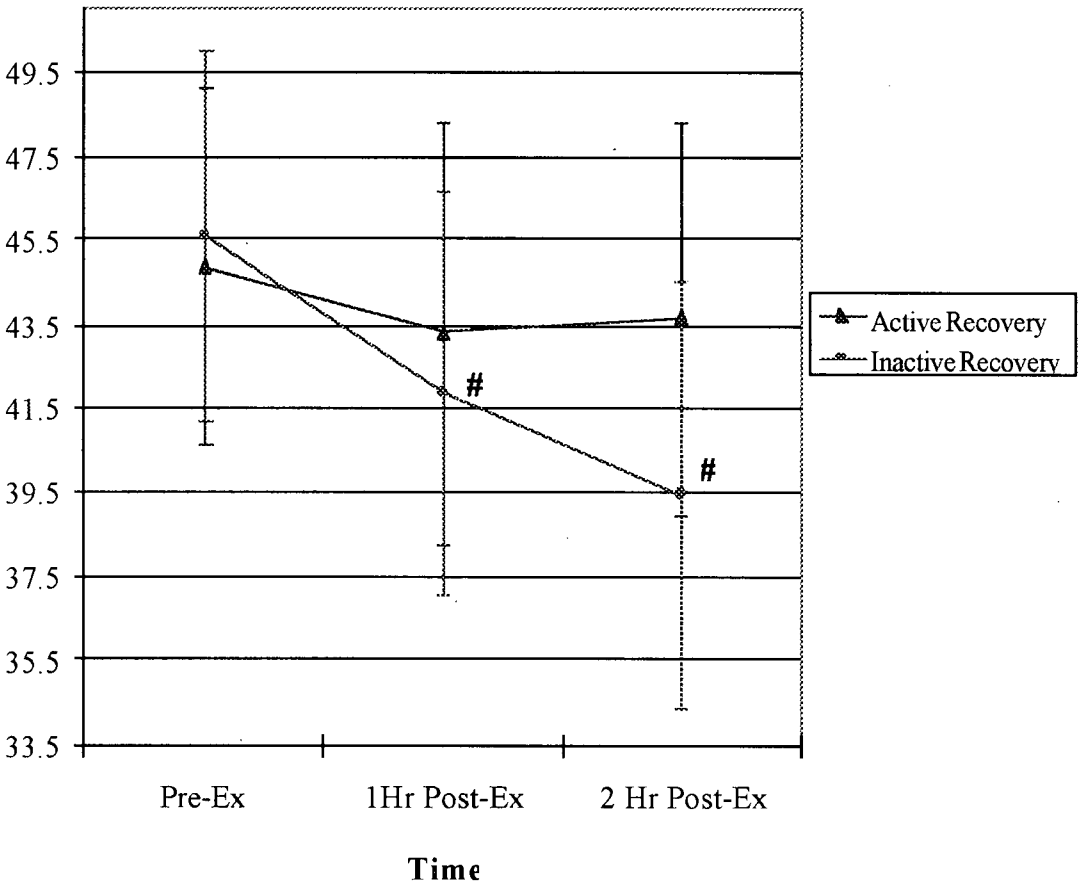
A significant condition effect was observed ($F=6.26$, $p<0.05$), indicating that averaged over time subjects in AR had significantly higher DM than in IR condition ($43.94 \pm 4.55$ *vs.* $42.32 \pm 5.26$ ml/min/mmHg, mean ± SD). A significant time effect was also found ($F=8.63$, $P<0.01$). Further analysis using Newman-Keuls test showed that subjects in IR condition experienced significant decrease in DM following exercise at both 1Hr Post-Ex and 2Hr Post-Ex ($p<0.05$), while in AR condition DM was maintained and subjects showed no significant change in DM following 45 minutes of sub-maximal exercise (Table 5). There was no significant condition x time interaction effect ($F=3.18$, $P=0.066$). Figure 6 shows mean DM in both conditions over time.

Table 5. Membrane diffusion capacity (ml/min/mmHg) during rest (Pre-Ex) and following 45 minutes of submaximal exercise in both AR and IR conditions.

| Condition | Pre-Ex | 1 Hr Post-Ex | 2 Hr Post-Ex |
|---|---|---|---|
| AR (N=10) | 44.87 ± 4.21 | 43.29 ± 5.06 | 43.65 ± 4.68 |
| IR (N=10) | 45.61 ± 4.41 | 41.88 ± 4.79 * | 39.46 ± 5.04 * |

Values are means (± SD). AR, active recovery; IR, inactive recovery.
* Significantly lower than Pre-Ex $p<0.05$

Figure 6. Group membrane diffusion capacity (DM) during rest (Pre-Ex) and following 45 minutes of submaximal cycle ergometry (Mean±SD)




# Significantly lower than Pre-Ex, $p < 0.05$

**Pulmonary Capillary Blood Volume ( Vc)**

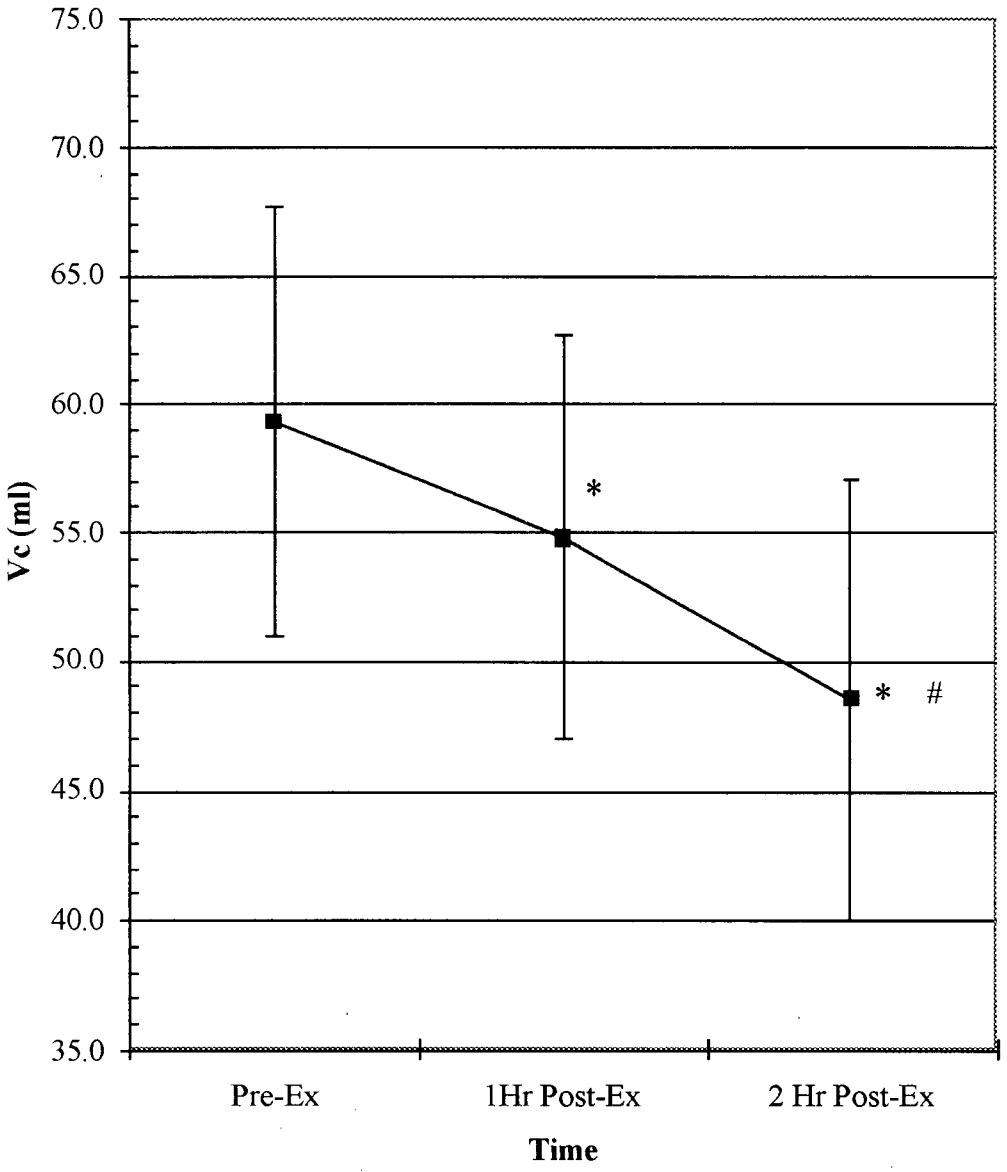
A non-significant condition effect was observed, indicating that mean Vc values did not differ significantly between AR and IR ($F=0.52$, $P=0.488$). Mean Vc averaged over time was different ( $F=46.79$, $P<0.001$). Further analysis showed a significant decrease in Vc one hour after exercise ($p<0.05$). Vc was further decreased ( $p<0.01$) during the second hour of recovery (Table 6). Figure 7 shows overall values over time. No significant condition x time interaction effect was observed ( $F=0.02$, $P=0.985$). Figure 8 shows that the trend for Vc over time was almost identical in AR and IR.

Table 6. Pulmonary capillary blood volume (ml) during rest (Pre-Ex) and following 45 minutes of submaximal exercise, group data.

| Condition | Pre-Ex | 1 Hr Post-Ex | 2 Hr Post-Ex |
|---|---|---|---|
| AR (N=10) | 58.80 ± 7.94 | 54.11 ± 7.55 | 48.12 ± 9.11 |
| IR (N=10) | 59.83 ± 9.20 | 55.46 ± 8.52 | 48.95 ± 8.40 |
| Mean (± SD) | 59.31 ± 8.38 | 54.78 ± 7.87 * | 48.53 ± 8.54 * # |

Values are means (± SD). AR, active recovery; IR, inactive recovery.
* Significantly lower than Pre-Ex, $p<0.05$
# Significantly lower than 1 Hr Post-Ex, $p<0.01$

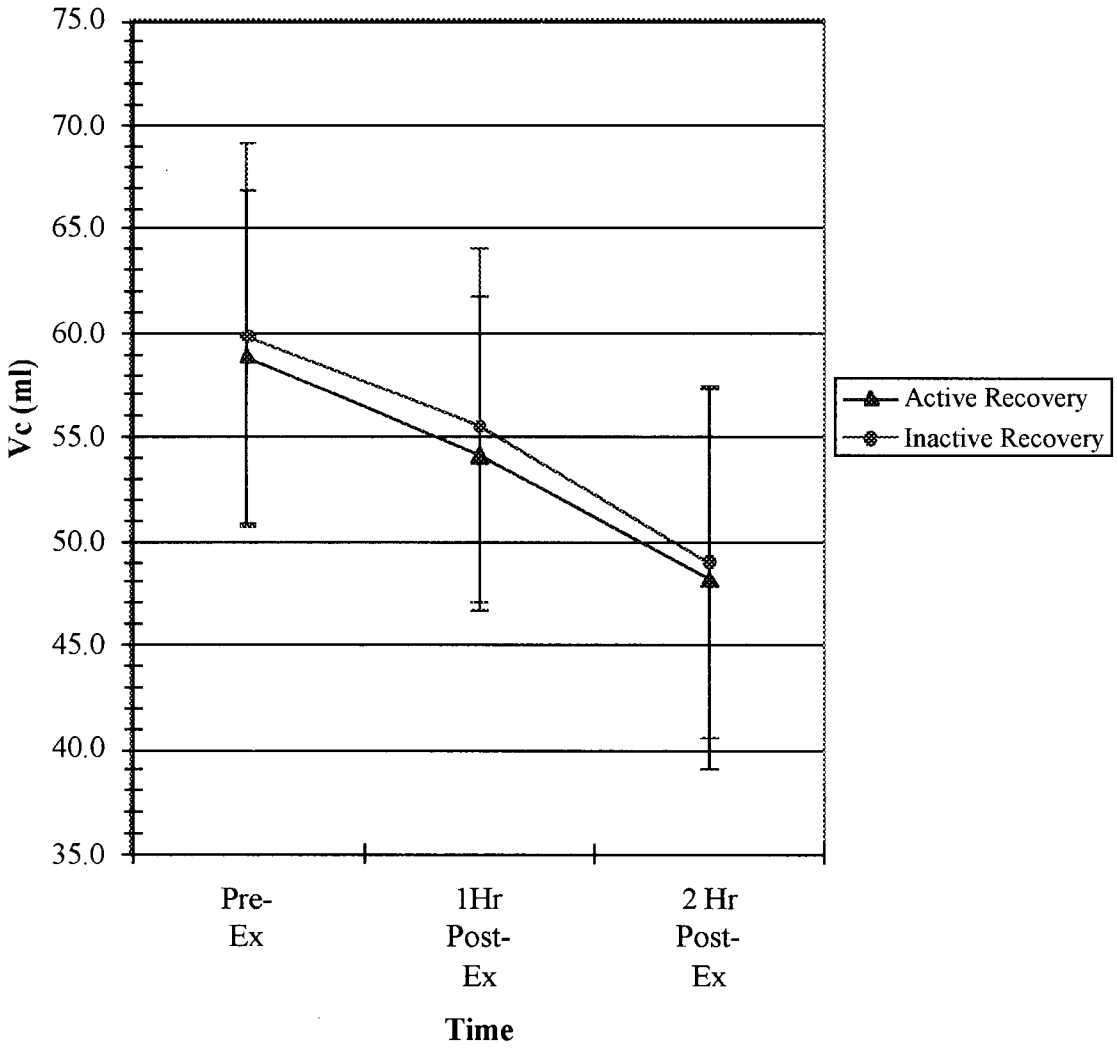
Figure 7. Overall pulmonary capillary blood volume (Vc) during rest (Pre-Ex) and following 45 minutes of submaximal cycle ergometry (Mean ± SD).




* Significantly lower than Pre-Ex, $p < 0.05$

\# Significantly lower than 1 HR Post-Ex, $p < 0.05$

Figure 8. Group pulmonary capillary blood volume (Vc) during rest (Pre-Ex) and following 45 minutes of submaximal cycle ergometry (Mean±SD)



**Heart rate at the time of DLco measurement**

HR at the time of DLco measurement was not significantly different between conditions ($F=1.81$, $P=0.212$) nor was there a significant condition x time interaction ($F=2.71$, $P=0.093$). A significant time effect was found, indicating that HR was different between time periods ($F=18.69$, $P<0.001$). Further analysis showed that HR was significantly increased 1 hour after the exercise ( $p<0.01$) and gradually returned to pre-exercise value 2 hours following exercise (Table 7). Figure 9 shows overall values over time. Figure 10 displays group means over time.

Table 7. Mean heart rate (bpm) immediately prior to the DLco measurement before (Pre-Ex) and after 45 minutes of submaximal exercise, group data.

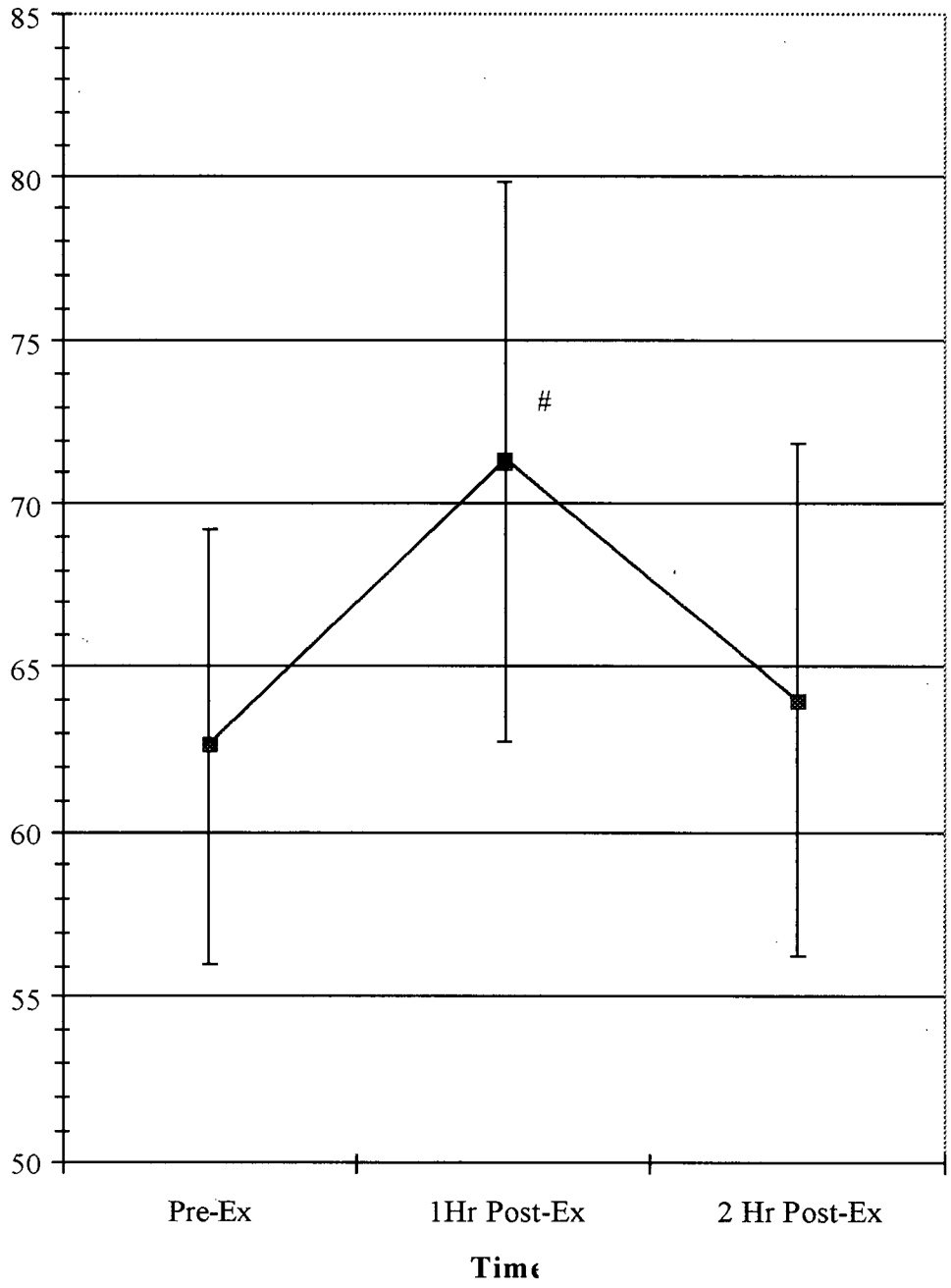
| Condition | Pre-Ex | 1 Hr Post-Ex | 2 Hr Post-Ex |
|---|---|---|---|
| AR (N=10) | 62.53 ± 7.09 | 69.19 ± 7.95 | 63.10 ± 7.55 |
| IR (N=10) | 62.74 ± 6.49 | 73.47 ± 8.90 | 64.97 ± 8.37 |
| Mean (± SD) | 62.64 ± 6.62 | 71.33 ± 8.50 * | 64.04 ± 7.81 |

Values are means (± SD). AR, active recovery; IR, inactive recovery.
* Significantly lower than Pre-Ex $p<0.01$

**[Hb] and body mass at each DLco measurement**

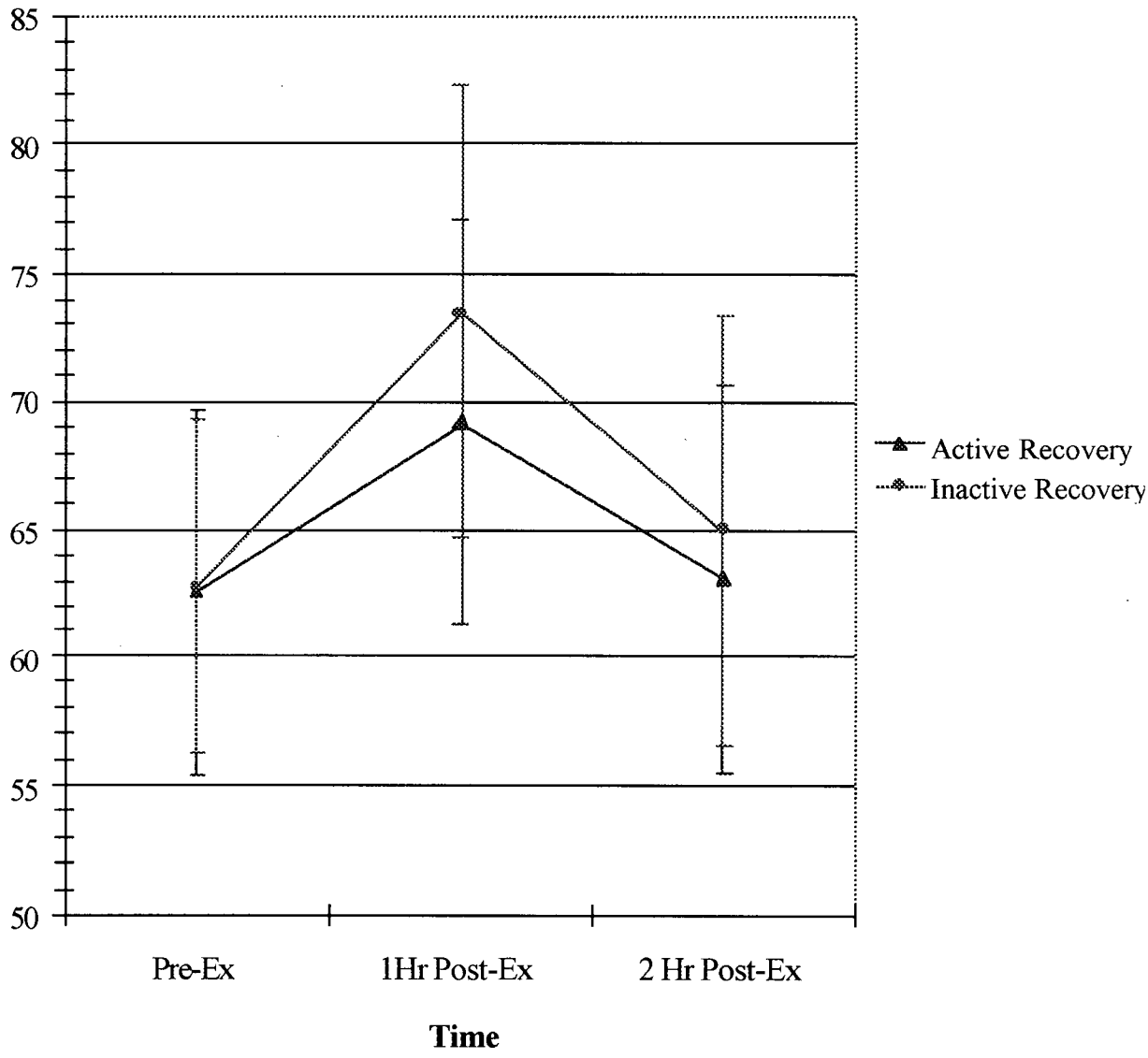
There were no significant time, condition, or time x condition interaction effects for hemoglobin concentration and body mass (Appendix C). Table B7 and B8 show individual values.

Figure 9. Overall heart rate immediately prior to DLco measurement (Mean±SD).




# Significantly higher than Pre-Ex and 2 Hr Post-Ex, $p < 0.01$

Figure 10. Group heart rate immediately prior to each DLco measurement (Mean±SD)



## DISCUSSION

A diffusion limitation, especially due to pulmonary edema, may contribute to a $V_A/Q_c$ mismatch in hypoxic conditions (Schaffartzik et al. 1992) and to the arterial hypoxemia observed in elite athletes at sea level ( Caillaud et al. 1993 ). Despite numerous attempts to explain the presence of pulmonary edema by a reduction in post-exercise pulmonary diffusion capacity, results from studies using direct measurement techniques such as CT scan and MRI have been mixed (Caillaud et al. 1995; McKenzie et al. 1996; Gallagher et al. 1988). Generally it is believed that both duration and intensity of exercise are important in the development of pulmonary edema (Caillaud et al. 1995; Sharratt et al. 1996; Manier et al. 1991 and 1993). To date it has been unclear if the changes observed in DM are exclusively due to the presence of pulmonary edema or are partially related to the changes in Vc. This study represents the first attempt to utilize a simulated race protocol and to combine the beneficial effects of active recovery in order to identify possible mechanisms for the reduction in post-exercise DL, DM and Vc.

### Exercise Protocol

The 45 minutes of sustained heavy exercise at individual ventilatory threshold produced significant decrease in DL, DM and Vc and confirms many previous studies (Miles et al. 1983; Manier et al. 1991 and 1993; Hanel et al. 1994; Sheel, 1995; Lama et al. 1995; Stewart et al. 1996). The 45-minute protocol was chosen over shorter durations as used in other studies (Hanel et al. 1993; Sheel ,1995 ; Stewart et al. 1996 ) for the following two

reasons : (1) previous studies (Manier et al. 1991 and 1993 ; Caillaud et al. 1995 ) implicated the role of exercise duration on the alteration of the alveolar capillary diffusion capacity following intense exercise. Studies using CT scan or MRI suggested a certain length of exercise might be required to accumulate a sufficient amount of interstitial fluid detectable by imaging techniques 60 to 90 minutes after exercise (McKenzie et al. 1996, Caillaud et al. 1995); (2) the simulation of a real race with above AT effort at the beginning and all out sprinting at the end. During the 45-minute cycling, individual HR was continuously monitored and the resistance was adjusted if needed. Over the 45 minutes period, subjects had mean HRs almost identical to the predicted HR@VT (Table 3), indicating that subjects were exercising at normal race pace for endurance events such as bicycle time trial or a marathon. Except for two subjects, all reported that they could not go any harder for the same period of time. However, the mean power output was about 17.4% less than the predicted value for POWER@VT. This is probably due to cardiac drift because the estimation of POWER@VT was based on the test data of the $VO_{2max}$ test which on average lasted less than 14 minutes.

**Diffusion capacity for alveolar-capillary membrane (DM)**

Reductions in DM have been observed to persist for 2 hours following exercise ( Sheel, 1995; Manier et al. 1991;1993; Miles et al. 1983). Hanel et al. (1994) found DM to reach its lowest value 2 hours after rowing and was restored by 4 hours. In the present study, subjects in IR experienced significant decrease in DM for at least 2 hours following exercise, and this confirms the findings in the previous studies. However, the depressed

post-exercise DM was recovered following the active recovery protocol. This is the most significant finding of the present study and gives stronger support for the presence of pulmonary edema as the result of sustained heavy exercise.

During heavy exercise, stress failure due to high pressure in the pulmonary vasculature may have occurred and allowed the leakage of fluid into the interstitial space. This effect has been observed in racehorses which achieved high pulmonary pressures (West et al. 1993) and in exercising pigs (Schaffartzik et al., 1993). Schoene et al. (1986) suggested that permeability of the pulmonary capillaries may have also been altered and allowed fluid accumulation. Lymphatic system is one of the main mechanisms responsible for the clearance of the accumulated interstitial fluid. In the study by Coates et al. (1984) using sheep and goats, pulmonary lymph flow increased approximately 2.5 fold during intense exercise. However, highly trained athletes may accumulate more fluid than the lymphatic system can clear (Sheel, 1995) which could result in pulmonary edema that persists even after the exercise. Coates et al. (1984) also found a significant linear correlation between lymph flow and cardiac output ( $r=0.87$, $p<0.01$). In the present study, it was reasoned that if pulmonary edema does occur during strenuous exercise, then a post-exercise active recovery which would increase the post-exercise lymphatic flow should facilitate the recovery of the decrease in DM. The present study supports this theory.

DM can also be depressed as the result of a decrease in effective surface area for gas exchange due to a decrease in Vc and/or the worsening of ventilation to perfusion match (Pistelli et al. 1991). Sharratt et al. (1996) found significant decrease in DLco, DM

and Vc one hour after subjects performed a 10 minute exercise at 25% and 50 % $VO_{2max}$. It is unlikely that pulmonary edema would have developed at these low exercise intensities; therefore, the depressed DM was more likely to be due to a decreased surface area for gas exchange related to the reduction in central blood volume. In the present study, subjects experienced a similar pattern and magnitude in the reduction in Vc post-exercise in both conditions. Therefore, although we did not directly measure the between-condition difference in effective surface area available for gas exchange, it is unlikely that the difference in post-exercise DM can be linked to the changes in Vc. However, since we did not directly measure the alveolar ventilation with respect to perfusion in pulmonary capillaries, it is unknown if the practice of active recovery created a more homogeneous ventilation with respect to perfusion in the present study.

**Pulmonary capillary blood volume (Vc)**

With or without an active recovery period, subjects experienced a similar pattern of a significant reduction in Vc measured at one and two hours post-exercise. This confirms the findings in many previous studies (Sheel 1995; Hanel et al, 1993 ; Stewart et al. 1996 ; Sharratt et al. 1996). Lama (1996) suggested that the depressed post-exercise Vc may be a reflection of the redistribution of blood flow consequent to heavy exercise, as blood flow is shunted away from the thorax to clear metabolic waste products from exercised muscle. However, Sheel (1995) has shown that after a short bout (120 seconds) of maximal exercise subjects' post-exercise Vc remains depressed for at least 6 hours post-exercise, therefore making the redistribution of blood flow an unlikely explanation for the

depressed Vc long after the end of exercise. This present study seems to support this view. It was reasoned that if the redistribution of blood flow was an important factor for depressed post-exercise Vc, then a 30-minute active recovery at low intensity which facilitates the removal of metabolic waste products should recover the depressed Vc to a certain degree. Our data did not support the importance of the redistribution of blood flow on the alteration in Vc following heavy exercise.

So could the depressed Vc after exercise be a gravitational effect ? In the present study and several other ones ( Stewart, 1997; Sheel 1995; Lama, 1996; Sharratt et al. 1996), subjects were advised to remained seated for at least 20 minutes in the upright position before the DLco measurement. We suspect that the change in blood distribution due to prolonged upright resting and gravity could cause the reduction in the central blood volume therefore to depress Vc. Sharratt et al. (1996) studied a group (N=8) of healthy subjects and found a significant reduction in post-exercise Vc regardless of exercise intensity ( 25%, 50% or 75% $VO_{2max}$). It is unlikely that 10 minutes of cycling at an intensity equivalent to 25% or 50% of $VO_{2max}$ would cause a significant accumulations of metabolic waste product in the exercised legs or a significant redistribution of blood which would last for an hour after exercise. Therefore, the reductions in post-exercise Vc might be more of a postural effect rather than an exercise effect. Several studies have looked at the effect of redistribution of blood due to postural change on DL, DM and Vc. Initial studies by Bates and Pearce (1956) and Ogilvie et al.(1957) found an increase in DLco when changing from a sitting to a supine position. The increase in DLco has been attributed to an increase in Vc in the upper pulmonary lobes (Lewis et al. 1958; Newman

1962). In the transition from sitting to supine position, there is an increase in the pulmonary blood flow as venous return to the thorax increases. Prisk et al (1993) found a 35% increase in Vc among 7 healthy subjects when moving from a standing to supine position with no exercise.

In the prolonged upright seated position, the inactivity eliminates the effect of the "muscle pump" in the lower limbs and also reduces the venous return, causing a reduction in central blood volume which can partially explain the decrease in post-exercise Vc. Nevertheless, this phenomenon needs further investigation. In the study by Sheel (1995), if we compare the Vc values measured at each time interval ( pre-exercise , 1hr, 2 hr, 4 hr, and 6 hr post-exercise), with constant measurement procedures ( i.e. subjects remain seated for 20 minutes before each measurement), the post-exercise Vc kept decreasing over time. Sheel's finding suggests that mechanisms other than shunting and gravitational blood pooling might play important role on the reduction of post-exercise Vc over time.

**Pulmonary Diffusion Capacity for Carbon Monoxide (DLco)**

It is known that DLco is reduced following short-term maximal exercise ( Sheel, 1995; Rasumussen, et al ,1992; Hanel et al ,1994) and endurance activities such as triathlons or marathons ( Manier et al., 1991 and 1993). This study confirms that DLco is depressed following 45 minutes of submaximal exercise with a maximal effort near the end. DLco was significantly depressed one hour post-exercise and continued to decrease during the second hour of post-exercise recovery, confirming the results by Sheel (1995)

in which he found the DLco kept declining and reached a minimum value at 6 hours post-exercise.

Thirty minutes of active recovery at an intensity equivalent to 10% maximum did not recover ( to baseline ) the depressed DLco measured at one and two hours post-exercise. Although no significant treatment effect was found, a significant condition x time interaction effect ( $F=4.13$, $P<0.05$) was demonstrated, suggesting that groups were heterogeneous over time (Figure 7). As mentioned before, the value of DLco is determined by DM, Vc and $\theta$ ; therefore, to explain the change in DLco pre- and post-exercise one has to look into the changes in the three components. By examining Figure 6 and 8, it seems that the non-significant treatment effect in DLco was due to the similar changes in Vc in both conditions, and the significant interaction effect in DLco was due to the significant treatment effect in DM.

**Effect of heart rate on the DLco measurement**

Previous studies have demonstrated a strong positive correlation between cardiac output and DL, DM and Vc ( $r= 0.92$; $r=0.71$ and $r=0.92$ respectively) during exercise ( Johnson et al, 1960). In a more recent study, Hsia et al. (1995) showed that DL, DM and Vc increase linearly with respect to cardiac output from rest to near-maximal exercise. In the present study, HR was found to be significantly elevated 1 hour post-exercise ($p <0.05$) in both conditions, and then returned to resting value 2 hours post-exercise. Assuming the stroke volume was similar pre- and post-exercise, cardiac output, although

not directly measured, probably remained elevated 1 hour post-exercise. Caillaud et al. (1995) pointed out that the elevated postrace HR observed after endurance races such as triathlon and marathon might mask part of the reduction in Vc and DL. Therefore, the decrement in DLco, Dm and Vc could be more profound if cardiac output remained similar pre- and 1 hour post-exercise. Nevertheless, the significant reductions in DLco and Vc during the second hour of recovery as observed in the present study could be partially explained by the significant reduction of cardiac output from 1 Hr Post-Ex to 2 Hr Post-Ex (Figure 9).

**Summary**

The main findings of this study confirm that there is a decrease in DL, DM and Vc following sustained submaximal exercise with a maximal effort near the end. Mean heart rate at one hour post-exercise was found to be higher than resting baseline, indicating that some of the decrease in DL, DM and Vc might have been masked by the elevated cardiac output. The most significant finding was that the depressed post-exercise DM was recovered by 30 minutes of active recovery, suggesting the presence of pulmonary edema during and after the sustained effort. Changes of Vc were in similar pattern and magnitude in both AR and IR conditions, suggesting that the distribution of central blood volume due to gravity might have greater effect on Vc than shunting mechanism. This study represents the first attempt to examine the effect of active recovery on post-exercise pulmonary diffusion capacity.

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## Appendix A: Review of the Literature

### A.1 Accumulation of interstitial pulmonary fluid : a diffusion limitation responsible for the development of EIH

Elite athletes undergo training adaptations in skeletal muscle and the cardiovascular system which may eventually surpass the capabilities of their pulmonary system (Dempsey and Fregosi, 1985). Using an exercising horse model, Wagner et al. (1989) suggested that approximately two-thirds of EIH can be related to diffusion limitation, and the accumulation of interstitial pulmonary fluid was proposed as the one of the key mechanisms responsible for the deterioration of pulmonary gas exchange during heavy exercise. A significant increase in membrane resistance to diffusion (1/DM) following heavy exercise has been demonstrated in several studies (Manier et al. 1991; Sheel, 1995 ; Stewart et al. 1997), and indirectly supports the occurrence of perivascular and/or alveolar wall edema. Since one of the primary factors which limits the rate of $O_2$ transfer through the alveolar membrane is the distance between the blood and gas phase of the lung tissue, an increase in this distance would decrease the diffusion capability of the respiratory system. Schaffartzik and co-workers (1992) found that recovery from exercise-induced $V_A/Qc$ mismatch is prolonged, requiring 20 minutes to recover after exercise. This result is consistent with the hypothesis that pulmonary interstitial edema is one of the mechanisms worsening pulmonary gas exchange during exercise (Schaffartzik et al. 1992).

## A.2 Pulmonary diffusion capacity during exercise

Normally there is a large physiological reserve of diffusion capacity, shown by nearly a twofold increase in DLco from rest to peak exercise (Hsia et al. 1992 and 1995). A major determinant of DLco that has not generally been taken into consideration in routine pulmonary function testing is variation in pulmonary blood flow (Qc). During exercise, DLco normally increases as a linear function of pulmonary blood flow; to a lesser extent it is also related to changes in lung volume (Hsia et al. 1995). DM and Vc have also been reported to increase with increasing pulmonary blood flow (Hsia et al. 1994); hence, differences in pulmonary blood flow might lead to significant intra- and inter-subject variability of the measurements. The anatomic basis for the increase in DLco, DM and Vc during exercise is still a matter of debate, but several potential courses of augmentation have been postulated, as follows: 1) capillaries open or become distended as pulmonary blood flow or pressure increases (Hanson et al. 1989); 2) the alveolar membrane unfolds and the effective surface area for gas exchange increases as lung volume increases (Bachofen et al. 1987); and 3) the distribution of red blood cell transit times within the capillary network becomes more homogeneous as flow increases (Presson et al. 1994). If all other factors remain constant, DL could be increased either by increasing DM or Vc or both. An increase in DM could be due to an increase in the effective area available for diffusion by perfusion of areas ventilated but not previously perfused, or by ventilation of areas perfused but not previously ventilated (Danzer et al. 1968).

### A. 3 Effect of exercise on the post-exercise pulmonary diffusion capacity

Several studies have shown that DLco is significantly reduced following strenuous short - term exercise (Sheel, 1995; Hanel et al. 1993 and 1994; Stewart et al. 1996) and long-term exhaustive exercise such as triathlons or marathons (Caillaud et al. 1995; Miles et al. 1983; Manier et al. 1991). DL can be partitioned (Roughton and Forster, 1957) into the diffusion capacity of the blood gas barrier (DM) and the gas uptake capacity of capillaries, which is a function of the product of the specific blood gas conductance ($\theta$) and pulmonary capillary blood volume (Vc). The overall relationship can be expressed as follows :

$$\frac{1}{DLco} = \frac{1}{DM} + \frac{1}{\theta \times Vc}$$

Therefore, DL could be decreased either by decreasing DM or Vc or both.

#### A.3.1 Post-exercise alveolar-capillary membrane diffusion capacity (DM)

The structural determinant of DM is the ratio of the effective diffusion surface to the total barrier thickness. The effective surface is formulated as a fraction of the alveolar surface area, the most robust measure of lung design, whereas the effective barrier thickness is the harmonic mean distance between alveolar surface and erythrocyte surface (Weibel et al. 1993). Therefore, DM decreases when effective exchange area decreases, or when harmonic thickness increases.

*Alveolar-capillary membrane thickness :*

One of the main factors that can increase the alveolar capillary membrane thickness is the presence of subclinical pulmonary edema. According to Weibel et al. (1993), one of the four structurally distinct compartments that constitute the model for estimating the diffusion path across the alveolar-capillary membrane is the surface lining layer. The thickness of the surface lining layer is subject to functional variation in pulmonary fluid balance and will increase when alveolar edema forms (Bachofen et al. 1988). Wagner et al. (1986) suggested that during heavy exercise stress failure of the pulmonary capillaries and injury to the vascular endothelium at high capillary hydrostatic pressure may cause leakage of fluid and transient pulmonary edema. Several authors have speculated that subclinical pulmonary edema is present following maximal exercise (Wagner et al. 1986; Manier et al. 1991 and 1993; Miles et al. 1983). The evidence for this includes increases in residual volume, decreases in transthoracic electrical impedance and a decrease in resting pulmonary diffusion capacity.

Studies using computerized tomography (CT scan) or magnetic resonance imaging (MRI) to detect the increase in lung density and water content as the direct evidence for subclinical pulmonary edema have shown mixed results (Caillaud et al. 1995; McKenzie et al. 1996; Gallagher et al. 1988). McKenzie et al. (1996) found no changes in lung density using CT scan and MRI; however, a significant decrease in DLco was observed 90 minutes after 5 minutes of high intensity exercise on the cycle ergometer in 8 well-trained male cyclists. The results of this study indicate that either a detectable amount of pulmonary edema did not develop after the short bout of intense exercise, or interstitial

fluid did accumulate but was cleared within 90 minutes of exercise. To investigate the role of exercise duration on the development of pulmonary edema, Caillaud et al. (1995) studied 8 triathletes and found a significant increase in mean lung density using CT scanning, and a significant decrease in DLco after performing 120±20 minutes of exercise. This suggests the presence of mild subclinical pulmonary edema which would partially explain the decrease in DLco.

After reviewing the afore-mentioned studies (McKenzie et al. 1996, Caillaud et al. 1995, Manier et al. 1991 and 1993), it appears that if the accumulation of interstitial fluid occurs during strenuous exercise, the amount of fluid that accumulates is a function of the exercise duration. This would then explain the results by McKenzie et al. (1996) that 5 minutes of maximal exercise did not cause a detectable amount of interstitial fluid accumulation using MRI and CT scanning techniques. Schaffartzik et al. (1992) suggested that the failure of some other studies (Gallagher et al., 1988 ; Marshall et al., 1971) to directly detect increased pulmonary extravascular water may be a consequence of the small volume of fluid that is involved. The increase in extravascular lung water caused by exercise may not be sufficient to be seen by relatively insensitive methods such as indicator-dilution and X-ray techniques.

*Effective diffusion surface:*

There are three anatomically identifiable surfaces that are related to the diffusion field: the outer alveolar epithelial surface, the inner capillary endothelial surface, and the

erythrocyte surface (Weibel et al. 1993). The total barrier surface can be estimated by the area of the active diffusion front, considering a diffusion vector that extends from the alveolar surface to the erythrocyte surface. According to Weibel et al. (1993), to estimate the active erythrocyte surface poses a number of problems. For example, very often two red cells are so close to each other that a significant part of their membrane surface is not exposed to the $O_2$ diffusion path.

Post-exercise changes to the effective diffusion surface has yet to be investigated. Studies utilizing postural changes have suggested that the changes in DM can be related to: 1) how homogeneously distributed ventilation is with respect to the diffusion surface (Pistelli et al, 1991); and 2) the changes in Vc (Stewart, et al. 1997). Using a multiple inert gas technique, Wagner and co-workers (1986) provided evidence of greater ventilation/perfusion ($V_A$/Qc) mismatching during exercise, with the abnormalities persisting for at least several minutes after exercise. Schaffartzik et al. (1992) found that a significant $V_A$/Qc mismatch may persist for up to 20 minutes after exercising at near maximal intensity in hypoxic condition. Since the effective diffusion surface decreases as a result of increased $V_A$/Qc mismatching, the results from the previous two studies can partially explain the significantly depressed DM observed 30 minutes after marathon running or incremental exercise testing to volitional fatigue (Manier et al. 1991 and 1993).

### A. 3.2 Post-exercise pulmonary capillary blood volume (Vc):

Numerous studies have demonstrated a significant reduction in Vc following submaximal exercise ( Hanel et al. 1993 and Sharratt et al. 1996) and maximal exercise (Sheel, 1995 ;

Hanel et al. 1994; Lama, 1995; Stewart et al. 1996 and 1997). A reduction in central blood volume following exercise has also been previously described using trans-thoracic electrical impedance (Rasumussen et al., 1992).

A reduction in Vc has been shown to account for a large portion of the decrease in pulmonary diffusion capacity following exercise ( Sheel, 1995 ; Hanel et al. 1993). Hanel and her co-workers (1993) have shown that even a short bout (6 minutes) of submaximal (61% $VO_{2max}$) exercise can significantly affect post-exercise DLco. She suggested that since a significant change in pulmonary capillary membrane integrity and the presence of subclinical pulmonary edema were unlikely to occur during mild exercise, a fall in central blood volume might explain the decrease in DLco following exercise. Sheel (1995) found that the degree of change in Vc parallels the change in DLco following a short bout of strenuous exercise, suggesting that the majority of decrease in DLco can be attributed to a lower pulmonary capillary blood volume.

Mechanisms responsible for the reduction in Vc following exercise are still unclear. Lama (1995) suggested that the observed post-exercise decrease in Vc may reflect a redistribution of blood flow, as blood flow is shunted away from the thorax to clear metabolic waste products from exercised muscles.

Appendix B: Individual Subject Data
Table B1: Anthropometric and pulmonary function test data

| | Age (yrs) | Height (cm) | Mass (kg) | FVC (litres) | $FEV_{1.0}$ (litres) | $FEV_{1.0}/FVC$ |
|---|---|---|---|---|---|---|
| NR | 23 | 180.4 | 79.6 | 6.0 | 5.4 | 0.9 |
| IS | 25 | 170.0 | 63.6 | 4.3 | 3.9 | 0.9 |
| PC | 20 | 177.0 | 79.0 | 5.4 | 4.9 | 0.9 |
| BH | 20 | 172.0 | 57.5 | 4.9 | 4.4 | 0.9 |
| BM | 22 | 175.0 | 67.0 | 6.0 | 5.2 | 0.9 |
| DL | 24 | 181.4 | 66.6 | 6.3 | 5.3 | 0.8 |
| JO | 20 | 170.3 | 66.0 | 4.9 | 4.6 | 0.9 |
| MC | 23 | 177.5 | 60.6 | 5.9 | 4.8 | 0.8 |
| ID | 23 | 193.9 | 85.0 | 7.5 | 5.9 | 0.8 |
| MW | 23 | 177.7 | 76.5 | 5.4 | 4.7 | 0.9 |
| Mean | 22 | 177.5 | 70.1 | 5.7 | 4.9 | 0.9 |
| ± SD | 2 | 7.0 | 9.2 | 0.9 | 0.6 | 0.0 |

Table B2: Maximal exercise test data (VO2max test) and the predicted values for HR at ventilatory threshold (HR@VT) and power at VT (POWER@VT).

| | HRmax (bpm) | VO2max (L/min) | VO2max ml/kg/min | POWERpeak (watts) | %SaO2min | HR@VT (bpm) | POWER@VT (watts) |
|---|---|---|---|---|---|---|---|
| NR | 195 | 5.2 | 65.8 | 451 | 93.6 | 176 | 357 |
| IS | 192 | 3.6 | 56.3 | 345 | 95.2 | 165 | 233 |
| PC | 195 | 4.1 | 52.4 | 407 | 94.3 | 170 | 309 |
| BH | 202 | 3.4 | 59.2 | 354 | 90.4 | 175 | 231 |
| BM | 199 | 4.2 | 62.5 | 381 | 94.7 | 167 | 265 |
| DL | 188 | 4.4 | 66.7 | 439 | 94.1 | 169 | 320 |
| JO | 195 | 4.4 | 66.5 | 396 | 92.0 | 172 | 276 |
| MC | 171 | 3.7 | 61.3 | 376 | 94.1 | 160 | 267 |
| ID | 192 | 5.2 | 60.9 | 512 | 91.7 | 172 | 340 |
| MW | 189 | 4.6 | 59.6 | 442 | 91.1 | 165 | 320 |
| Mean | 192 | 4.3 | 61.1 | 410 | 93.1 | 169 | 292 |
| ± SD | 8 | 0.6 | 4.6 | 51 | 1.7 | 5 | 44 |

Table B3. Mean HR (HR@Ex) in AR and IR, mean power (POWER@Ex) and peak power (POWERpeak) during the 45-minute submaximal exercise, and mean heart rate (HR@AR) and mean power (POWER@AR) during the active recovery period.

| SUBJECT | HR@Ex in AR (bpm) | HR@Ex in IR (bpm) | POWER@Ex (watts) | POWERpeak (watts) | HR@AR (bpm) | POWER@AR (watts) |
|---|---|---|---|---|---|---|
| NR | 172 | 175 | 313 | 430 | 101 | 45 |
| IS | 168 | 176 | 194 | 350 | 116 | 35 |
| PC | 179 | 175 | 250 | 400 | 108 | 41 |
| BH | 181 | 178 | 190 | 345 | 137 | 35 |
| BM | 159 | 168 | 215 | 340 | 107 | 38 |
| DL | 171 | 168 | 259 | 415 | 112 | 44 |
| JO | 177 | 180 | 249 | 396 | 105 | 40 |
| MC | 153 | 154 | 233 | 352 | 83 | 38 |
| ID | 174 | 167 | 298 | 515 | 114 | 51 |
| MW | 162 | 165 | 249 | 454 | 106 | 44 |
| Mean | 170 | 171 | 245 | 400 | 109 | 41 |
| ± SD | 9 | 8 | 40 | 56 | 13 | 5 |

Table B4. Individual heart rate prior to each pre- and post-exercise pulmonary diffusion measurement in AR and IR conditions.

| Active Recovery | | | | Inactive Recovery | | |
|---|---|---|---|---|---|---|
| SUBJECT | Pre-Ex | 1 Hr Post-Ex | 2 Hr Post-Ex | Pre-Ex | 1 Hr Post-Ex | 2 Hr Post-Ex |
| NR | 59 | 62 | 60 | 56 | 63 | 54 |
| IS | 58 | 64 | 65 | 60 | 63 | 63 |
| PC | 63 | 74 | 66 | 62 | 74 | 69 |
| BH | 78 | 86 | 80 | 69 | 84 | 80 |
| BM | 72 | 66 | 55 | 77 | 86 | 70 |
| DL | 58 | 65 | 56 | 59 | 63 | 53 |
| JO | 61 | 76 | 66 | 65 | 82 | 67 |
| MC | 56 | 60 | 55 | 56 | 69 | 64 |
| ID | 65 | 74 | 68 | 66 | 77 | 71 |
| MW | 56 | 66 | 61 | 58 | 75 | 58 |
| Mean | 63 | 69 | 63 | 63 | 73 | 65 |
| ± SD | 7 | 8 | 8 | 6 | 9 | 8 |

Table B5 Pulmonary diffusion capacity (DLco), alveolar-capillary membrane diffusion capacity (DM) and pulmonary capillary blood volume (Vc) pre and post 45 minute submaximal exercise test in Active Recovery condition.

| SUBJECT | TEST | DLco | DM | Vc |
|---|---|---|---|---|
| NR | Pre-Ex | 32.1 | 45.3 | 60.8 |
| | 1 Hr Post-Ex | 34.3 | 50.2 | 59.7 |
| | 2 Hr Post-Ex | 32.3 | 49.3 | 51.7 |
| IS | Pre-Ex | 31.9 | 46.4 | 56.0 |
| | 1 Hr Post-Ex | 29.4 | 42.2 | 53.1 |
| | 2 Hr Post-Ex | 27.8 | 41.3 | 46.4 |
| PC | Pre-Ex | 33.0 | 44.2 | 64.5 |
| | 1 Hr Post-Ex | 34.0 | 48.3 | 57.1 |
| | 2 Hr Post-Ex | 32.7 | 44.2 | 62.4 |
| BH | Pre-Ex | 31.5 | 46.5 | 58.1 |
| | 1 Hr Post-Ex | 27.4 | 39.5 | 53.4 |
| | 2 Hr Post-Ex | 26.9 | 49.4 | 35.1 |
| BM | Pre-Ex | 35.8 | 51.5 | 62.5 |
| | 1 Hr Post-Ex | 33.8 | 49.3 | 56.8 |
| | 2 Hr Post-Ex | 32.0 | 47.4 | 51.9 |
| DL | Pre-Ex | 31.8 | 44.8 | 56.4 |
| | 1 Hr Post-Ex | 30.0 | 42.8 | 51.6 |
| | 2 Hr Post-Ex | 29.6 | 41.7 | 52.4 |
| JO | Pre-Ex | 29.0 | 40.3 | 58.0 |
| | 1 Hr Post-Ex | 25.7 | 38.8 | 42.6 |
| | 2 Hr Post-Ex | 24.1 | 36.3 | 40.2 |
| MC | Pre-Ex | 26.1 | 37.6 | 39.1 |
| | 1 Hr Post-Ex | 25.3 | 34.8 | 41.7 |
| | 2 Hr Post-Ex | 24.7 | 36.8 | 34.3 |
| ID | Pre-Ex | 27.8 | 42.0 | 64.7 |
| | 1 Hr Post-Ex | 29.1 | 40.9 | 58.7 |
| | 2 Hr Post-Ex | 29.4 | 43.6 | 50.5 |
| MW | Pre-Ex | 35.8 | 50.2 | 67.8 |
| | 1 Hr Post-Ex | 33.5 | 46.0 | 66.4 |
| | 2 Hr Post-Ex | 32.1 | 46.5 | 56.2 |

Table B6 Pulmonary diffusion capacity (DLco), alveolar-capillary membrane diffusion capacity (DM) and pulmonary capillary blood volume (Vc) pre and post 45 minute submaximal exercise test in Inactive Recovery condition.

| SUBJECT | TEST | DLco | DM | Vc |
|---|---|---|---|---|
| NR | Pre-Ex | 34.5 | 48.0 | 65.4 |
| | 1 Hr Post-Ex | 28.4 | 37.9 | 62.2 |
| | 2 Hr Post-Ex | 29.3 | 42.2 | 52.1 |
| IS | Pre-Ex | 32.5 | 45.4 | 57.9 |
| | 1 Hr Post-Ex | 31.5 | 42.8 | 59.8 |
| | 2 Hr Post-Ex | 29.7 | 39.9 | 58.2 |
| PC | Pre-Ex | 34.4 | 47.4 | 65.7 |
| | 1 Hr Post-Ex | 32.1 | 43.4 | 64.2 |
| | 2 Hr Post-Ex | 31.9 | 48.5 | 48.4 |
| BH | Pre-Ex | 34.6 | 49.8 | 60.0 |
| | 1 Hr Post-Ex | 30.4 | 45.6 | 48.4 |
| | 2 Hr Post-Ex | 28.8 | 40.9 | 50.4 |
| BM | Pre-Ex | 34.4 | 48.5 | 68.4 |
| | 1 Hr Post-Ex | 34.1 | 49.7 | 67.9 |
| | 2 Hr Post-Ex | 30.8 | 41.9 | 60.3 |
| DL | Pre-Ex | 32.5 | 48.4 | 50.8 |
| | 1 Hr Post-Ex | 27.3 | 37.8 | 50.6 |
| | 2 Hr Post-Ex | 25.9 | 38.6 | 40.4 |
| JO | Pre-Ex | 28.7 | 41.7 | 49.6 |
| | 1 Hr Post-Ex | 24.5 | 34.6 | 45.4 |
| | 2 Hr Post-Ex | 20.7 | 29.6 | 37.3 |
| MC | Pre-Ex | 26.4 | 37.8 | 43.9 |
| | 1 Hr Post-Ex | 25.1 | 37.1 | 42.8 |
| | 2 Hr Post-Ex | 22.8 | 34.4 | 36.9 |
| ID | Pre-Ex | 28.6 | 39.4 | 64.1 |
| | 1 Hr Post-Ex | 31.6 | 44.8 | 53.5 |
| | 2 Hr Post-Ex | 30.2 | 41.2 | 56.3 |
| MW | Pre-Ex | 36.6 | 49.8 | 72.5 |
| | 1 Hr Post-Ex | 32.1 | 45.3 | 59.8 |
| | 2 Hr Post-Ex | 26.5 | 37.4 | 49.1 |

**Table B7 Hemoglobin (Hb) and body weight pre- and post- 45 minutes of submaximal exercise test in Active Recovery condition.**

| SUBJECT | TEST | Hb (g / dL) | Weight (Kg) |
|---|---|---|---|
| NR | Pre-Ex | 14.8 | 79.2 |
| | 1 Hr Post-Ex | 14.5 | 79.3 |
| | 2 Hr Post-Ex | 15.4 | 79.4 |
| IS | Pre-Ex | 14.5 | 63.9 |
| | 1 Hr Post-Ex | 14.7 | 64.1 |
| | 2 Hr Post-Ex | 14.7 | 64.0 |
| PC | Pre-Ex | 16.6 | 78.7 |
| | 1 Hr Post-Ex | 16.5 | 78.8 |
| | 2 Hr Post-Ex | 16.4 | 78.9 |
| BH | Pre-Ex | 13.7 | 57.5 |
| | 1 Hr Post-Ex | 14.5 | 57.5 |
| | 2 Hr Post-Ex | 15.4 | 57.6 |
| BM | Pre-Ex | 15.6 | 67.2 |
| | 1 Hr Post-Ex | 15.5 | 67.4 |
| | 2 Hr Post-Ex | 15.1 | 67.6 |
| DL | Pre-Ex | 16.0 | 66.5 |
| | 1 Hr Post-Ex | 15.8 | 66.8 |
| | 2 Hr Post-Ex | 15.6 | 66.6 |
| JO | Pre-Ex | 15.5 | 67.7 |
| | 1 Hr Post-Ex | 15.5 | 68.0 |
| | 2 Hr Post-Ex | 15.3 | 68.1 |
| MC | Pre-Ex | 14.6 | 60.6 |
| | 1 Hr Post-Ex | 15.2 | 60.5 |
| | 2 Hr Post-Ex | 15.0 | 60.7 |
| ID | Pre-Ex | 16.3 | 85.3 |
| | 1 Hr Post-Ex | 16.5 | 85.4 |
| | 2 Hr Post-Ex | 15.9 | 85.3 |
| MW | Pre-Ex | 15.1 | 76.2 |
| | 1 Hr Post-Ex | 15.8 | 76.3 |
| | 2 Hr Post-Ex | 15.3 | 76.4 |

**Table B8 Hemoglobin (Hb) and body weight pre- and post- 45 minutes of submaximal exercise test in Inactive Recovery condition.**

| SUBJECT | TEST | Hb (g / dL) | Weight (Kg) |
|---|---|---|---|
| NR | Pre-Ex | 14.8 | 79.4 |
| | 1 Hr Post-Ex | 14.5 | 80.0 |
| | 2 Hr Post-Ex | 15.0 | 79.8 |
| IS | Pre-Ex | 15.2 | 63.7 |
| | 1 Hr Post-Ex | 15.5 | 63.9 |
| | 2 Hr Post-Ex | 14.9 | 64.0 |
| PC | Pre-Ex | 15.8 | 78.7 |
| | 1 Hr Post-Ex | 16.2 | 78.8 |
| | 2 Hr Post-Ex | 16.0 | 78.9 |
| BH | Pre-Ex | 14.9 | 57.6 |
| | 1 Hr Post-Ex | 15.5 | 57.7 |
| | 2 Hr Post-Ex | 15.1 | 57.8 |
| BM | Pre-Ex | 15.9 | 67.0 |
| | 1 Hr Post-Ex | 15.5 | 67.2 |
| | 2 Hr Post-Ex | 16.0 | 67.3 |
| DL | Pre-Ex | 16.0 | 66.7 |
| | 1 Hr Post-Ex | 16.3 | 66.8 |
| | 2 Hr Post-Ex | 15.6 | 66.7 |
| JO | Pre-Ex | 13.3 | 67.9 |
| | 1 Hr Post-Ex | 14.2 | 68.1 |
| | 2 Hr Post-Ex | 13.7 | 68.1 |
| MC | Pre-Ex | 15.0 | 60.4 |
| | 1 Hr Post-Ex | 15.2 | 60.5 |
| | 2 Hr Post-Ex | 15.1 | 60.7 |
| ID | Pre-Ex | 15.7 | 85.5 |
| | 1 Hr Post-Ex | 15.6 | 85.7 |
| | 2 Hr Post-Ex | 16.1 | 85.6 |
| MW | Pre-Ex | 15.1 | 76.6 |
| | 1 Hr Post-Ex | 15.3 | 76.8 |
| | 2 Hr Post-Ex | 15.3 | 76.9 |

Appendix C
Statistical Analyses

**ANOVA table for DLco**

| | DF | SS | MS | F-Value | P-Value |
|---|---|---|---|---|---|
| TIME | 2 | 123.03 | 61.52 | 17.08 | < 0.001 |
| RECOVERY METHOD | 1 | 2.36 | 2.36 | 0.54 | 0.48 |
| INTERACTION | 2 | 13.63 | 6.82 | 4.13 | 0.033 |

**ANOVA table for DM**

| | DF | SS | MS | F-Value | P-Value |
|---|---|---|---|---|---|
| TIME | 2 | 144.63 | 72.31 | 8.63 | 0.002 |
| RECOVERY METHOD | 1 | 39.38 | 39.38 | 6.26 | 0.034 |
| INTERACTION | 2 | 60.91 | 30.45 | 3.18 | 0.066 |

**ANOVA table for Vc**

| | DF | SS | MS | F-Value | P-Value |
|---|---|---|---|---|---|
| TIME | 2 | 1172.28 | 586.14 | 46.79 | < 0.001 |
| RECOVERY METHOD | 1 | 17.11 | 17.11 | 0.52 | 0.488 |
| INTERACTION | 2 | 0.67 | 0.34 | 0.02 | 0.985 |

**ANOVA table for HR**

| | DF | SS | MS | F-Value | P-Value |
|---|---|---|---|---|---|
| TIME | 2 | 871.87 | 435.93 | 18.69 | < 0.001 |
| RECOVERY METHOD | 1 | 67.42 | 67.42 | 1.81 | 0.212 |
| INTERACTION | 2 | 41.88 | 20.94 | 2.71 | 0.093 |

**ANOVA table for Hb**

| | DF | SS | MS | F-Value | P-Value |
|---|---|---|---|---|---|
| TIME | 2 | 0.39 | 0.20 | 1.66 | 0.217 |
| RECOVERY METHOD | 1 | 0.15 | 0.15 | 0.21 | 0.655 |
| INTERACTION | 2 | 0.01 | 0.00 | 0.04 | 0.957 |

**ANOVA table for Weight**

| | DF | SS | MS | F-Value | P-Value |
|---|---|---|---|---|---|
| TIME | 2 | 0.39 | 0.20 | 1.66 | 0.217 |
| RECOVERY METHOD | 1 | 0.15 | 0.15 | 0.21 | 0.655 |
| INTERACTION | 2 | 0.01 | 0.00 | 0.04 | 0.957 |