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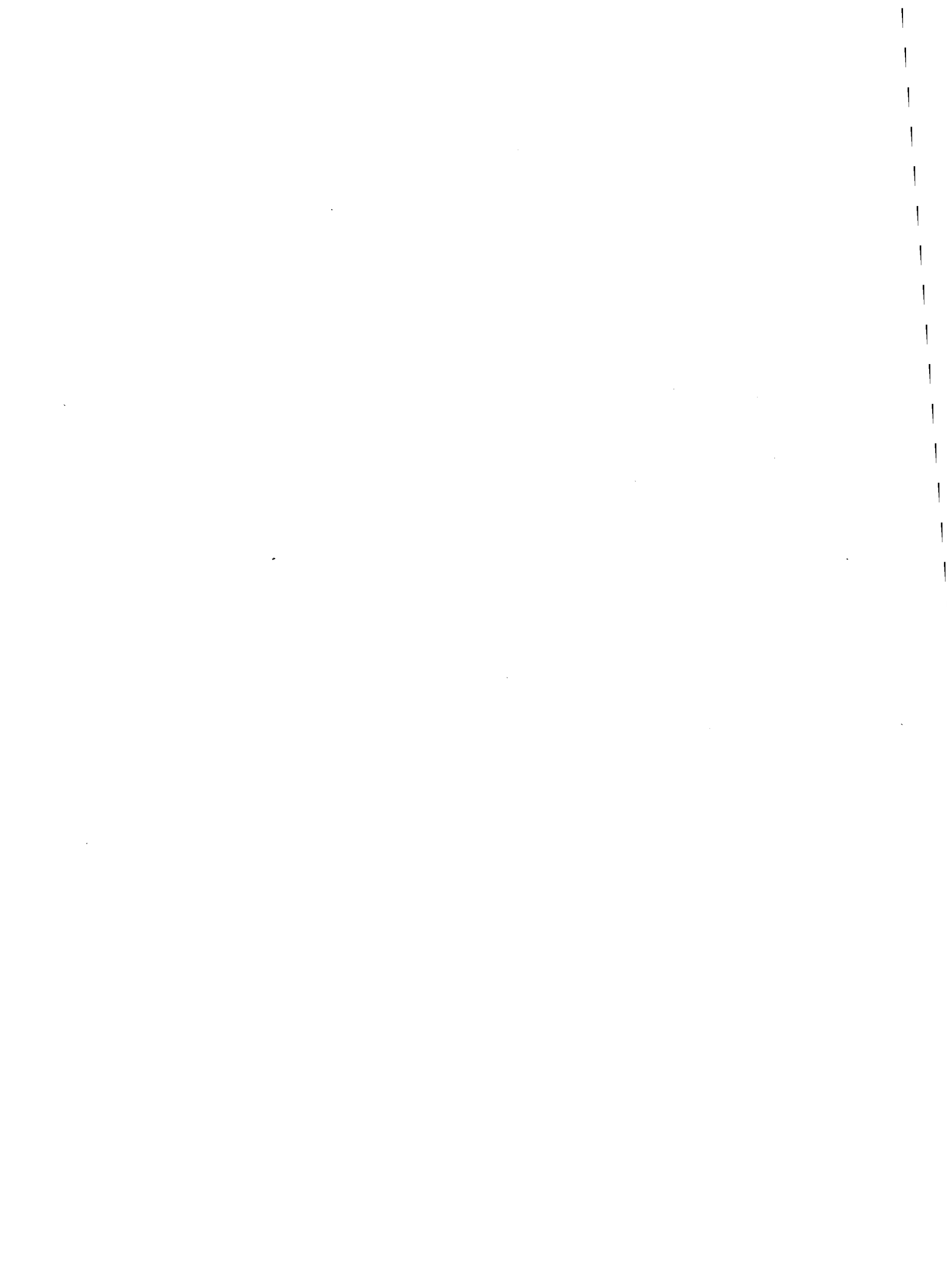
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THE EFFECT OF AN ACUTE BOUT OF EXERCISE ON SELECTED  
PULMONARY FUNCTION MEASUREMENTS

*The University of Arizona*

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THE EFFECT OF AN ACUTE BOUT OF EXERCISE ON  
SELECTED PULMONARY FUNCTION MEASUREMENTS

by  
Michael Joseph Buono

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A Dissertation Submitted to the Faculty of the  
COMMITTEE ON ANIMAL PHYSIOLOGY (GRADUATE)  
In partial fulfillment of the Requirements  
For the Degree of  
DOCTOR OF PHILOSOPHY  
In the Graduate College  
THE UNIVERSITY OF ARIZONA

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THE UNIVERSITY OF ARIZONA  
GRADUATE COLLEGE

As members of the Final Examination Committee, we certify that we have read  
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entitled THE EFFECT OF AN ACUTE BOUT OF EXERCISE ON SELECTED  
PULMONARY FUNCTION MEASUREMENTS

and recommend that it be accepted as fulfilling the dissertation requirement  
for the Degree of Doctor of Philosophy.

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College.

I hereby certify that I have read this dissertation prepared under my  
direction and recommend that it be accepted as fulfilling the dissertation  
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Jack H. Wilmore  
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16 May 1982  
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SIGNED: Michael J. Buono

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## ABSTRACT

A series of five studies were conducted to examine the effect of exercise on selected pulmonary function measurements. Studies I and II determined the effect of an acute bout of exercise on various lung volumes immediately post-exercise and over a 24-hour post-exercise period. There were significant mean increases of 210 ml (20.6%) and 260 ml (20.8%) in the 5-minute post-exercise residual volume (RV) measurement for studies I and II, respectively. There also were significant mean increases of 170 ml (3.4%) and 190 ml (2.7%) in the 5-minute post-exercise total lung capacity (TLC) for studies I and II, respectively, while vital capacity (VC) remained unchanged. RV and TLC remained significantly increased over the pre-exercise values through 30 and 15 minutes of recovery, respectively. Studies III through V were undertaken to determine the physiological mechanism underlying the responses reported in studies I and II.

In study III, transthoracic electrical impedance (TEI) was significantly decreased below the pre-exercise value through 30-minutes of recovery, indicating that there was an increase in thoracic fluid volume following exercise. However, TEI measurements alone cannot separate between intra- and extravascular fluid shifts. Therefore, studies IV and V attempted to identify whether the decrease in TEI and increase in RV reported in study III were due to intra- or extravascular fluid shifts. Study IV examined the TEI, RV, and TLC responses before

and following exercise, as central blood volume (CBV) was experimentally increased via G-suit inflation, and decreased via venous occlusion tourniquets. The results suggest that RV is relatively insensitive to intravascular volume shifts within the thorax. Study V determined and followed the effect of an acute bout of exercise on lung diffusion capacity ( $D_{LCO}$ ).  $D_{LCO}/V_A$  did not increase significantly following exercise, suggesting that the decrease in TEI following exercise is the result of extravascular fluid accumulation.

It was concluded that a sub-clinical pulmonary edema occurs following exercise. A logical sequence of events based on the results of studies I through V was proposed as a possible explanation for the responses of RV and TLC following exercise.

## CHAPTER 1

### INTRODUCTION

Following a maximal expiration, a certain volume of air remains in the lungs, i.e. the residual volume (RV). The RV serves an important physiological function as it allows for uninterrupted gas exchange between the alveoli and the blood throughout the various phases of the respiratory cycle. Since there is always air in the lungs, oxygen and carbon dioxide exchange can occur even during a forced expiration, thus maintaining relatively constant gas tensions in arterial blood.

In recent years, several studies have shown that RV increases during exercise (10,30) and remains elevated immediately upon completion of exercise (6,20,28). Although the effect of exercise on RV is well documented, the physiological mechanisms responsible for the increase are unclear. Also, none of the previous studies (6,10,20,28,30) examined the time course of RV as it returned to the pre-exercise value following exercise.

Therefore, in an attempt to help answer some of the above questions, a series of five different studies were conducted to examine several aspects of the effect of an acute bout of exercise on RV and other selected pulmonary function measurements. The five studies represent the collected efforts of over two years of research, with each preceding study being designed to help answer questions which arose during the previous one. Hopefully, the five studies represent a

logical progression of thought, aimed at addressing several unanswered questions in the field of exercise physiology.

### Review of Literature

During exercise the increased utilization of oxygen and increased production of carbon dioxide impose increased demands on the respiratory system. These metabolic demands are met by an increased minute ventilation, tidal volume, breathing frequency, and pulmonary blood flow. The mechanisms involved in these adaptations have been thoroughly studied and have been reviewed by Åstrand and Rodahl (1). The effect of an acute bout of exercise on post-exercise pulmonary function measurements, however, have been relatively neglected.

Several studies have shown that RV increases during exercise (10,20) and remains elevated immediately following exercise (6,20,28). Hanson et al. (10) found a mean increase of 22% and 3.4% in RV and total lung capacity (TLC), respectively, during level walking (3 mph) on a treadmill. In a more recent study, Stubbing et al. (30) measured RV and TLC during progressive cycle ergometry. Measurements were recorded at rest and then at workloads starting at  $200 \text{ kpm}\cdot\text{min}^{-1}$ , to a maximum of  $800 \text{ kpm}\cdot\text{min}^{-1}$ . The RV increased 5.9%, 8.6%, 9.5%, and 15.4% at the 200, 400, 600, and  $800 \text{ kpm}\cdot\text{min}^{-1}$  workloads, respectively. The mean exercise TLC values were not significantly different from the pre-exercise values, however, they did report increases as large as 390 ml in one subject. Maron et al. (20) found a mean increase of 24% in RV immediately following a marathon. The RV had returned to the pre-exercise value when the subjects were retested 24-hours following the

race. Unfortunately, these were the only two post-exercise RV measurements performed. Therefore, it is impossible to determine the RV recovery time course from this data.

Although the effect of exercise on RV is well-documented, the physiological mechanisms responsible for the increase are unclear. Basically, three possible theories have developed. First, if there is peripheral airway constriction following exercise, the airways would close sooner during expiration, thus trapping more air. The increased volume of trapped air would increase the RV. However, a recent study by Doerr et al. (4) suggests that peripheral airway constriction does not occur following exercise. They administered a bronchodilator (isoproterenol) following a marathon and found no effect on lung volumes or flow rates as compared to the post-race values without the bronchodilator. They concluded that enhanced bronchial smooth muscle tone did not occur following exercise.

According to the second theory, if CBV was reduced following exercise, there would be more room in the thoracic cavity for air, thus resulting in an increased RV (21). Along these lines, Braunwald and Kelly (3) have shown that central blood volume (CBV), as assessed by the indicator-dilution technique, decreased below pre-exercise values following 10 minutes of moderately heavy leg exercise. Also, Okada et al. (25), using a radiographic technique, found a mean exercise/rest pulmonary blood volume ration of 0.95, suggesting a decrease in intrathoracic blood volume during exercise. These studies suggest that the increase in RV may be mediated by exercise-induced changes in CBV.

Recently, a third possible mechanism has been postulated (20). Maron et al. (20) suggest that if interstitial edema occurred following exercise, this could cause a decrease in the size of the bronchioles, thus causing them to close sooner during a forced expiration. It had previously been reported (9) that the diameter of the small airways is reduced during the early stages of pulmonary edema as a result of fluid accumulating in the peribronchial sheath.

Why would fluid accumulate in the lung during exercise? According to the Starling hypothesis for bulk capillary fluid transfer there are several factors that could lead to pulmonary edema: elevated capillary hydrostatic pressure, increased capillary permeability to plasma proteins, increased capillary surface area, decreased plasma colloid osmotic pressure, and decreased lymphatic drainage (29). At least two of these factors could promote fluid accumulation during exercise. First, several investigations have shown that pulmonary artery pressure increases 5-10 torr at the onset of exercise (5,19). This increase in capillary hydrostatic pressure would promote fluid accumulation. Also, since pulmonary capillary blood volume is increased during exercise (2), total capillary surface area for fluid exchange must also increase. These two factors would tend to increase interstitial edema formation during exercise.

Several studies (23,31) using the double indicator-dilution technique have shown an increase in pulmonary extravascular water volume (PEWV) during exercise in man. However, in a more recent study, Marshall et al. (22) using a direct dissection technique, concluded

that PEWV did not increase during exercise in dogs. The methods employed in that study, however, are only capable of detecting a 10% increase in PEWV (22). Hughes and Rosenzweig (8) have previously shown that the volume of trapped gas (VTG) in excised dog lungs increased 83% with a mere 8% increase in lung weight (i.e. interstitial edema). Also, increases in lung weight as small as 1% resulted in a 34% increase in VTG. Along these lines, Lemen et al. (18) have found a 44% increase in RV in alloxan-treated dogs, which was attributed to the development of PEWV, although there was no radiological or clinical evidence of pulmonary edema. Therefore, it would appear that small changes in peribronchial edema can bring about large changes in RV, as was shown by Hughes and Rosenzweig (8).

In recent years, several investigators have attempted to use transthoracic electrical impedance (TEI) as a more sensitive measure for the early detection of pulmonary edema (15,19,27). This technique is based on Ohm's Law, and the fact that various biological substances have different electrical conductivities (15,19). The impedance, or resistance, of the thorax to the flow of electricity varies inversely to the amount of fluid contained in the thorax. Ionized fluids have a comparatively low resistance (19), so TEI decreases as the volume of fluid in the thorax increases, and vice versa. Therefore, measurement of changes in TEI allows an indirect assessment of relative changes in thoracic fluid content. Pomerantz et al. (27) reported that TEI detects fluid accumulation within the thorax well before there is any indication of edema with the classical methods of monitoring. In a study they

performed on 43 dogs and 9 patients, they were able to detect fluid accumulation as small as 50 ml within the intact thorax. In a more recent study, Jaeger et al. (11), used TEI to determine if sub-clinical pulmonary edema was a common occurrence among non-acclimatized men following rapid ascent to 4300 meters. Upon physical examination no signs of pulmonary edema were present, and no definite radiographic evidence of fluid accumulation was found. However, TEI was significantly decreased and RV was significantly increased. Again, these results suggest that TEI may provide a more sensitive measurement of incipient pulmonary edema, and that small changes in interstitial edema can alter pulmonary function mechanics.

#### Purpose

The purpose of this series of studies was as follows. Study I was conducted to determine the immediate post-exercise effect of a bout of exercise on residual volume and selected pulmonary function measurements. Although similar studies have been reported in the past, it was felt that confirmation of previous research, and the establishment of procedures and techniques were necessary before further research was attempted. Study II was a repeated measures study designed to follow the effect of an acute bout of exercise on RV immediately post-exercise and at selected intervals over a 24-hour post-exercise period. This allowed for determination of the time course of the RV as it returned to normal. In an attempt to help determine whether transthoracic fluid shifts were the cause for the post-exercise pulmonary function responses, study III examined the relationship between TEI and RV

following exercise. Study IV determined the relationship between alterations in CBV and various lung volumes following exercise. CBV was experimentally altered via G-suit inflation (increasing CBV) and venous occlusion tourniquets (decreasing CBV). This allowed for determination of the responses of various post-exercise lung volumes to intravascular fluid shifts. Finally, study V determined the pre- and post-exercise carbon monoxide diffusion capacity ( $D_{LCO}$ ) of the lungs. This was done to help differentiate between intra- and extravascular fluid shifts, within the thorax, following exercise.

## CHAPTER 2

### EXPERIMENTAL DESIGN AND METHODOLOGY

#### Subjects

Participants in all studies were volunteers. All were non-smokers and free from respiratory disorders as determined by questionnaire. The subjects used in study I were 11 female runners from the University of Arizona cross country team. Subjects for studies II and III were 12 and 18 college-aged males, respectively. Subjects from study III were recruited to participate in studies IV (n=10) and V (n=5), respectively. The procedures for each study had been approved by the University of Arizona Human Subjects Committee and were carefully reviewed with each subject. Written informed consent was obtained prior to testing.

#### Methods

In all five studies, the subjects reported to the laboratory at least 3 hours post-absorptive, and age, height, and weight were obtained. In studies I through IV the pre-exercise RV was determined using the closed-circuit oxygen dilution technique described by Wilmore (34). The reproducibility of this method was established on a sample of 195 men with a correlation of 0.993 and a standard error of 28 ml (34). A minimum of two RV measurements were administered to each subject. If necessary, additional trials were administered until two

trials within 100 ml were obtained. The mean of these two measurements is the reported value. Each subject then performed two forced vital capacity (FVC) maneuvers using a Collins 13.5 liter spirometer. Subsequently, the largest FVC tracing for each subject was analyzed according to the methods of Kory et al. (14). All pulmonary function measurements were converted to BTPS conditions.

In all studies the subjects performed continuous treadmill tests to exhaustion on a Quinton motor-driven treadmill. The protocol consisted of a 2-minute warm up at 3 mph, followed by 2-minutes at 6 mph, and 2-minutes at 8 mph, each at 0% grade. Thereafter, the grade was increased 2% per minute, with the speed remaining constant, until exhaustion. Oxygen uptake ( $\dot{V}O_2$ ), minute ventilation ( $\dot{V}_E$ ), carbon dioxide production ( $\dot{V}CO_2$ ), and respiratory exchange ratio (RER) values were obtained every minute and during the final seconds of the test using a Beckman Metabolic Measurement Cart. The gas analyzers were calibrated at the beginning and end of each test using a standard gas previously analyzed with a micro-Scholander apparatus.

During the treadmill tests, an ECG was obtained utilizing a  $CM_5$  lead on a Hewlett-Packard electrocardiograph. Heart rates were determined just prior to exhaustion.

After completion of the treadmill tests, the subject cooled down by walking at 3 mph at a 0% grade for 2-minutes. At the end of the cool down period, the subject was immediately disconnected from the electrocardiograph, and the 5-minute post-exercise RV measurements were administered as previously described. Two FVC maneuvers followed

the RV measurements. In study II, the subjects repeated the RV and FVC measurements at 15 and 30 minutes, and at 1, 2, and 24 hours post-exercise. In study III, the subjects only repeated the RV and FVC measurements at 15, 30, and 60 minutes post-exercise.

#### Transthoracic Electrical Impedance

During studies III and IV, TEI was measured using four aluminized mylar tape electrodes (Surcon, Inc. Minnesota) and a Minnesota Impedance Cardiograph (model 304 A), according to the procedures outlined by Kubicek et al. (15). A constant sinusoidal current (4 mA rms) at 100 KHz was applied to the two outer electrodes, one on the forehead and the other around the abdomen. Changes in voltage, which reflect changes in impedance, were measured from the two inner electrodes, one around the neck and one around the lower thorax (15). TEI data were recorded when the subject had reached maximal expiration during the RV determinations.

#### Central Blood Volume Alterations

In an effort to determine the relationship between alterations in CBV and RV following exercise, 10 of the original 18 subjects from study III were recruited to perform two additional treadmill runs. RV, FVC, TLC, and TEI were measured at the same time intervals, using the same procedures as previously reported for study III. However, before and following one of the additional treadmill runs, CBV was experimentally increased via G-suit inflation. The G-suit was a government issued jumpsuit garment which when inflated, selectively

increased the pressure in the legs and lower abdomen. Using a similar suit, Weissler et al. (32) found a mean increase of 27% in CBV following inflation to 70 torr. In the present study, the suit was inflated to 80 torr 1-minute before the pre- and post-exercise RV and FVC determinations. TEI was recorded at maximum expiration during the RV determinations. CBV was also experimentally decreased before and following the other additional treadmill run using venous occlusion tourniquets. Peripheral blood pooling was produced by placing pneumatic blood pressure cuffs high on three extremities (both legs and left arm) and inflating to diastolic pressure levels, as previously outlined by Weissler et al. (32). Such a procedure has been previously shown to cause a mean decrease of 7% in CBV (32). In the present study, the cuffs were applied and inflated two minutes before the pre- and post-exercise RV and FVC maneuvers. TEI was recorded as previously described.

#### Diffusion Capacity of the Lung

Five of the original 18 subjects from study III were recruited to perform an additional treadmill run in which  $D_{LCO}$  was measured before the run and at 5, 15, 30, and 60-minutes post-exercise. The Krogh (13) single breath diffusion capacity procedure was used. A Collins Modular Lung Analyzer (Model 11B) equipped with helium and carbon monoxide analyzers was used. The subjects inspired a mixture containing 15% helium, 0.3% carbon monoxide, 28% oxygen, and the balance nitrogen. Alveolar gas samples were collected following a

10-second breath holding period. Alveolar volumes ( $V_A$ ) were determined from helium dilution.

#### Statistical Analysis

A standard statistical package (SPSS) was employed to analyze the data using repeated t-tests and repeated measures analysis of variance (ANOVA) where applicable. When significance was determined by ANOVA, Tukey's post-hoc comparisons were used to assess the differences between means.

## CHAPTER 3

### RESULTS

#### Study I

The subjects physical and performance characteristics are presented in Table 1. The pre- and post-exercise pulmonary function measurements appear in Table 2. There was a significant mean increase of 210 ml (20.6%) in the immediate post-exercise RV measure. FVC was not significantly changed following the exercise bout, therefore, there was a significant mean increase in the post-exercise TLC of 170 ml (3.4%). Both the forced expiratory volume exhaled in 1 second ( $FEV_1$ ) and the forced mid-expiratory flow ( $FEF_{25-75\%}$ ) were unchanged following exercise.

#### Study II

The physical and performance data of the subjects are presented in Table 3. All of the pre- and post-exercise pulmonary function data appear in Table 4. There was a significant mean increase of 260 ml, or 20.8% in the 5-minute post-exercise RV measurement. RV remained significantly increased over the pre-exercise value through 30 minutes of recovery. FVC was not significantly changed following exercise, therefore, there was a significant mean increase in the 5-minute post-exercise TLC of 190 ml, or 2.7%. TLC remained significantly increased over the pre-exercise value through 15-minutes of recovery. Both the

TABLE 1

Selected Physical and Performance Characteristics of Subjects  
in Study I (N = 11 females).

Characteristic	$\bar{X} \pm$ S.D.
Age (yr)	19.7 $\pm$ 1.7
Weight (kg)	50.61 $\pm$ 5.56
Height (cm)	163.4 $\pm$ 5.5
$\dot{V}O_2$ max (ml/kg·min)	55.9 $\pm$ 5.0
HR max (bpm)	192 $\pm$ 8.6
Treadmill time to exhaustion (sec)	748 $\pm$ 66

TABLE 2

Pre- and Post-Exercise Pulmonary Function Data for Study I  
(Values are means  $\pm$  S.D.)

	FVC (L)	FEV <sub>1</sub> (L) <sup>1</sup>	FEV <sub>1</sub> /FVC (%) <sup>1</sup>	FEF <sub>25-75%</sub> (L/min)	RV (L)	TLC (L)	RV/TLC (%)
Pre	3.89	3.37	86.8	251.6	1.04	4.94	20.9
	$\pm$ .42	$\pm$ .44	$\pm$ 8.6	$\pm$ 74.2	$\pm$ .29	$\pm$ .61	$\pm$ 4.0
Post	3.87	3.37	87.0	257.1	1.25	5.11	24.6
	$\pm$ .51	$\pm$ .64	$\pm$ 11.4	$\pm$ 92.5	$\pm$ .32	$\pm$ .67	$\pm$ 4.3
Diff.	-.02	.00	.2	5.4	.21*	.17*	3.8*

\*p < 0.05

TABLE 3

Selected Physical and Performance Characteristics of Subjects  
in Study II (N = 12 males).

Characteristic	$\bar{X} \pm$ S.D.
Age (yr)	26.1 $\pm$ 3.8
Weight (kg)	72.04 $\pm$ 7.97
Height (cm)	177.5 $\pm$ 7.3
$\dot{V}O_2$ max (ml/kg·min)	52.2 $\pm$ 10.5
HR max (bpm)	185 $\pm$ 10
Treadmill time to exhaustion (sec)	640 $\pm$ 105

TABLE 4

Pre- and Post-Exercise Pulmonary Function Data in Study II (Values are means  $\pm$  S.D.)

	FVC (L)	FEV <sub>1</sub> (L) <sup>1</sup>	FEV <sub>1</sub> /FVC (%) <sup>1</sup>	FEF <sub>25-75%</sub> (L/sec)	RV (L)	TLC (L)	RV/TLC (%)
Pre-	5.84 $\pm$ .78	4.79 $\pm$ .61	82.2 $\pm$ 6.2	4.97 $\pm$ 1.24	1.25 $\pm$ .19	7.09 $\pm$ .91	17.7 $\pm$ 1.8
5 min post-	5.78 $\pm$ .78	4.80 $\pm$ .68	83.2 $\pm$ 7.7	5.26 $\pm$ 1.55	1.51 $\pm$ .27*	7.28 $\pm$ .91*	20.7 $\pm$ 2.7*
15 min post-	5.78 $\pm$ .77	4.83 $\pm$ .63	83.9 $\pm$ 6.4	5.35 $\pm$ 1.40	1.46 $\pm$ .23*	7.25 $\pm$ .95*	20.2 $\pm$ 1.9*
30 min post-	5.80 $\pm$ .79	4.77 $\pm$ .68	82.4 $\pm$ 7.0	5.17 $\pm$ 1.38	1.40 $\pm$ .25*	7.20 $\pm$ .97	19.5 $\pm$ 2.1*
1 hr post-	5.80 $\pm$ .82	4.69 $\pm$ .60	81.2 $\pm$ 7.0	5.12 $\pm$ 1.49	1.36 $\pm$ .22	7.13 $\pm$ .97	18.8 $\pm$ 2.2
2 hr post-	5.80 $\pm$ .79	4.72 $\pm$ .60	81.6 $\pm$ 5.7	5.01 $\pm$ 1.23	1.29 $\pm$ .21	7.08 $\pm$ .93	18.3 $\pm$ 2.1
24 hr post-	5.77 $\pm$ .80	4.72 $\pm$ .60	81.7 $\pm$ 6.1	5.05 $\pm$ 1.16	1.25 $\pm$ .17	7.02 $\pm$ .91	17.9 $\pm$ 1.9

\*Significantly different ( $p < 0.01$ ) from pre-exercise value.

FEV<sub>1</sub> and FEF<sub>25-75%</sub> were unchanged following exercise. The RV/TLC ratio was significantly increased over the pre-exercise value through 30-minutes of recovery. This resulted from the fact that RV increased proportionally more than TLC.

### Study III

Table 5 shows the physical and performance characteristics of the subjects. All of the pre- and post-exercise lung volumes and TEI measures appear in Table 6. There was a significant mean increase of 200 ml (16.1%) in the 5-minute post-exercise RV. RV remained significantly increased over the pre-exercise value through 30-minutes of recovery. FVC was not changed following exercise, therefore, the mean 5-minute post-exercise TLC was significantly increased by 130 ml (1.9%). TEI was significantly decreased below the pre-exercise value through 30-minutes of recovery, indicating that there was a significant increase in thoracic fluid volume following exercise. Since TEI was measured at RV, and this lung volume was changing with time, TEI values were also normalized by expressing them as ohms per liter of RV. Such a procedure has previously been used by Jaeger et al. (11). This value was also significantly decreased below the pre-exercise value through 30-minutes of recovery.

### Study IV

A sub group of 10 subjects from study III were recruited to participate in this study. They performed two additional treadmill tests to exhaustion. RV, FVC, TLC, and TEI were measured pre- and

TABLE 5

Selected Physical and Performance Characteristics of Subjects  
in Study III (N = 18 males).

Characteristic	$\bar{X} \pm$ S.D.
Age (yr)	25.4 $\pm$ 3.5
Weight (kg)	79.21 $\pm$ 16.69
Height (cm)	177.8 $\pm$ 11.0
$\dot{V}O_2$ max (ml/kg·min)	54.1 $\pm$ 7.5
Treadmill time to exhaustion (sec)	622.7 $\pm$ 73.9

TABLE 6

Pre- and Post-Exercise Measurements in Study III  
(Values are means  $\pm$  S.D.)

	Pre	5-min post	15-min post	30-min post	60-min post
RV (L)	1.24 $\pm$ .19	1.44 $\pm$ .26*	1.38 $\pm$ .22*	1.36 $\pm$ .20*	1.29 $\pm$ .21
FVC (L)	5.47 $\pm$ .73	5.41 $\pm$ .73	5.42 $\pm$ .73	5.44 $\pm$ .78	5.45 $\pm$ .71
TLC (L)	6.71 $\pm$ .79	6.84 $\pm$ .81*	6.80 $\pm$ .78*	6.79 $\pm$ .80	6.73 $\pm$ .76
TEI (ohms)	24.7 $\pm$ 2.5	24.2 $\pm$ 2.5*	24.4 $\pm$ 2.4*	24.5 $\pm$ 2.4*	24.7 $\pm$ 2.5
TEI/RV**(ohms)	20.2 $\pm$ 2.8	17.3 $\pm$ 2.8*	17.9 $\pm$ 2.5*	18.3 $\pm$ 2.6*	19.4 $\pm$ 2.4

\*Significantly different ( $p < 0.05$ ) from pre-exercise value.

\*\*TEI expressed relative to residual lung volume.

post-exercise, as CBV was altered via G-suit inflation and venous occlusion tourniquets. Table 7 shows that there were no significant differences between the  $\dot{V}O_2$  max or time to exhaustion for the three treadmill tests (i.e. control, G-suit, venous occlusion). Table 8 shows all of the pre- and post-exercise lung volumes and TEI data for all 3 conditions. There was a significant mean increase of 180 ml (14.5%), 120 ml (9.6%), and 230 ml (17.0%) in the control, G-suit, and venous occlusion 5-minute post-exercise RV, respectively. RV remained significantly increased over the pre-exercise value through 30-minutes of recovery, during the control condition. For the other two conditions, it remained significantly elevated only through 15-minutes of recovery. FVC was not significantly changed following exercise for any of the 3 conditions. The 5-minute post-exercise TLC was significantly increased by 130 ml (1.9%), 160 ml (2.6%), and 160 ml (2.4%) over the pre-exercise value for the control, G-suit, and venous occlusion conditions, respectively. In all 3 conditions TLC was significantly elevated over the pre-exercise value during the 5-minute post-exercise period. TEI and TEI/RV were significantly increased over the pre-exercise value through 30-minutes, 15-minutes, and 15-minutes for the control, G-suit, and venous occlusion conditions, respectively. Table 8 also shows that G-suit inflation and venous occlusion resulted in significant changes in TEI at all measured time intervals, as compared to the control values. However, there were no significant differences in any measured lung volume, across the 3 conditions.

TABLE 7

$\dot{V}O_2$  max and Treadmill Time to Exhaustion in Study IV for the  
Control, G-Suit, and Venous Occlusion Conditions  
(Values are means  $\pm$  S.D.)

	$\dot{V}O_2$ max (ml/kg·min)	Treadmill time to exhaustion (sec)
Control	53.4 $\pm$ 6.0	598.5 $\pm$ 72.1
G-Suit	54.1 $\pm$ 6.5	616.9 $\pm$ 69.3
Venous Occlusion	53.9 $\pm$ 5.3	615.0 $\pm$ 57.7
F-Value	1.02	1.79

TABLE 8

Pre- and Post-Exercise Measurements in Study IV  
for the Three Experimental Conditions  
(Values are means  $\pm$  S.D.)

	Pre	5-min post	15-min post	30-min post	60-min post
<u>Control:</u>					
RV (L)	1.24 $\pm$ .17	1.42 $\pm$ .19*	1.37 $\pm$ .13*	1.36 $\pm$ .17*	1.29 $\pm$ .17
FVC (L)	5.47 $\pm$ .74	5.41 $\pm$ .81	5.41 $\pm$ .80	5.41 $\pm$ .85	5.44 $\pm$ .79
TLC (L)	6.70 $\pm$ .79	6.83 $\pm$ .83*	6.78 $\pm$ .78	6.76 $\pm$ .82	6.72 $\pm$ .80
TEI (ohms)	24.6 $\pm$ 2.2	24.2 $\pm$ 2.0*	24.3 $\pm$ 2.0*	24.3 $\pm$ 2.0*	24.6 $\pm$ 2.0
TEI/RV (ohms/L)	20.3 $\pm$ 3.3	17.3 $\pm$ 2.5*	17.9 $\pm$ 2.1*	18.1 $\pm$ 2.7*	19.3 $\pm$ 2.5
<u>G-Suit:</u>					
RV (L)	1.25 $\pm$ .27	1.37 $\pm$ .28*	1.32 $\pm$ .36*	1.29 $\pm$ .24	1.28 $\pm$ .25
FVC (L)	4.86 $\pm$ .74	4.92 $\pm$ .86	4.83 $\pm$ .83	4.88 $\pm$ .83	4.80 $\pm$ .91
TLC (L)	6.11 $\pm$ .84	6.27 $\pm$ .94*	6.15 $\pm$ .91	6.17 $\pm$ .91	6.07 $\pm$ .98
TEI (ohms)	22.7 $\pm$ 2.3 <sup>+</sup>	22.3 $\pm$ 2.1 <sup>++</sup>	22.3 $\pm$ 2.0 <sup>++</sup>	22.4 $\pm$ 2.0 <sup>+</sup>	22.5 $\pm$ 2.2 <sup>+</sup>
TEI/RV (ohms/L)	18.2 $\pm$ 2.9	16.3 $\pm$ 2.3*	17.0 $\pm$ 2.8*	17.4 $\pm$ 2.7	17.6 $\pm$ 2.5
<u>Venous Occlusion:</u>					
RV (L)	1.35 $\pm$ .29	1.58 $\pm$ .35*	1.49 $\pm$ .33*	1.43 $\pm$ .32	1.38 $\pm$ .28
FVC (L)	5.38 $\pm$ .96	5.31 $\pm$ 1.08	5.33 $\pm$ 1.01	5.34 $\pm$ 1.08	5.40 $\pm$ 1.05
TLC (L)	6.73 $\pm$ .98	6.89 $\pm$ 1.01*	6.82 $\pm$ .96	6.76 $\pm$ .96	6.78 $\pm$ 1.02
TEI (ohms)	25.6 $\pm$ 2.5 <sup>+</sup>	25.4 $\pm$ 2.4 <sup>++</sup>	25.6 $\pm$ 2.5 <sup>++</sup>	25.7 $\pm$ 2.5 <sup>+</sup>	25.7 $\pm$ 2.6 <sup>+</sup>
TEI/RV (ohms/L)	19.0 $\pm$ 3.1	16.1 $\pm$ 3.0*	17.2 $\pm$ 2.8*	18.0 $\pm$ 2.6	18.6 $\pm$ 2.1

\*Significantly different ( $p < 0.05$ ) from pre-exercise value.

<sup>+</sup>Significantly different ( $p < 0.05$ ) from corresponding control value.

### Study V

Five of the subjects from study III were recruited to participate in this study. They performed an additional treadmill test, following which  $D_{LCO}$  was measured.  $\dot{V}O_2$  max was not measured during this additional treadmill test, since it was conducted at the University of Arizona Health Sciences Center Pulmonary Function Laboratory, and no metabolic measuring equipment was available. However, there was no significant difference between the subjects' treadmill time to exhaustion for the control (study III) and  $D_{LCO}$  tests ( $605 \pm 70$  vs  $622 \pm 81$  seconds). Table 9 shows the pre- and post-exercise  $D_{LCO}$  data. There were no significant differences between the pre- and post-exercise  $D_{LCO}$  measurements.

TABLE 9

Pre- and Post-Exercise Diffusion Capacity in Study V  
(Values are means  $\pm$  S.D.)

	$D_{LCO}/V_A$ (ml/min·mmHg)
Pre	14.3 $\pm$ 1.7
5-min post	15.3 $\pm$ 1.9
15-min post	14.7 $\pm$ 1.9
30-min post	13.8 $\pm$ 1.7
60-min post	12.7 $\pm$ 1.6
F-Value	1.21

## CHAPTER 4

### DISCUSSION

Studies I, II and III all showed significant increases in the 5-minute post-exercise RV measurement. This agrees with the results of others (5,20,28). Also, all three of the present studies found a significant increase, of similar absolute magnitude to that of the increase in RV, in the 5-minute post-exercise TLC. These results agree with the findings of Hanson et al. (10) and Stubbing et al. (30), both of whom have previously reported similar increases in RV and TLC during exercise. The results from studies I, II, and III suggest that the increases previously reported (10,30) in RV and TLC during exercise are carried over into the post-exercise period.

An increase in the post-exercise TLC, as was shown in studies I, II, and III, has not been previously reported in the literature. However, this may simply be due to an oversight by previous researchers. Several studies (16,17) have reported no change in FVC following exercise, which agrees with the results of studies I, II, and III. However, neither of these previous studies (16,17) measured RV following exercise. Therefore, they could not report of TLC changes.

Maron et al. (20) found an increase in RV immediately following a marathon. The RV had returned to the pre-exercise value when the subjects were retested 24-hours following the race. Since these were the only two post-exercise RV measurements, it was impossible to

determine the recovery time of RV. Studies II and III determined the recovery time course for the RV and TLC as they returned to the pre-exercise value following exercise. Both studies show that RV and TLC remained significantly increased, over the pre-exercise values, through 30 and 15 minutes of recovery, respectively (Tables 4 and 6).

Although studies I and II determined and followed the effect of an acute bout of exercise on RV and other selected pulmonary function measurements, they did not help to elucidate the physiological mechanisms responsible for the increases in RV and TLC. Therefore, studies III, IV, and V were undertaken in an attempt to determine the mechanisms underlying the responses reported in studies I and II. Several groups have postulated that the changes seen in the post-exercise lung volumes may be caused by fluid shifts within the thorax (20,21). Therefore, study III determined the relationship between TEI and various lung volumes following exercise. As can be seen in Table 6, TEI and TEI/RV were both significantly decreased through 30-minutes of recovery. Since there is an inverse relationship between thoracic fluid volume and TEI (15), these data suggest that there is a significant increase in thoracic fluid volume following exercise. Such an increase in thoracic fluid volume following exercise would be consistent with the results of Marshall et al. (23) and Vaughan et al. (31), both of whom have reported increases in PEWV in man during exercise. Increases in capillary hydrostatic pressure and total capillary surface area, both of which increase during exercise (2,5), would promote interstitial edema formation during exercise.

Several studies (8,18) have shown that small increases in interstitial edema can result in large increases in RV. This is thought to be due to the edema causing a reduction in small airway diameter (9), thus allowing the airways to close sooner trapping more air. As the lymphatic system reabsorbs the accumulated fluid (33), it would seem reasonable to assume that the RV would return to control levels, which is consistent with the results from studies II and III.

Interestingly, interstitial edema of this magnitude does not seem to compromise FVC. Lemen et al. (18) found a 44% increase in RV, attributed to interstitial edema, yet an unchanged FVC, thus resulting in an increase in TLC. In a more recent study, Jaeger et al. (11) found a significant decrease in TEI in nonacclimated men abruptly exposed to high altitude. They also reported significant increases in RV and TLC which they attributed to the increase in thoracic fluid. Both of these studies (11,18) are consistent with the increase in RV and TLC reported in studies I, II, and III. The increases in TLC could also be due to increased patency, and possibly an increased number of conducting airways, as speculated by Kagawa and Kerr (12).

The unchanged  $FEV_1$  and  $FEF_{25-75\%}$  following exercise, as reported in studies I and II, agrees with the results of others (16,20). However, one may argue that if interstitial edema was causing a reduction in the diameter of the small airways, this should cause an increase in airway resistance, thus resulting in a decrease in  $FEV_1$  and  $FEF_{25-75\%}$ . However, studies I and II both show unchanged flow rates following exercise. This apparent paradox is resolved by the

fact that the peripheral airways contribute only approximately 10% of the total airway resistance, so that a decrease in small airway caliber would have little effect on the total resistance (10). Another fact complicates the interpretation of the post-exercise flow rate data from studies I and II. Since TLC was changing during the various time intervals, all of the FVC maneuvers were performed from unequal maximal lung volumes. Lefcoe (16) suggests that an increase in TLC would increase  $FEV_1$  and  $FEF_{25-75\%}$ . Unfortunately, the extent to which a small increase in TLC, such as that reported in studies I and II, will increase  $FEV_1$  and  $FEF_{25-75\%}$  is unknown at this time.

Although study III shows that there is an increase in thoracic fluid volume following exercise, TEI measurements alone cannot separate between intra- and extravascular fluid shifts. Therefore, one could argue that the decrease in TEI following exercise could simply be due to an increase in intravascular volume (i.e. blood), and not interstitial edema. Studies IV and V took two different approaches in an attempt to identify whether the decrease in TEI and increase in RV as reported in study III were due to intra- or extravascular fluid shifts.

Study IV was based on the assumption that if the increase in RV following exercise was due to an increase in thoracic intravascular fluid, then experimental increases and decreases in thoracic blood volume should bring about parallel changes in RV. Study IV examined the TEI, RV, and TLC responses before and following an acute bout of exercise, as intrathoracic blood volume was experimentally altered via G-suit inflation and venous occlusion tourniquets. The bouts of

exercise were similar for the three conditions as there were no significant differences between the treadmill times of  $\dot{V}O_2$  max values for the three tests (Table 7). Intrathoracic blood volume was experimentally increased and decreased via G-suit inflation and venous occlusion as evidenced by the 1.9 volt decrease and .9 volt increase in the pre-exercise TEI, respectively. Following their respective treadmill tests, G-suit inflation and venous occlusion resulted in increased and decreased TEI values respectively, at all measured time intervals as compared to the control condition. However, as can be seen in Table 8, the post-exercise RV response was not altered from the control condition by either treatment. This suggests that RV is relatively insensitive to intravascular volume shifts within the thorax.

Study V determined and followed the effect of an acute bout of exercise on  $D_{LCO}$ .  $D_{LCO}$  can be considered an indirect measure of thoracic intravascular volume, since several studies (2,24) have shown that a linear relationship exists between increases in  $D_{LCO}$  and pulmonary blood volume. Therefore, it was felt that if TEI decreased and  $D_{LCO}$  increased, this would suggest that the increased intrathoracic fluid would have to be partially or totally explained by shifts in intravascular fluid. However, if TEI decreased and  $D_{LCO}$  remained the same, then one would assume that the increased intrathoracic fluid was not blood, and must be extravascular fluid, i.e. interstitial edema. As can be seen in Table 9,  $D_{LCO}/V_A$  did not increase significantly following exercise. This agrees with the results of Brashear et al. (2) who reported that  $D_{LCO}$  was similar to the pre-exercise values at

10 minutes following completion of exercise. In a more recent study, Maron et al. (20) reported no significant change in  $D_{LCO}$ , as compared to the pre-exercise value, following a marathon. These studies are consistent with the results from study V. Therefore, these data suggest that the decrease in TEI following exercise is a result of extravascular fluid accumulation.

From the results of studies I through IV and the results from previous research, the following sequence of events is proposed as a possible explanation for the responses of RV to an acute bout of exercise. During exercise capillary hydrostatic pressure is elevated (4), and the total capillary surface area for fluid exchange is increased (2), both of which promote extravascular fluid accumulation. This was confirmed by the decrease in TEI following exercise. The fluid preferentially accumulates in the peribronchial interstitial space (29), thus causing a decrease in small airway diameter (9). Therefore, during a forced expiration the peripheral airways close sooner, thus trapping more air, as evidenced by the increase in RV. The interstitial edema, however, does not seem to compromise FVC (18). Following exercise, as the lymphatic system reabsorbs the accumulated fluid (33), the RV would return to control levels.

## CHAPTER 5

### SUMMARY

A series of five different studies were conducted to examine several aspects of the effect of exercise on selected pulmonary function measurements. Studies I and II determined and followed the effect of an acute bout of exercise on various lung volumes immediately post-exercise and over a 24-hour post-exercise period. There were significant mean increases of 210 ml (20.6%) and 260 ml (20.8%) in the 5-minute post-exercise RV measurement for studies I and II, respectively. There also were significant mean increases of 170 ml (3.4%) and 190 ml (2.7%) in the 5-minute post-exercise TLC for studies I and II, respectively. RV and TLC remained significantly increased over the pre-exercise values through 30 and 15 minutes of recovery, respectively. Since these first two studies only documented the various post-exercise pulmonary function responses, studies III through V were undertaken in an attempt to determine the physiological mechanisms underlying the responses reported in studies I and II.

Study III reported that TEI was significantly decreased below the pre-exercise value through 30-minutes of recovery, indicating that there was a significant increase in thoracic fluid volume following exercise. However, TEI measurements alone cannot separate between intra- and extravascular fluid shifts. Therefore, studies IV and V took two different approaches in an attempt to identify whether the

decrease in TEI and increase in RV reported in study III were due to intra- or extravascular fluid shifts. Study IV examined the TEI, RV, and TLC responses before and following an acute bout of exercise, as CBV was experimentally altered via G-suit inflation (increasing CBV), and venous occlusion tourniquets (decreasing CBV). The results suggest that RV is relatively insensitive to intravascular volume shifts within the thorax. Study V determined and followed the effect of an acute bout of exercise on  $D_{LCO}$ .  $D_{LCO}/V_A$  did not increase significantly following exercise, suggesting that the decrease in TEI following exercise is the result of extravascular fluid accumulation.

It was concluded that a sub-clinical pulmonary edema occurs following exercise. A logical sequence of events, based on the results of studies I through V, and the results of previous research, was proposed as a possible explanation for the responses of RV and TLC to exercise.

#### SUMMARY OF TERM ABBREVIATIONS

1. Residual volume	RV
2. Total lung capacity	TLC
3. Transthoracic electrical impedance	TEI
4. Central blood volume	CBV
5. Lung diffusion capacity	$D_{LCO}$
6. Pulmonary extravascular water volume	PEWV
7. Volume of trapped gas	VTG
8. Forced vital capacity	FVC
9. Alveolar volume	$V_A$
10. Forced expiratory volume exhaled in 1 second	FEV <sub>1</sub>
11. Forced mid-expiratory flow	FEF <sub>25-75%</sub>

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