University of Alberta

The Effects of a 2000-m Rowing Race and 10-weeks of Combined Strength and Endurance Training on Left Ventricular Systolic Function.

by

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A thesis submitted to the Faculty of Graduate Studies and Research

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ABSTRACT

The purposes of this investigation were to determine changes in left ventricular (LV) function immediately following and during recovery from a 2000-m rowing trial, prior to and following 10- weeks of training, and to determine the effect of 10-weeks of training on resting LV morphology in male rowers. Immediately following the 2000-m trial there was a significant improvement in contractility, which returned to resting levels during recovery. Following training, relative and absolute LV mass increased significantly. There was also a significant increase in wall thickness and cavity area, suggesting eccentric LV hypertrophy. Following training there was a decrease in contractility and an increase in end-diastolic cavity area suggesting an increased reliance on the Frank-Starling mechanism. Thus, responses during recovery from a 2000-m trial suggest an increase in contractile function, while 10-weeks of combined training is a sufficient stimulus for increasing LV morphology and an increased reliance on Frank-Starling vs. contractility.

To my parents.

Whose sacrifice and support made it possible
for me to pursue all that I wanted.
And whose guidance and example shaped me into the person I am

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LIST OF SYMBOLS, NOMECLATURE, AND ABBREVIATIONS

BSA	Body Surface Area
DBP	Diastolic Blood Pressure
EDCA	End-Diastolic Cavity Area
EDMA	End-Diastolic Myocardial Area
EDTA	End-Diastolic Total Area
ESCA	End-Systolic Cavity Area
ESMA	End-Systolic Myocardial Area
ESTA	End-Systolic Total Area
FAC	Fractional Area Change
HR	Heart Rate
LVM	Left Ventricular Mass
LVMi	Left Ventricular Mass Index
SA	Stroke Area
SBP	Systolic Blood Pressure
SBP/ESCA	Systolic Blood Pressure to End-Systolic Cavity Area Relation
$\mathbf{VO_{2max}}$	Maximal Oxygen Consumption
VT	Ventilatory Threshold
σ	End-Systolic Wall Stress

CHAPTER 1

INTRODUCTION

1.1 Introduction

Rowing is an activity that places high demand on both the aerobic and anaerobic energy systems. Rowing training and competition includes the performance of a 2000-m trial that involves completing a 2000-m course a quickly as possible (Gilles and Bell, 2000). This type of trial places a great stress on the cardiovascular system, elicits a maximal oxygen consumption (VO_{2max}) intensity, as well as a high rate of anaerobic glycolysis (Hagerman et al. 1978, Pripstein et al. 1999). As rowing ergometers have become increasingly popular over the last decade, rowers have been using these devices to supplement their on-water training and a separate competition arena has emerged with the rowing ergometer as the primary mode of competition (Kramer et al. 1994, Gilles and Bell, 2000).

Fatigue in rowing can be partially attributed to the repeated muscular contractions required to perform the basic rowing stroke. Pripstein et al. (1999) have shown that during the first two minutes of a 2000-m race subjects exhausted 78% of their maximal anaerobic energy supplies and had completely taxed their anaerobic energy stores by the end of the 2000-m race. Kennedy and Bell (2000) have also shown a high correlation between 2000-m time and VO_{2max}. This suggests that large amounts of metabolic by-products may be produced during a 2000-m race, which would contribute to skeletal muscle fatigue through numerous means (Fitts, 1994).

The aerobic energy system may be responsible for up to 87% of the energy necessary to complete the race (Hagerman et al., 1978; Pripstein et al., 1999). As there is such a high demand on the aerobic system for the production of the energy needed to perform the 2000-m race, it is feasible that any impairment in cardiac function may impair performance. As there are numerous similarities between skeletal and cardiac muscle structure and the functional mechanisms under which they contract (O'Brien et al., 1991), it seems possible that the metabolic waste products produced during the 2000-m race may affect cardiac contraction in some manner, such as interference with excitation-contraction coupling processes.

1.2 Purpose and Hypothesis

The purpose of this investigation was to evaluate the effects of a single bout of high-intensity exercise on left ventricular (LV) systolic function immediately following and during recovery from a 2000-m, as well as to determine the effects of a 10-week combined strength and endurance-training program on LV structure and morphology. Finally, the effect of 10-weeks of combined strength and endurance training on LV systolic function immediately following and during recovery from a 2000-m rowing race was investigated. Multiple aspects of LV function including LV end-diastolic cavity area (EDCA), LV end-systolic cavity area (ESCA), LV end-systolic total area enclosed by the epicardium (ESTA), end-systolic myocardial area (ESMA), LV fractional area change (FAC), LV contractility (systolic blood pressure to ESCA relation), and LV end-systolic meridional wall stress (σ) were evaluated in ten male club level rowers using 2-D echocardiography at rest, immediately following and during recovery from a simulated 2000-m rowing race on a Concept IIC rowing ergometer. It was hypothesized that the

acute bout of high-intensity exercise would result in depression of LV systolic function (contractility), while the 10-week training program would result in an increase in resting LV morphology and improve post-exercise LV systolic function following the second acute exercise bout.

1.3 Significance of Study

It has been shown that high-intensity exercise can have detrimental effects on skeletal muscle contraction, which is due in part to an increase in hydrogen ions (H^+) (Fitts, 1994). As cardiac and skeletal muscle share many similarities in their contractile mechanisms, an increase in H^+ (decrease in blood pH) may have a similar effect on heart function, resulting in a decrease in heart function and a decrease in performance (O'Brien, 1991). Previous research (Pelliccia et al., 1991) has shown that rowers possess some of the largest hearts. However, there exists some debate as to the time course for this increase in LV mass and morphology.

1.4 Delimitations

The subjects involved in this investigation were 10 male club level rowers from the Edmonton area and had been rowing for 2 months and completed at least one simulated 2000-m rowing race prior to the beginning of the study. Subjects underwent a 2000-m simulated rowing race on a Concept IIC rowing ergometer. Echocardiographic data was measured using a Hewlett-Packard ultrasound instrument (Sonos 5500) with a 3.5-MHz transducer from the left apical area at the level of the mitral valve leaflets at rest, immediately following the 2000-m trial, 5-minutes after exercise, and 45-minutes after exercise. Changes in LV systolic function were examined using the following measures: EDCA, ESCA, ESTA, EDTA, ESMA, EDMA, FAC, SBP/ESCA, and σ.

Subjects then underwent a periodized 10-week combined strength and endurance-training program. Following the 10-week training program, subjects performed a second 2000-m rowing trial and a second set of echocardiographic measures was taken. All echocardiographic measures were made following the recommendations of the American Echocardiographic Society (Sahn, 1978). Blood lactate measurements accompanied each echocardiographic image to allow for an estimation of the anaerobic metabolism at each measurement time. Subjects also completed a maximal oxygen consumption test to determine aerobic fitness levels, as well as a predicted 8 repetition maximum test for the leg-press and bench-press to allow for the determination of strength levels.

1.5 Limitations

One of the major limitations of this study was the inability to obtain echocardiographic images during exercise due to motion of the subject and increased ventilation. Thus, echocardiographic images were obtained at a rested state and immediately following the 2000-m exercise trial.

A second limitation of the investigation was standardizing the time between the completion of the exercise bout and the post-exercise echocardiograph. Following such high-intensity exercise, athletes often have difficulty moving from the rowing ergometer to the echocardiograph bed. Subjects were encouraged to move as quickly as possible and were aided by the investigators during the transfer. Measurements of lactate immediately following the exercise bout were difficult to standardize with respect to time as well. Following the 5-minute post exercise echocardiograph a blood lactate sample was taken. This was difficult as subjects were still recovering from the 2000-m trial and had difficulty moving.

A third limitation was the movement of the subjects from a semi-supine position to an upright position and back to a supine position during the different stages of testing. During the 2000-m trial the subjects were in a semi-supine position demanded by the movement pattern. Following the 2000-m trial subjects were required to walk to the echocardiographic measurement bed and then lie in a supine position for image acquisition. These changes in position may have artificially loaded and unloaded the heart resulting in changes in measurements. However, each subject underwent the same pattern during the testing sessions, resulting in similar alterations to each subject.

A final limitation with respect to the 2000-m trial was the motivation of the subjects. All subjects had to complete the 2000-m trial on their scheduled day due to the time restrictions of the echo-technician. Therefore, if a subject performed poorly this data had to be used, as the 2000-m trial could not be repeated.

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CHAPTER 2

REVIEW OF LITERATURE

High-intensity exercise results in skeletal muscle fatigue through a number of possible mechanisms, including accumulation of H^+ ions (Fitts, 1994). As cardiac muscle and skeletal muscle share similarities in their contractile mechanisms, there exists the possibility that cardiac muscle may fatigue in a similar manner as skeletal muscle. The clearance of metabolic waste products from skeletal muscle into the circulatory system may have a negative affect on the contractile properties of the cardiac muscle by diffusion into the cardiac myocyte, resulting in a decrease in performance.

2.1 Rowing

The sport of rowing involves a combination of muscular strength and endurance, as well as high aerobic and anaerobic fitness (Secher, 1993). As the legs, trunk and upper extremities contribute to the rowing stroke approximately 70% of the total muscle mass is involved in the stroke and the majority of this muscle is composed of slow-twitch fibres (Steinacker, 1993). A rower may rely upon the aerobic energy system to supply the majority of the energy required to perform the 2000-m race (Pripstein et al., 1999). Thus, rowers tend to have absolute maximal oxygen consumptions that reach scores over 6.00 $l \cdot min^{-1}$ (Steinacker, 1993). Rowing also places a large strain on the anaerobic energy system, producing some of the highest recorded lactate measures (32 $mmol \cdot l^{-1}$) and lowest blood pH measures (6.85) recorded (Neilson, 1999). Rowing also stimulates large fluctuations in systolic blood pressure due to a brief period of valsalva

during the catch phase of the rowing stroke (Clifford et al., 1994). These fluctuations in systolic blood pressure, combined with the consistent volume and pressure overload experienced during training may result in the stimulus necessary for the increases in heart size that has been seen in rowers (Pelliccia et al., 1991). The physiological traits associated with rowing, especially the high lactate and low pH seen during the 2000-m rowing race may result in a depression of LV contractility by interfering with the contractile mechanisms during excitation-contraction coupling.

2.2 Fatigue

Skeletal muscle fatigue may occur through a number of possible mechanisms including; excitatory input to higher motor sensors, excitatory drive to lower motor neurons, motor neuron excitability, neuromuscular transmission, sarcolemma excitability, excitation-contraction coupling, contractile mechanism, and metabolic energy supply and metabolic accumulation (Bigland-Ritchie, 1984).

Two types of exercise, long-term low-intensity exercise and short-term high-intensity exercise characterize fatigue. Long-term low-intensity exercise stresses the type I muscle fibres, which results in a much different fatigue process than short-term high-intensity exercise, which stresses all fibre types, but most important, the type II fibres (Wenger and Reed, 1976). By stimulating the type II fibres, eliciting a high contraction frequency and a high level of anaerobic metabolism, high-intensity exercise may lead to an increase in lactic acid formation and thus increased intracellular H^+ ($[H^+]_i$) and lactate, as well as causing an increase in other metabolites such as inorganic phosphate. High-intensity exercise may also have large negative effects on the excitation-contraction

coupling process, such as inhibition of Ca^{2+} release from sarcoplasmic reticulum (SR) (Fitts, 1994).

Skeletal muscle contraction is accomplished via the activation of actin-myosin cross-bridge cycling. Action potentials from the neurons stimulate the release of calcium from the SR. This Ca^{2+} binds with the active site troponin-C, which in turn releases the attachment of actin to tropomyosin allowing the actin to interact with the myosin heads (Fitts, 1994). The increase in H^+ ions due to fatigue may reduce peak tension and contribute to fatigue by decreasing intracellular pH. The decrease in intracellular pH has been associated with an increase in the amount of Ca^{2+} required to initiate contraction (Fitts, 1994). Favero et al. (1995) investigated the interactions of several intracellular metabolites on Ca^{2+} release and found that a decrease in pH from 7.1 to 6.5 resulted in a significant decrease in Ca^{2+} release. This decrease in Ca^{2+} resulted in less binding of myoplasmic Ca^{2+} to troponin-C and a decrease in force production. It has also been suggested that H^+ ions directly inhibit force by reducing cross-bridge cycling, through competition with Ca^{2+} for active binding sites on troponin-C (Fitts, 1994).

2.3 Lactate Production

As a working cell begins to experience anaerobic metabolism there is an increase in the production of lactic acid. Lactate is produced during high-intensity exercise, although its direct cause is somewhat debated. Some view lactate production as a strain on mitochondrial respiration due to a decrease in O_2 supply, which increases the $NADH/NAD^+$ ratio shifting the lactate/pyruvate ratio towards lactate (Katz and Sahlin, 1988). While others suggest that lactate production is more dependent on glycolytic rate increasing the $NADH/NAD^+$ ratio and shifting the lactate/pyruvate ratio towards lactate,

as opposed to O_2 supply (Connett et al., 1990). In either situation there is an increase in the lactate produced, which can be associated with an increase in H^+ and therefore a decrease in pH. Richardson et al. (1998) have shown a linear fall in intracellular pH and a concomitant linear rise in net muscle lactate efflux, suggesting that there is a strong relationship between increasing lactate and an associated decrease in muscle pH.

To this point the reduction in force production in skeletal muscle has been attributed to intracellular mechanisms (increase in $[H^+]_i$ and it's interference with the contractile process). However, not all lactic acid produced in skeletal muscle remains in the myocyte. Mainwood and Worsley-Brown (1974) suggest this movement of lactic acid out of the myocyte may be dependant on a number of mechanisms. First, they suggest that the pH level and lactate concentration of the blood may be a determining factor in the speed and amount of lactic acid efflux from the cell. If there is a low pH or a high level of lactate in the blood, the efflux of lactic acid is reduced. They also suggest that lactic acid is effluxed from the myocyte both through simple diffusion and through an active process, which is dependent on the extracellular pH and lactate. As pH decreases and extracellular lactate concentration increases, the active mechanism is employed. Finally, they suggest that lactic acid effluxes from the myocyte both as the undissociated form of lactic acid, as well as in the dissociated form of lactate and hydrogen ions and that this is dependent on extracellular pH levels and lactate concentratotion. The dissociated form of lactate effluxes from the myocyte when pH increases, when pH is lower lactic acid efflux is favoured.

2.4 Cardiac Contraction

Cardiac muscle is similar to skeletal muscle as they both contain actin-myosin cross-bridges and require intracellular Ca^{2+} for excitation-contraction coupling to take place. However the initiation of excitation-contraction coupling in cardiac muscle relies on different mechanisms (Tibbits and Hamman, 1991) and the structures of the mechanisms required for contraction are different as well. In the cardiac muscle the t-tubules are larger, and form diads with the SR, while skeletal muscle has smaller t-tubules and form triads with the SR. Both of these arrangements allow for the sarcolemma to be close to the Ca^{2+} release mechanisms. Cardiac muscle differs from skeletal muscle at this point due to the requirement of Ca^{2+} to initiate any further contractile action. An action potential initiates the release of Ca^{2+}. The Ca^{2+} then enters the SR through a structure known as the ryanodine receptor, which in turn activates the release of Ca^{2+} from the SR (Brown and Kowalski 1997). This process is known as calcium-induced calcium release (Chapman, 1983). Thus, the cardiac muscle has a greater potential for the possible fatiguing effects of an increase in H^+, as there are a greater number of steps in the contraction process.

As cardiac muscle rarely becomes anaerobic, it is the efflux of lactate and H^+ from the skeletal muscle into the blood and then into the cardiac myocyte, which may lead to impairment of heart function. Orchard and Kentish (1990) suggest that a decrease in extracellular pH can result in impairment of cardiac contraction due to interference with the voltage-dependant Ca^{2+} channels as the increase in H^+ interferes with the negative charge of the channel. They also suggest that the H^+ may block a specific site in the Ca^{2+} channel. Fabiato and Fabiato (1978) placed skinned cardiac fibres from rat

ventricles and skinned skeletal muscle fibres from frog semitendinosus in solutions of various pHs. They found that decreasing pH resulted in decreases in both submaximal and maximal tension. They also found that decreasing pH from 7.40-7.00 resulted in a greater decrease in submaximal tension than a reduction from 6.60-6.20. The opposite was seen for maximal tension and the effects were greater in cardiac muscle versus skeletal muscle. They attributed these findings to the greater depressive effects of a low pH on the SR, a decrease in the amount of Ca^{2+} released from the SR, and the amount of Ca^{2+} released from the SR is less than that required to completely activate the actin and myosin filaments. Samaja et al. (1999) have shown that high levels of lactate and H^+ in isolated hearts resulted in a reduction in the force of contraction of the heart. They have also shown that a decrease in blood pH resulted in an increase in the myocyte intracellular H^+ concentration and suggest that the impairment in contractility is a result of impaired Ca^{2+} sequestration into the SR. They also claim that the effects of high H^+ levels may have reversible effects, while high lactate levels may have irreversible effects on contractility. Berger et al. (1999) found that lactic acid induced acidosis resulted in impairment of the relaxation of the ventricle in profused hearts and suggest that this is may be due to two mechanisms; 1) intracellular acidosis and 2) the pH-independent effects of the lactate ions themselves. These studies suggest that both pH associated changes and lactate itself can have a negative effect on both the contraction and the relaxation of the heart.

It has been shown that rowers have produced some of the highest reported blood lactate values following a 2000-m rowing race. Nielsen (1999) has shown that upon completion of a 2000-m rowing race elite level rowers have obtained blood lactates of up

to 32 mmol·l^{-1} and a reduction in blood pH to 6.85. He suggests that this low pH and high blood lactate reduce O_2 saturation and thus O_2 uptake due to the inability of cardiac output to compensate for the decrease in O_2 saturation. With such high levels of circulating blood lactate and large decrease in blood pH, there may be a possible negative effect on LV function following such a high intensity exercise bout.

2.5 Acute Exercise and Heart Function

Previous research has examined the effect of acute exercise on the structure and function of the heart. Goodman et al. (1991) have shown an improvement in LV function during cycle ergometer exercise increasing from a low level to a maximal effort. They suggest that the improvement in function at lower intensities was due to the Frank-Starling mechanism; while at higher intensities there was an improvement in contractility. However, maximal intensity in this investigation is based on a maximal oxygen consumption test, therefore subjects are not required to hold the high-intensity exercise for a prolonged period, which may not allow for sufficient levels of blood lactate and H^+ to accumulate. Pokan et al. (2000) have also shown improvements in systolic and diastolic function with cycle ergometer exercise and a return to normal function during recovery. Again these subjects were not subjected to a prolonged bout of high-intensity exercise. Improvements in LV function may also be related to diastolic function. Matsuda et al. (1983) suggest that there is an improvement in diastole during exercise and that this is related to the early filling portion of ventricular relaxation. They have also shown that this improved relaxation is a benefit during exercise, however they have chosen an exercise intensity of 50% of VO_{2max}. Such research supports the notion that a single-bout of exercise can improve LV function. This improvement in LV function is

necessary to supply the active muscles with the required O_2 and substrates necessary for continued contraction. The above authors also support the idea that following exercise, LV function returns to normal. However, the major drawback to these investigations is the lack of a prolonged state of maximal exercise. Whether or not LV function is compromised due to this type of high-intensity exercise is not known.

2.6 Cardiac Fatigue

Other research has focused its attention towards prolonged periods of activity. Such investigations have coined the term 'cardiac fatigue' to explain the decreased functioning of the heart after prolonged exercise bouts. In the late 1980's much of the attention to LV function was directed towards prolonged severe exercise. The Ironman™ triathlon was a common mode of testing. Douglas et al. (1987) examined the effect of the Ironman™ triathlon on LV systolic and diastolic function. Baseline measures of LV function were taken at rest, athletes then completed the competition and LV function was measured immediately following the event. LV function during recovery was measured at rest, one day post-event. They found that fractional shortening decreased after the event and returned to near normal values upon recovery. They suggest a mechanism of decreased fractional shortening (approximately 10%) that is attributable to a reduction in EDV and a decrease in contractility. There was also an altered filling pattering during diastole. Seals et al. (1988) examined the effects of long-term strenuous exercise similar to that of Douglas et al. (1987). They found similar decreases in systolic function. Whyte et al. (2000) examined the effect of different distances on the incidence of cardiac fatigue. They determined that both ultra-endurance events like the Ironman™ and shorter events, such as a half Ironman result in depressed systolic and diastolic function.

Haykowsky et al. (2001) found reductions in LV systolic function following a half-Ironman™ race (mean race time of 5hrs). They attributed this reduction in systolic function to a decrease in LV contractility, which resulted in a decrease in LV end-systolic myocardial area with a concomitant increase in LV end-systolic cavity area and wall stress. These reductions occurred despite a decrease in systolic blood pressure. Therefore it seems that decreases in LV systolic function may be attributed to decreases in LV contractility. Further evidence exists that even shorter duration bouts of exercise can stimulate cardiac fatigue. Vanoverschelde et al. (1991) found that exercise durations of 90 minutes reduced ventricular performance in healthy subjects. However, exercise intensities were similar to those of the longer events.

2.7 Athlete's Heart

Another area of interest related to the heart is that of chronic exercise and its effect on cardiac structure and function. Chronic exercise has been shown to improve the function of the heart (Fagard, 1997). The main mechanisms that contribute to this improvement are based on the loading condition of the heat. In dynamic (endurance) sports there is a constant volume overload that is experienced by the heart (Fagard, 1996). During this overload there is an increase in heart rate and stroke volume, the major components of cardiac output. The increased LV wall stress results in an increase in the internal diameter of the ventricle with an associated increase in the ventricular wall thickness, known as eccentric hypertrophy. Static sports, such as weightlifting place a different stress on the heart. This stress is a result of an increased pressure overload, which results in a thickening of the ventricular wall, with no change in the ventricular internal diameter. This change in heart shape is known as concentric hypertrophy

(Fagard, 1996). Both types of hypertrophy serve to balance the relationship between systolic pressure (pressure experienced during the contraction phase of the heart cycle) and the ratio of wall thickness to ventricular radius. Many athletic activities combine these two types of training and as a result their heart may experience a combination of the two types of overloading and thus a combined morphologic change (Fagard, 1997). Rowers in particular have training regimes that combine both types of overload. There is also a static and dynamic component to the rowing stroke itself. As a result of this static and dynamic overloading rowers have been found to have some of the largest hearts measured (Pelliccia et al., 1991; Cavallaro et al., 1993; Spirito et al., 1994; Gustafsson et al., 1996).

2.8 Conclusion

Rowing serves as an excellent medium for the combination of the above factors to investigate as to the effects of high-intensity exercise on LV function. The sport of rowing involves both endurance training and strength training. Athletes compete at both long duration events and shorter, more intense bouts. Specifically, the 2000-m event requires a combination of high strength and power, but also a high level of aerobic and anaerobic fitness. As stated above rowers have been shown to have some of the highest blood lactate levels and lowest pH levels of any athlete following the 2000-m race (Nielsen, 1999). Also, due to the concurrent strength and endurance training that rowers undergo, they have been shown to have some of the largest hearts ever measured (Pelliccia et al., 1991; Cavallaro et al., 1993; Spirito et al., 1994; Gustafsson et al., 1996). However, these investigations are limited to the training effect of rowing on heart function, but do not investigate the effect of an acute bout of exercise on heart function.

As well, investigations into decreases in cardiac performance are limited to longer exercise durations at a lower intensity. Thus this population should provide a strong model for the investigation of the effects of high intensity exercise on heart.

2.9 References

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CHAPTER 3

EFFECTS OF A 2000-M ROWING RACE AND 10-WEEKS OF COMBINED STRENGTH AND ENDURANCE TRAINING ON LEFT VENTRICULAR SYSTOLIC FUNCTION

3.1 Introduction

The 2000-m rowing race is one of the most demanding high intensity bouts of exercise. Pripstein (1999) suggests that the majority of energy required for the 2000-m rowing race is derived from the aerobic energy system. Kennedy and Bell (2000) support this notion suggesting that 2000-m rowing race time correlates well with VO_{2max}. Although the aerobic energy system may be the major supplier of energy for the 2000-m rowing race, anaerobic energy sources play a major role as well. Neilson (1999) has shown that following a 2000-m rowing trial; rowers may experience some of the highest blood lactate levels and lowest blood pH levels ever reported (32 $mmol \cdot L^{-1}$ and 6.85 respectively). Thus, it appears that all energy systems are employed during the 2000-m rowing race.

High lactate levels and low pH levels have been shown to decrease the contractile function of perfused hearts (Samaja et al., 1999 and Berger et al., 1999). Fatigue during a 2000-m rowing race has been partially attributed to the increase in anaerobic metabolites and a decrease in contractile function of the skeletal muscle. With such high levels of lactate and low levels of pH, and the similarities between the contractile properties of skeletal muscle and the heart, it seems possible that both the high levels of

blood lactate and low blood pH may have a negative effect on the contractile function of the heart during and possibly immediately following the 2000-m rowing race.

It has also been shown that rowers possess some of the largest left ventricular (LV) dimensions and LV masses (Pelliccia et al., 1991). These increases in heart dimension and size have been attributed to the high levels of strength and endurance training that is required for success in elite levels of rowing competition. This increased volume load due to increased heart rate and cardiac output results in an increase in diastolic wall stress. In order to compensate for the increased wall stress the ventricle replicates sarcomeres in series, elongates fibres and increases cavity size. The increase in cavity size results in an increase in systolic wall stress, which is compensated for by replication of sarcomeres in parallel and an increase in wall thickness (Grossman et al., 1975). Although it has been reported that rowers possess large hearts, the time course for development of this enlargement is still not clear. While Cavallaro et al. (1993) found increases in LV end-systolic, LV end-diastolic diameters and LV mass following 5 months of training, Haykowsky et al. (1999) only showed an increase in LV dimension, but not LV mass following 10-weeks of training. It is suggested that long-term commitments rowing training may provide the stimulus for increased LV size, but the time-course for increases in LV mass are unknown.

The purpose of this investigation was to evaluate LV systolic function immediately following and during recovery from a 2000-m rowing race. Secondly, the effects of a 10- week combined strength and endurance-training program on LV structure and function at rest were evaluated. Finally, the effects of the 10-week strength and endurance-training program on LV systolic function immediately following and during

recovery from a 2000-m rowing race were investigated. It was hypothesized that the high levels of lactate and low pH associated with a 2000-m rowing race would result in a decrease in LV systolic function following the 2000-m rowing race and that function would return to resting values during recovery. It was also hypothesized that an increase in LV morphology would be seen after 10-weeks of combined training. Finally, it was hypothesized that following the 10-week training program, LV systolic function would be improved following the 2000-m rowing race.

3.2 Subjects and Experimental Design

Ten male subjects from a local rowing club and from the student population of the University of Alberta volunteered to participate in this investigation. Inclusion criteria required that the subjects be healthy, active and been rowing and strength training at least twice a week in the month leading up to the study. Subjects were also required to sign a PAR-Q and informed consent. This investigation was reviewed and approved by the Faculty of Physical Education and Recreation Human Ethics Committee at the University of Alberta. The experimental design involved the investigation of the acute responses immediately following and during recovery from high-intensity exercise bout, before and after a 10-week combined strength and endurance-training program. Subject characteristics appear in table 3-1.

Physiological Testing

Testing included 2-D echocardiographic measures and lactate determination (Accusport, Mannheim, Germany), at rest and following a simulated 2000-m rowing race on a Concept IIC rowing ergometer (Morrisville, Vermont). Post exercise echocardiograms and lactate determinations were completed immediately following, 5-

and 45-minutes after the 2000-m simulated rowing race. Following a minimum of 24-hr of rest the subjects returned to the laboratory for the completion of a maximal oxygen consumption test. Subjects returned again on another day to perform multiple repetition maximum testing for the 45° seated leg-press and supine bench-press.

Simulated 2000-m Rowing Race

The 2000-m simulated rowing race was performed on a Concept Model IIC rowing ergometer (Morrisville, VT). This test involved subjects rowing at a self-selected pace so as to complete the predetermined distance of 2000-m as quickly as possible, as previously described by Gilles and Bell (2000). Mean power output (PO), split times, heart rate (HR), and total time were recorded every 200-m. Subjects were required to complete the 2000-m trial once it began and had the choice of verbal encouragement; however, they were required to maintain a pace as set out by themselves (no verbal pacing or timing encouragement was given). Subjects were able to view the monitor on the rowing ergometer and set their own pace according to this feedback.

Maximal Oxygen Consumption Test

The maximal oxygen consumption test was also performed on a Concept IIC rowing ergometer as previously described (Gilles and Bell, 2000). Expired gases were determined and analyzed via computerized metabolic measurement systems (Medgraphics CPD, Minneapolis, MN for the pre-test and Sensormedics Horizon MMC, California for the post-test) calibrated from know gas concentrations. The change in metabolic measurement systems was made due to problems that occurred with the first metabolic measurement system at the time of the post-testing. Heart rate was monitored using telemetry from a Polar Pacer heart rate monitor (Polar USA, Connecticut). The test

commenced at a PO of 50W for females and 100W for males with an increase of 50W every 2-minutes until volitional fatigue or VO_{2max} was achieved. Stroke rate was self-selected by the subjects to achieve the desired PO. VO_{2max} was determined when the following criteria were met; a peak or plateau in VO_2 (< 0.100 l·min^{-1}) with an increase in PO, a respiratory exchange ratio of 1.15 or greater, age predicted maximal heart rate (220-age) was obtained, and volitional exhaustion. Data obtained from this test was used in determining fitness levels and the aerobic training intensities for the 10-week aerobic training program.

Multiple Repetition Maximum Testing

A multiple repetition maximum (mRM) test was completed for bilateral leg press at a 45°-incline and supine bench press exercises as previously described (Syrotuik et al., 2001). Subjects were asked to warm-up on a cycle ergometer for a minimum of 5-minutes prior to the commencement of the testing. Testing on the leg-press began with a measurement of the 90° flexion point using a goniometer and a mark was placed on the side of the leg-press machine to indicate this point. This provided a standardized range of motion in which the subject brought their legs down to a consistent flexed position and then pressed upwards at a 45° angle to an extended position. The first set provided a specific warm-up and was done with a light intensity resistance (a load that the subject was able to lift 10-12 times comfortably). The second set required the resistance to be increased so that the subject was able to perform 8-10 repetitions that was described as being difficult in intensity. Following a 2-3 minute rest, a third set was performed where the subject used a self-selected load that created momentary muscular fatigue, preferably between the 8th and 10th repetition. If the subject was able to complete more than 10 or

less than 6 repetitions, a fourth set was performed, with the load adjusted accordingly to result in failure between the 8th and 10th repetition. The actual load lifted and the repetition number at which failure occurred was recorded and used to predict an estimate of 1RM for each lift using a commercially available software package (Power 5.1, Lincoln, Nebraska).

The testing protocol for the bench press was the same as the leg press. Briefly, subjects were requested to keep their feet on the floor, and back in contact with the bench. The bar was lowered from the extended position, followed by a brief pause when touching the bar to their chest, followed by full extension. In addition to the two testing lifts, subjects completed self-tests of the lateral pull down, seated row, upright row, leg extension, hamstring curl, calf raise, biceps curl, and triceps extension using the same protocol. All mRM scores were entered into the same software program for generation of the strength-training program.

Echocardiographic Measures

All echocardiographic measures were performed in accordance with the American Society of Echocardiography guidelines (Sahn et al., 1978) using a Hewlett-Packard ultrasound instrument (Sonos 5500, Hewlett-Packard, Massachusetts) with a 3.5-MHz transducer from the left apical area at the level of the mitral valve leaflets at rest, immediately following the 2000-m trial, 5-minutes after exercise, and 45-minutes after exercise. All echocardiographic measurements were made with an accompanied measurement of systolic blood pressure (SBP) taken by one of the investigators at the time of image acquisition. Changes in LV structure and function were examined using the following measures obtained from 2-D echocardiographic images; LV end-diastolic

cavity area (EDCA), LV end-diastolic total area (EDTA), LV end-diastolic myocardial area (ESMA), LV end-systolic cavity area (ESCA), LV end-systolic total area (ESTA) enclosed by the epicardium, LV end-systolic myocardial area (ESMA), LV stroke area (SA), LV fractional area change (FAC), left-ventricular contractility (systolic blood pressure to ESCA ratio), and LV end-systolic meridional wall stress (σ). LV mass (LVM) was calculated as described previously (Devereux et al., 1986). The same echocardiographic technician performed all echocardiographic procedures during each phase of testing and echocardiograms were recorded on video for analysis at a later date at which time all echocardiographic images were averaged over 3 cardiac cycles and analyzed by a trained cardiologist.

Calculations

1. EDMA = EDTA – EDCA
2. ESMA = ESTA – ESCA
3. SA = EDCA – ESCA
4. FAC = (EDCA – ESCA)/EDCA
5. $\sigma = 1.33 \times SBP (ESCA/ESMA)$
6. $LVM = 0.83[(EDD + MWT + MWT)^3 - (EDD)^3] + 0.6$ (modified from Devereux et al., 1986)
7. $BSA = 0.007184 \times height^{0.725} \times weight^{0.425}$ (Dubois and Dubois, 1916)

Lactate Measurements

All lactate measurements were taken using a finger prick blood sample at rest, 5-min after and 45-min after exercise. Subjects had their finger cleaned with alcohol and a sterile auto-lancet was used to prick the finger. A single drop of blood was placed on a

sterile plastic strip that was inserted into an automated Accusport blood lactate analyzer for the determination of blood lactate concentrations. Following this procedure the subject had a gauze pad placed over the finger prick and applied pressure to stop bleeding. Once bleeding had ceased a Band-Aid™ was placed on the finger.

The validity and reliability of the lactate analyzer was assessed using biochemical analysis in our laboratory (see appendix 1). The Accusport portable lactate analyzer has been compared to numerous methods for determining lactate concentration both at rest and following various intensities of exercise. Naik et al. (1996) showed very high correlations (r=0.976) between the Accusport and theYSI 1500 (Yellow Springs Inc.) in 30 athletes performing 4 minute stage incremental cycling.

Training

Subjects were required to perform 10-weeks of combined strength and endurance training and were provided with specific programs that were periodized using 3-week cycles for endurance training and 2-week cycles for strength training. This period of training time was chosen as it has been shown that longer periods of concurrent training can have negative effects on strength gains (Bell et al., 2000). Strength and endurance training was completed on separate days 3 times per week, respectively: Monday, Wednesday, and Friday for endurance training, Tuesday, Thursday, and Saturday for strength training.

The aerobic training program consisted of three supervised training days per week on Concept IIB and IIC rowing ergometers. Each training session included a warm-up period, followed by performance of the prescribed exercise intensity and duration, ending with a cool down period. Mondays were continuous training days in which the subjects

exercised at an intensity just below ventilatory threshold (VT) as determined during the VO_{2max} test. VT was determined as the lowest point in VE/VCO_2 vs. power output prior to a systematic increase (Bhambhani and Singh, 1985). Subjects rowed at a 5 second split time range that was associated with an intensity just below ventilatory threshold (VT). Wednesdays consisted of a split session performed at an intensity associated with the VT (i.e. the split time that the subject's VT occurred at during testing). The two split sessions were split by a 5-minute period of light rowing and/or stretching. Fridays were interval training days at an intensity equivalent to the mean 500-m split time range (min:sec/500-m) of the initial simulated 2000-m race. Subjects rowed for 500-m at a high intensity followed by 500-m at a lower intensity (30-45 second split time that was slower than their high interval split time). Subjects recorded total time, HR range, stroke rate, and average split time for each. The average daily and weekly intensities and distances are shown in appendix 2.

The strength-training program was periodized and progressively overloaded every 2 weeks. Subjects performed a variety of upper and lower body exercises including: incline leg press, bench press, seated row, leg extension, leg curl, lateral pull down, upright row, calf raise, biceps curl, and triceps extension. Subjects recorded attendance for all strength sessions. The training intensities were determined from the initial strength testing exercises. Each exercise was assigned to one of four intensity schedules. Bench press and leg press were in schedule 1, lateral pull down and seated row were in schedule 2, leg-extension, and leg curl were in schedule 3, while upright row, calf raise, biceps curl, and triceps extension were in schedule 4. The average daily and weekly intensities are presented in appendix 3.

Statistical Analysis

Statistical analysis was performed on a commercially available statistical software package (STATISTICA, Oklahoma City, Oklahoma) using a two-way analysis of variance (ANOVA) with repeated measures to compare before, immediately following and 5- and 45-min after the 2000-m rowing race, as well as before and after training for echocardiographic and blood lactate measurements. Strength, VO_{2max}, and 2000-m times were analyzed with a one-way ANOVA with repeated measures (before and after training). Any significant F-ratios were evaluated with a Neuman-Keuls post-hoc test (Vincent, 1995). Alpha was set a priori at $p<0.05$ for all analyses. All values are reported as Mean ± SD.

3.3 Results

Subject Characteristics

The mean age and height of the participants was 31.2 ± 12.1 years and 184.4 ± 4.0 cm respectively (Table 3-1). Mean weight prior to the 10-week strength and endurance-training program was not statistically significant from the post training weight (79.2 ± 9.0 kg vs. 79.3 ± 8.6 kg).

Performance Trials

Performance data are presented in Table 3-1. VO_{2max} and maximal power output significantly increased following the 10-week training program (19.4 % and 14.8 % respectively). 2000-m time significantly decreased upon completion of the training program (456.8 ± 30.9 s vs. 436.1 ± 18.2 s). Mean power output for the 2000-m trial also increased significantly following the combined strength and endurance-training program. There was also a significant increase in predicted 1RM leg press strength (564.3 ± 159.8

kg vs. 746.3 ± 97.7 kg). There was no change in 1RM bench press strength (160.6 ± 29.9 kg vs. 169.1 ± 26.9); however the difference was equivalent to a 5 % increase.

Lactate

There was a main effect for blood lactate response before and during recovery from the 2000-m rowing trial, but there was no main effect for training or no interaction effect between lactate measures at any measurement time. Prior to training, lactate significantly increased from rest to the 5-min measurement (Table 3-2). Lactate production significantly decreased from 5-min post at 45-min post, but was still significantly higher than at rest. Following training, lactate production increased significantly from rest at 5-min post exercise. At 45-min post, lactate was significantly less than 5-min post, but not significantly different than rest.

3.3.1 Effects of a 2000-m trial on left ventricular function prior to 10-weeks of combined strength and endurance training

Changes in LV structure and function at rest and during recovery from a 2000-m rowing trial are shown in Table 3-3. Volume conversions are presented in Table 3-4.

Heart Rate

At each measurement time, heart rate was significantly different from all other measurement times (Figure 3-1).

Blood Pressure Responses

Blood pressure responses are shown in Figure 3-2. SBP increased significantly from rest immediately following the 2000-m trial. SBP then decreased significantly from immediately after the 2000-m trial at 5-min after exercise, but was not significantly different from rest. SBP was significantly lower at 45-min after exercise compared to

immediately after and 5-min after exercise, but was not significantly different from rest. There were no significant differences in DBP at any measurement time.

LV Measures

There were no significant differences between EDCA, ESTA at any measurement time (Table 3-3). ESCA responses are shown in Figure 3-3. ESCA decreased significantly immediately following the 2000-m trial (10.21 ± 1.90 cm^2 vs. 6.20 ± 2.14 cm^2). 5-min after exercise ESCA increased significantly from immediately following the 2000-m trial, but was still significantly less than rest. At 45-min after exercise, ESCA was significantly higher than immediately after and 5-min after exercise, but was not significantly different from rest.

Stroke Area

SA decreased immediately following the 2000-m trial, however this was not significantly different than rest (Table 3-4). At 5-min after exercise, SA increased significantly from rest and immediately following the 2000-m trial. At 45-min after exercise, SA decreased significantly from 5-min after exercise, but was not significantly different from rest or immediately following the 2000-m trial. See Figure 3-4 for changes in SA.

Fractional Area Change

FAC increased significantly from rest immediately following the 2000-m trial (0.51 ± 0.06 vs. 0.63 ± 0.09). FAC remained significantly higher than rest at 5-min after exercise, but was not significantly different from immediately after exercise. 45-min following the 2000-m trial, FAC was significantly less than immediately after and 5-min after exercise, but not significantly different from rest (Figure 3-5).

Systolic Blood Pressure to End-systolic Cavity Area Relation

SBP/ESCA ratio was used as a surrogate measure of LV contractility (Sagawa et al., 1977). SBP/ESCA ratio responses are shown in Table 3-3 and Figure 3-6. SBP/ESCA ratio increased significantly from rest immediately following the 2000-m trial (10.77 ± 3.14 vs. 27.27 ± 10.38). At 5-min after exercise, the SBP/ESCA ratio significantly decreased from immediately following the 2000-m trial, but remained significantly elevated from rest. 45-min after the 2000-m trial, the SBP/ESCA ratio decreased significantly from immediately after and 5-min after exercise, but this was not significantly different from rest.

Wall Stress

There were no significant differences in wall stress at any measurement time (Figure 3-7).

3.3.2 Effect of 10-weeks of combined strength and endurance training on left ventricular structure and function at rest.

LV function was measured at rest following the 10-week strength and endurance-training program. Measures before and after training are shown in Table 3-5.

There were no significant differences before training vs. after training in FAC or wall stress, however there were significant changes in all other measured variables. ESCA increased 6.66 % following 10-weeks of training (10.21 ± 1.90 cm^2 vs. 10.89 ± 1.56 cm^2). SA significantly increase from 10.43 ± 1.66 cm^2 to 11.93 ± 1.6 cm^2 after the training program. LVM significantly increased before training vs. after training (179.07 ± 46.91 g vs. 210.46 ± 51.13 g). When corrected for body surface area, the LVMi ratio

was still significantly greater after training compared with before training (88.12 ± 20.11 g/cm^2 vs. 103.47 ± 22.04 g/cm^2) (Figure 3-8).

Individual data for resting ESCA, ESTA, ESMA, EDCA, EDTA, EDMA and LVMi was analyzed at rest before and after training and are presented in Figures 3-9 to 3-15, respectively. ESCA increased in 7 of 10 subjects, while ESTA and ESMA increased in 9 out of 10 subjects. EDCA increased in 8 out of 10 subjects, while EDTA and EDMA increased in all subjects and 9 out of 10 subjects, respectively. LVMi increased in all but 1 subject. One subject was omitted from the analysis of LVMi as a post measure for weight was not obtained and BSA could not be calculated. Subject 1 had the greatest increase in ESCA and EDCA along with the largest heart (LVMi) pre training and the largest increase in LVMi following training, however this was not the trend for all subjects. Those subjects with the lowest LVMi prior to training had the lowest LVMi after training with the exception of 2 subjects.

3.3.3 Effects of a 2000-m trial on left ventricular function following 10-weeks of combined strength and endurance training

Upon completion of the 10-week combined strength and endurance-training program, the subjects completed a second 2000-m trial. There was no interaction effect between exercise measurement time (rest, immediately after, 5-min after, and 45-min after) and training (before and after training) for any measured variable (Table 3-6). There was a significant main effect for exercise measurement time in all variables except for ESTA, ESMA, EDTA, EDMA, and wall stress and a significant main effect for training in ESCA, EDCA, EDTA, EDMA, SA, and SBP/ESCA ratio. Responses to the

2000-m trial after the completion of the 10-week strength and endurance-training program are shown in Table 3-7.

Heart Rate

At each measurement time, heart rate was significantly different from all other measurement times (Figure 3-1).

Blood Pressure Responses

SBP increased significantly from rest immediately following the 2000-m trial (Figure 3-2). SBP then decreased significantly from immediately after exercise at 5-min after exercise, but was still significantly greater than rest. SBP was significantly lower at 45-minutes after exercise compared to immediately after and 5-min after the 2000-m trial, but was not significantly different from rest. DBP was not significantly greater than rest immediately following the 2000-m trial. However, DBP was significantly greater immediately after than at 45-min after exercise.

LV Measures

There were no significant differences between ESMA at any measurement time. ESCA decreased significantly immediately following the 2000-m trial (10.89 ± 1.56 cm^2 vs. 6.61 ± 1.20 cm^2) (Figure 3-3). 5-min after exercise, ESCA increased significantly from immediately after the 2000-m trial, but was still significantly less than rest. ESCA continued to increase and at 45-min after exercise it was significantly different from immediately after and 5-min after exercise, but was not significantly different from rest. ESTA decreased significantly from rest immediately following the 2000-m trial (Table 3-3). At 5-min after exercise, ESTA was still significantly less than rest, but not significantly different from immediately post. 45-min after the 2000-m trial ESTA was

not significantly different from rest, but was significantly greater than immediately after and 45-min after. EDCA was significantly lower following the 2000-m trial (22.82 ± 2.17 cm^2 vs. 18.81 ± 2.43 cm^2).

Stroke Area

There were no significant differences in SA between any measurement time (Figure 3-4).

Fractional Area Change

FAC increased significantly from rest immediately following the 2000-m trial (0.52 ± 0.05 vs. 0.65 ± 0.07). FAC remained significantly higher than rest immediately after the 2000-m trial and 5-min after exercise. 45-min following the 2000-m trial, FAC was significantly less than immediately after and 5-min after, but not significantly different from rest (Figure 3-5).

Systolic Blood Pressure to End-systolic Cavity Area Ratio

SBP/ESCA ratio increased significantly from rest immediately following the 2000-m trial (9.97 ± 2.78 vs. 23.99 ± 5.12 (Figure 3-6). 5-min after the 2000-m trial the SBP/ESCA ratio significantly decreased from immediately after, but remained significantly elevated from rest. The SBP/ESCA ratio then decreased significantly from immediately after and 5-min after exercise, but this was not significantly different from rest.

Wall Stress

There were no significant differences in wall stress at any measurement time (Figure 3-7).

3.4 Discussion

The purpose of this study was three fold. First, LV structure and function was evaluated at rest, immediately following and during recovery from a high intensity exercise bout in the form of a 2000-m rowing race. Second, the effects of a 10-week combined strength and endurance-training program on LV structure and morphology at rest was investigated. Third, the effects of the 10-week strength and endurance training program on LV structure and function at rest, immediately following and during recovery from a 2000-m rowing race were determined. It was hypothesized that the 2000-m rowing race would result in a decrease in LV systolic function, specifically a decrease in LV contractility due to an increase in blood lactate and a decrease in blood pH. The results of this study do not support this hypothesis as there was an increase in contractile function immediately following the 2000-m race, despite an increase in blood lactate, that returned to near resting values during recovery. It was also hypothesized that 10-weeks of combined strength and endurance training would result in an increase in LV morphology due to the adaptations associated with increased wall stress resulting from volume and pressure overload from training. The results support this hypothesis, as there was an increase in EDCA, LVM and LVMi at rest. Finally, it was hypothesized that following 10-weeks of combined strength and endurance training LV systolic function would be improved immediately following the 2000-m trial. However, following training there was no difference in LV systolic function following the 2000-m race compared with the pre-training 2000-m trial.

Although there was a significant increase in blood lactate following the 2000-m trial before training, there was an increase in both the FAC and the SBP/ESCA ratio in

combination with decrease in ESCA and no change in EDCA or wall stress. These results would suggest an increase in the contractile state of the myocardium, but no increase in Frank-Starling response. This supports the findings of Goodman et al. (1991) who found an increase in the SBP/ESV ratio during moderate to intense exercise. These later researchers found an increase in LV contractility of 93 % from rest to maximal exercise, which is less than the 153 % increase found in this study. The lack of improvement in preload (increase in EDCA) was not supported by Goodman's work, as he found an increase in EDV. Di Bello et al. (1994) also showed an improvement in contractile state of the left ventricle along with an improvement in the Frank-Starling mechanism during maximal supine exercise. The lack of improvement in preload in this investigation may be attributed to a decrease in diastolic filling due to increased heart rate. It may also be a reflection on the prolonged maximal intensity required of the 2000-m rowing race. Goodman et al. (1991) suggest that exercise below VT is more dependent on the Frank-Starling mechanism, while exercise above VT is more dependent on contractile function. Improvements in contractile function may outweigh the need for improving loading conditions during prolonged near maximal efforts. The return of SBP/ESCA ratio to resting values during recovery is a result of a decrease in SBP and a concomitant increase in EDCA and ESCA, which may be a result of a decrease in circulating catecholamines (Hartley, 1974).

The 23 % increase in FAC follwing the 2000-m trial before training is supported by Lorell and Grossman (1986) who suggest that an improvement in FAC of greater than 5 % is expected with exercise. This increase in FAC would also suggest an improvement in contractile function that was due to a large decrease in ESCA. These results support

the work of Plotnick et al. (1986) who found an increase in ejection fraction immediately following exercise that was the result of a decreased end-diastolic volume and a decreased end-systolic volume. The return of FAC to resting values during recovery was a result of an increase in both EDCA and ESCA. Again, this is paralleled by Plotnick et al. (1986), who suggest that EF recovers due to a decrease in afterload with a continued sympathetic stimulation.

The increase in contractility following the 2000-m rowing trial before training can be attributed to a number of mechanisms: first, our subjects did not achieve the high levels of lactate production and pH reduction as seen by Neilson (1999). If lactate production or decreases in pH contribute to a decrease in contractility there may be a critical level, which was not achieved in this investigation. Second, an increase in circulating catecholamines associated with high intensity exercise may outweigh the negative effect of increased blood lactate and decreased blood pH. Third, the decrease in diastolic filling time during exercise limits the time for the potential diffusion of lactate and H^+ ions from the blood into the cardiac myocyte.

The increase in heart rate following the 2000-m race was expected and is attributed to vagal withdrawal and an increase in inotropic stimulation from circulating catecholamines (Hopkins et al., 1996). The return to resting values during recovery is a result of the removal of circulating catecholamines, a decrease in sympathetic drive and in increase in vagal tone. The 46 % increases in SBP from rest to immediately after the 2000-m trial was expected, although greater than those found by Rosiello et al. (1987) and Clifford et al. (1994) who saw a 31% and 34% increase, respectively in SBP following a 6-min all out rowing trial. The increase in SBP has been attributed to an

increase in cardiac output resulting in an increase in LV mechanoreceptor activation, an increase in the activity of atrial baroreceptors and an increase in sympathetic drive (Lundbrook, 1983). The return to resting values during recovery was also expected and can be attributed to decreased sympathetic drive during recovery (Blomqvist, 1983). The lack of change in DBP following the 2000-m trial was similar to that of Rosiello et al. (1987), who also found no change in DBP following an incremental exercise test on the rowing ergometer. This lack of change in DBP has been attributed, in part, to a decrease in peripheral resistance (MacDougall, 1994).

There was no significant change in SA following the 2000-m rowing race and after converting SA to stroke volume (SV) (Table 3-5), there were also no further changes observed despite a non-significant decrease in SV was seen following the 2000-m race. Although this was not significant, the significant increase in cardiac output (Table 3-5) following the 2000-m trial was probably due to an increase in HR (Ekblom and Hemansen, 1968). Although there was no significant difference in SA following the 2000-m race, there was a significant increase in SA at 5-min after the 2000-m rowing race. This increase was a result of a larger increase in EDCA. This finding cannot be adequately explained, but perhaps it is a result of the subjects being required to move into a supine position to obtain the echocardiographic images. This movement into the supine position could result in an increase in venous return and an increase in preload (Poliner et al., 1980). This does not seem to be a consistent observation as EDCA and SA are lower 45-min after the 2000-m race and subjects were again required to move to a supine position.

The increase in contractile function found in this investigation was consistent with the responses to increasing exercise intensity (Blomqvist, 1983). However, it was inconsistent with the hypothesis that increasing levels of blood lactate and decreasing blood pH reduce LV contractile function in perfused hearts (Samja et al., 1999). Neilson (1999) has shown that following a 2000-m rowing trial, subjects experienced some of the highest blood lactates and lowest blood pH levels ever reported, and these values are consistent with the concentrations used by Samja et al. (1999). Our subjects did not obtain these values (12.72 ± 4.64 mmol·L^{-1} vs. 32 mmol·L^{-1}), which may be a result of the differences in trained state of our subjects (club level rowers) and those used in Neilson's study (World class rowers). Although the subjects in this investigation increased fitness levels and improved performance time, there was no significant increase in lactate production following the 10-week training program. The perfused hearts used by Samaja et al. (1999) eliminated many of the hormonal and mechanistic responses to exercise allowing them to show a response to a single event. The lack of similar response in this investigation may be due to some protective mechanism for cardiac function in vivo such as increasing catecholamine response to exercise (Hopkins et al., 1996).

Structural and morphological changes in the left ventricle at rest following combined strength and endurance training

Prolonged exercise training has been shown to increase the absolute size of the heart, as well as heart size relative to body surface area compared with sedentary controls (Longhurst et al., 1980; Fagard et al., 1983; Urhausen et al., 1996). This change in heart size is due to two possible mechanisms. First, volume overload consistent with endurance training can lead to eccentric hypertrophy of the left ventricle characterized by

the addition of sarcomeres in series (Fagard, 1997). The volume overload from endurance training results in an increase in the diastolic wall stress, which causes the replication of sarcomeres in series, elongation of fibres, and an increase in chamber size. This results in a decrease in diastolic wall stress due to an increase in cavity dimension. The increase in chamber size results in an increase in systolic wall stress that is minimized by increasing the number of sarcomeres in parallel. Second, prolonged exposure to pressure overload as a result of resistance training may lead to concentric hypertrophy, which is characterized by the addition of sarcomeres in parallel in order to reduce systolic wall stress (Fagard, 1997). The resultant decrease in systolic wall stress is due to an increase in wall thickness with little or no increase in cavity dimension. It has been shown that rowers have some of the largest hearts ever recorded, which may be due to the combination of strength and endurance-training, that accompanies the sport of rowing or genetic endowment of those who choose to participate in rowing (Pelliccia et al., 1991; Cavallaro et al., 1993; Spirito et al., 1994; Gustafsson et al., 1996).

The results of this study support the notion that combined strength and endurance training in rowers produces an appropriate stimulus to increase LVM and LVMi. The 17.5 % increase in LVM was associated with a 10.6 % increase in EDCA and an 11.3 % increase in EDMA, suggesting eccentric hypertrophy. Cavallaro et al. (1993) found that following 5 months of training, male rowers showed significant increases in both LV internal dimension during diastole and LVMi (3.6 % and 33.5 % respectively). Although Haykowsky et al. (1998) found an increase in LV internal dimension, they found no increase in LVM following 10-weeks of combined strength and endurance training in rowers, suggesting that 10-weeks of training was an insufficient time period to produce

an increase in LVM. The results of this investigation support the findings of Cavallaro et al. (1993) as opposed to those of Haykowsky et al. (1998). One possibility for the difference between the studies is the trained state of the subjects. Although Cavallaro et al. (1993) do not state the fitness level of their subjects, they suggest that they were 'top level rowers'. Haykowsky et al. (1999) had moderately trained subjects (VO_{2max} = 4.346 ± 0.652 $L \cdot min^{-1}$ pre- training and 4.471 ± 0.624 $L \cdot min^{-1}$ post-training), while the participants in this study were less fit pre-training 3.405 ± 0.324 $L \cdot min^{-1}$ as well as post-training 4.064 ± 0.469 $L \cdot min^{-1}$. The initial trained state of the subjects (either highly trained or less fit vs. moderately trained) may play a role in the time course of the development of an increase in LVMi.

The substantial increase in LVM and LVMi (17.53 % and 17.41 % respectively) resulted in values that are similar to those found in a number of studies evaluating the 'athlete's heart'. Pelliccia et al. (1991) evaluated 947 elite level athletes for signs of cardiac hypertrophy and found LVM to be 206 ± 46 g and LVMi to be 105 ± 20 g/m^2. The training induced increases in the present study resulted in an LVM of 210.46 ± 51.13 g and an LVMi of 103.46 ± 22.04. Maron (1986) compared highly trained athletes with non-athlete controls and found LVM to be 46.3 % greater in the athletes than the controls (175 g vs. 256 g). Based on Maron's (1999) work, the subjects in this study could be considered non-athletic at the pre-training measurement time and following training LVM began to increase to the level of the trained athletes (Table 3-5). In a recent meta-analysis, Pluim et al. (1999) compared the incidence of 'athlete's heart' in groups that participated in different types of training (endurance, resistance, and combined) and although not significantly different, athletes that underwent combined strength and

endurance training had the highest LVM of the three. They do agree that training specific adaptations in LV structure and morphology do exist, but these adaptations may not be as straightforward as once thought. Finally, although there were increases in LVM and LVMi, these increases were well within the physiological range for LV hypertrophy (Maron, 1986).

The increased LVMi may be in part due to an increased volume load (increased EDCA) or an increased afterload following training as shown by an increased ESCA. Both of these stimuli result in greater wall stress, a stimulus for LV hypertrophy. In order to decrease wall stress, the myocardium thickens. Following 10-weeks of combined strength and endurance training, there was an increase in ESCA and a concurrent increase in ESMA (6.66 % and 5.54 % increase), which can explain the lack of change in wall stress (Grossman et al., 1975).

Another important finding of this study was the increase in EDCA following training. This suggests that there was an improvement in preload and a concurrent improvement on the Frank-Starling mechanism. Along with this improvement in loading conditions was a decrease in the ESCA, which suggests a decrease in the contractile function of the heart and more reliance on loading conditions (Blomqvist, 1983). The increase in EDCA resulted in an increased SA; however there was no change in FAC as ESCA also increased (Robotham et al., 1991).

Effect of 10-weeks of combined strength and endurance training on performance variables

There were significant improvements in most physiological variables following 10-weeks of combined strength and endurance training. The combined strength and

endurance training program employed in this investigation was consistent with those used by rowers in the off-season, and in previous investigations performed in this laboratory (Syrotuik et al., 2001; Haykowsky et al., 1998; Bell et al., 1991; Bell et al., 1993).

There was a 19.35 % increase in VO_{2max} that was associated with a 14.38 % increase in SA following training. As VO_{2max} is dependent on HR and SV (measured as SA in this investigation) (Ekblom and Hermansen, 1968) and there was no change in resting or maximal HR following training, the increase in VO_{2max} could be a result of the increased SA. Although there was an increase in SA following training that was similar to the increase in VO_{2max}, after training, increases in VO_{2max} are dependent upon a number of factors including an improvement in peripheral oxidative capacity and increased capillary to fibre ratio (Bell et al., 2000), which most likely occurred although they were not measured.

Improvements in VO_{2max} (19.35 %), power output at VO_{2max} (14.75 %), 2000-m trial time (-4.53 %), mean power output during the 2000-m trial (13.38 %) and predicted 1RM leg press (32.25 %) were greater than those seen by Haykowsky et al. (1998). Although the training program employed by both this study and Haykowsky et al. (1998) were very similar, the greater improvements, along with a lower initial fitness level of the participants in this study may have been the reason for the increased LVM and LVMi following training in the present study.

Following training, blood lactate concentrations were significantly lowered at 45-min after exercise. This suggests an improvement in lactate clearance during recovery following training. Blood lactate concentration was not significantly different immediately after exercise, before and after 10-weeks of training. These findings support

the notion that endurance training improves lactate clearance rather than reducing the rate of lactate production (Donovan and Brooks, 1983).

Effects of a 2000-m trial on left ventricular function following 10-weeks of combined strength and endurance training

There was no significant interaction effect between any variable at any measurement time before and after training. The responses of all variables followed similar trends during recovery from the 2000-m trial after training. The changes in LV morphology at rest that occurred following 10-weeks of combined strength and endurance training, do not appear to have any influence on the acute responses immediately following and during recovery from a 2000-m trial. The lack of improvement in LV function following a 2000-m rowing trial after 10-weeks of combined strength and endurance training does not support the work of Wolfe et al. (1986) who found an increase in SV during exercise, following endurance training of 3-15 months. They suggest that this increase in SV was associated with an increase in end-diastolic volume and use of the Frank-Starling mechanism. Although there was increase in SA at rest following 10-weeks of training, there was no significant difference in SA immediately following the 2000-m trial before and after training. Improvements in LV function immediately following a 2000-m rowing trial were attributed to improvements in contractile function as expressed by an increase in the SBP/ESCA ratio (due to decreased ESCA), as opposed to increases in the use of the Frank-Starling mechanism (as EDCA was lower immediately following the 2000-m trial). The decreased ESCA following the 2000-m rowing trial is supported by Pokan et al. (2000) who found decreases in ESCA following acute exercise. Although the SBP/EDCA ratio increased

following exercise, there was no improvement compared to before training. The reliance on improved contractile function, as opposed to improved loading conditions, is similar to the pre-trained state, suggesting that the time-course for improving loading conditions may be longer than 10-weeks.

Following training, SA 5-min after the completion of the 2000-m trial was lower than immediately after exercise, suggesting a return towards resting values. This differs from the trend before training in which SA increase at 5-min after exercise, suggesting that during the initial test there was a measurement error as opposed to a physiological response.

The lack of any interaction effect between training time and exercise time suggests that 10-weeks of combined strength and endurance training is insufficient to provide any alteration in responses to recovery from a 2000-m rowing trial. This does not support the hypothesis that 10-weeks of combined strength and endurance training would provide the necessary stimuli to improve LV function during recovery from a 2000-m rowing race.

Effects of 10-weeks of combined strength and endurance training on left ventricular function following a 2000-m trial

Following 10-weeks of combined strength and endurance training there were significant changes in ESCA and EDCA. The increase in ESCA, increase in EDCA, and decrease in the SBP/ESCA relation following training suggests less reliance on contractility and a greater reliance on the lower energy consuming Frank-Starling mechanism. The increase in EDCA can be attributed to the changes in LV morphology

following training. The volume overload during training resulted in an increase in the chamber size (increased EDCA) and an increase in wall thickness (increased EDMA).

Limitations

Limitations of the use of echocardiography to estimate LV systolic function have been previously discussed (Colan et al., 1984). Although only a single measure of LV contractility (SBP/ESCA ratio) was used in this investigation, the increase in SBP/ESCA with no significant increase in EDCA suggests that the improvement in LV systolic function was a result of an increased contractility. However, the mechanisms responsible for this improvement in contractility could not be determined. A second limitation of this study was the use of echocardiographic images during recovery to estimate changes during exercise. Although HR remained significantly elevated from rest, these values are lower than those obtained during the VO_{2max} test (a maximal effort). Pokan et al. (2000) have shown that SBP, end-diastolic dimension, end-systolic dimension, and fractional shortening were all significantly different 15 s into recovery from immediately before cessation of exercise, suggesting that recovery values do not equal exercise values. The standardization of measurement time was very difficult as it was often difficult to move subjects between the rowing ergometer and the echo-bed. The standardization of lactate measures was also difficult as they were dependent on echocardiographic measurement times. Finally, blood lactate was used as an indication of acidosis and despite an established relationship between lactate and H^+ production, blood lactate accumulation is not the most valid indication of pH levels.

Conclusion

This investigation demonstrated that LV systolic function was increased following a simulated 2000-m rowing race, and that these improvements in function returned to resting levels during recovery. There was no blood lactate or blood pH mediated decrease in LV contractility following an acute bout of high-intensity exercise. It was also shown that 10-weeks of combined strength and endurance training provided a stimulus for increasing LVM and LVMi. Although these increases in were significant, they were not at the level of pathologic LV hypertrophy, and were lower than those values found in other investigations of the athletic heart in rowers. Finally, LV systolic function immediately following and during recovery from a simulated 2000-m rowing race was not improved following 10-weeks of strength and endurance training. Future research should continue to determine the mechanisms involved and the role of high intensity exercise and training on the cardiovascular system.

3.5 References

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Table 3-1- Descriptive and performance variables before and after 10-weeks of combined strength and endurance training. Values are means ± SD.

	Pre	Post	% Change
Age (years)	31.2 ± 12.1	-	-
Weight (kg)	79.2 ± 9.0	79.3 ± 8.6	0.13
Height (cm)	184.4 ± 4.0	-	-
VO_{2max} Testing			
VO_{2max} ($L \cdot min^{-1}$)	3.405 ± 0.324	4.064 ± 0.469*	19.35
Stroke Rate	32.11 ± 2.85	32.78 ± 2.59	2.09
Power Output (W)	299.83 ± 50.57	344.04 ± 61.50*	14.75
2000-m Testing			
2000-m Time (s)	456.8 ± 30.9	436.1 ± 18.2*	-4.53
Mean Stroke Rate	28.20 ± 2.25	30.10 ± 2.38*	6.74
Mean 500-m Split (s)	114.21 ± 7.72	99.04 ± 31.08	13.28
Mean Power Output (W)	240.52 ± 45.90	272.71 ± 35.51*	13.38
Strength Testing			
Predicted 1RM Bench Press (kg)	160.6 ± 29.9	169.1 ± 26.9	5.29
Predicted 1RM Leg Press (kg)	564.3 ± 159.8	746.3 ± 97.7*	32.25

VO_{2max}- maximal oxygen consumption, 1RM- 1 repetition maximum
***- significantly different from BEFORE TRAINING**

Table 3-2. Blood lactate ($mmol \cdot L^{-1}$) measures following the 2000-m simulated rowing race. Values are means ± SD.

Measurement Time	Before Training	After Training
Rest	1.45 ± 0.65	1.47 ± 0.71
5-min Post	12.72 ± 4.64 [a]	12.97 ± 4.46 [c]
45-min Post	4.64 ± 2.86 [a,b]	3.09 ± 1.76 [d]

a- significantly different from rest (BEFORE TRAINING)
b- significantly different from 5-min (BEFORE TRAINING)
c- significantly different from rest (AFTER TRAINING)
d- significantly different from 5-min (AFTER TRAINING)

Table 3-3. Changes in left ventricular systolic function during recovery from a simulated 2000-m rowing race (Areas). Values are means± SD.

	Rest	Immediate After	5-min After	45-min After
HR (beats·min^{-1})	70.10 ± 8.01 ^{b,c,d}	147.06 ± 15.31 ^{a,c,d}	113.10 ± 18.06 ^{a,b,d}	95.30 ± 14.65 ^{a,b,c}
SBP (mmHg)	105.20 ± 11.67 ^{b}	153.20 ± 27.13 ^{a,c,d}	121.00 ± 29.12 ^{b,d}	92.60 ± 9.62 ^{b,c}
DBP (mmHg)	62.20 ± 9.16	67.00 ± 10.59	57.80 ± 11.33	57.80 ± 6.56
ESCA (cm^2)	10.21 ± 1.90 ^{b,c}	6.20 ± 2.14 ^{a,c,d}	7.79 ± 1.76 ^{a,b,d}	9.20 ± 2.20 ^{b,c}
ESTA (cm^2)	33.48 ± 3.96	30.37 ± 5.00	36.27 ± 13.28	33.48 ± 5.09
EDCA (cm^2)	20.64 ± 2.59	16.48 ± 2.44	19.74 ± 2.75	18.72 ± 2.89
SA (cm^2)	10.43 ± 1.66 ^{c}	10.32 ± 1.30 ^{c}	11.95 ± 1.81 ^{a,b,d}	9.45 ± 1.61 ^{c}
FAC	0.51 ± 0.06 ^{b,c}	0.63 ± 0.09 ^{a,d}	0.61 ± 0.06 ^{a,d}	0.51 ± 0.06 ^{b,c}
σ (dyn/cm^2)	63.27 ± 15.88	55.70 ± 31.50	54.08 ± 22.73	50.10 ± 16.62
SBP/ESCA	10.77 ± 3.14 ^{b,c}	27.27 ± 10.38 ^{a,c,d}	17.01 ± 5.56 ^{a,b,d}	10.38 ± 2.22 ^{b,c}

HR- heart rate, SBP- systolic blood pressure, DBP- diastolic blood pressure, ESCA- end-systolic cavity area, ESTA- end-systolic total area, EDCA- end-diastolic cavity area, SA- stroke area, FAC- fractional area change, σ- wall stress

a- significantly different from rest
b- significantly different from immediate post
c- significantly different from 5-min post
d- significantly different from 45-min post

Table 3-4. Changes in left ventricular systolic function during recovery from a simulated 2000-m rowing race (Volumes). Values are means ± SD.

	Rest	Immediate After	5-min After	45-min After
HR (beats·min^{-1})	70.10 ± 8.01	146.06 ± 15.31	113.10 ± 18.06	95.30 ± 14.65
SBP (mmHg)	105.20 ± 11.67 [b]	153.20 ± 27.13 [a,c,d]	121.00 ± 29.12 [b,d]	92.60 ± 9.62 [b,c]
DBP (mmHg)	62.20 ± 9.16	67.00 ± 10.59	57.80 ± 11.33	57.80 ± 6.56
ESV (mL)	47.44 ± 12.73 [b,c,d]	23.03 ± 12.20 [a,c,d]	31.79 ± 10.27 [a,b,d]	40.86 ± 13.82 [a,b,c]
EDV (mL)	135.49 ± 24.55 [b,d]	96.84 ± 22.19 [a,c,d]	126.83 ± 26.57 [b]	117.37 ± 26.12 [a,b]
SV (mL)	88.05 ± 17.26	73.81 ± 13.11 [c]	95.03 ± 20.30 [b]	76.50 ± 16.06
Q (L·min^{-1})	6.116 ± 1.099 [b,c]	10.933 ± 2.858 [a,d]	10.596 ± 2.015 [a,d]	7.316 ± 2.119 [b,c]
EF	0.65 ± 0.06 [b,c]	0.77 ± 0.08 [a,d]	0.75 ± 0.06 [a,d]	0.66 ± 0.07 [b,c]
Emax	2.44 ± 1.03 [b]	8.30 ± 4.35 [a,c,d]	4.35 ± 2.17 [b]	3.67 ± 0.78 [b]

HR- heart rate, SBP- systolic blood pressure, DBP- diastolic blood pressure, ESV- end-systolic volume, EDV- end-diastolic volume, SV- stroke volume, Q- cardiac output, EF- ejection fraction, Emax- contractility

a- significantly different from rest
b- significantly different from immediate post
c- significantly different from 5-min post
d- significantly different from 45-min post

Table 3-5. Structural and morphological changes in the left ventricle at rest following combined strength and endurance training. Values are means ± SD.

	Before Training	After Training	% Change
ESCA (cm^2)	10.21 ± 1.90	10.89 ± 1.56 *	6.66
ESTA (cm^2)	33.48 ± 3.96	35.46 ± 3.91 *	5.91
ESMA (cm^2)	23.27 ± 4.86	24.56 ± 4.00 *	5.54
EDCA (cm^2)	20.64 ± 2.59	22.82 ± 2.17 *	10.56
EDTA (cm^2)	41.01 ± 3.63	45.49 ± 4.44 *	10.92
EDMA (cm^2)	20.36 ± 4.40	22.66 ± 4.11 *	11.30
SA (cm^2)	10.43 ± 1.66	11.93 ± 1.63 *	14.38
FAC	0.51 ± 0.06	0.52 ± 0.05	1.96
σ ($dyn \cdot cm^{2-1}$)	63.27 ± 15.88	63.09 ± 15.09	-0.28
LVM (g)	179.07 ± 46.91	210.46 ± 51.13 *	17.53
LVMi (g/m^2)	88.12 ± 20.11	103.46 ± 22.04 *	17.41

ESCA- end-systolic cavity area, ESTA- end-systolic total area, ESMA- end-systolic myocardial area, EDCA- end-diastolic cavity area, EDTA- end-diastolic total area, EDMA- end-diastolic myocardial area, SA- stroke area, FAC- fractional area change, σ- wall stress, LVM- left ventricular mass, LVMi- left ventricular mass index

***- significantly different from BEFORE TRAINING**

Table 3-6. Changes in left ventricular systolic function during recovery from a simulated 2000-m rowing race following combined strength and endurance training. Values are means ± SD.

	Pre				Post				Main Effect Training	Main Effect Exercise
	Rest	Immediate Post	5-min Post	45-min Post	Rest	Immediate Post	5-min Post	45-min Post		
HR beats·min^{-1}	70.10 ± 8.01	147.06 ± 15.31	113.10 ± 18.06	95.30 ± 14.65	72.10 ± 14.62	142.20 ± 13.16	116.30 ± 14.31	93.50 ± 14.42		0.000
SBP (mmHg)	105.20 ± 11.67	153.20 ± 27.13	121.00 ± 29.12	92.60 ± 9.62	105.60 ± 18.03	158.22 ± 26.86	124.7 ± 25.06	91.80 ± 13.05		0.000
DBP (mmHg)	62.20 ± 9.16	67.00 ± 10.59	57.80 ± 11.33	57.80 ± 6.56	58.00 ± 13.98	61.11 ± 10.54	56.00 ± 11.74	45.60 ± 9.08		0.008
ESCA (cm^2)	10.21 ± 1.90	6.20 ± 2.14	7.79 ± 1.76	9.2 ± 2.20	10.89 ± 1.56	6.6 ± 1.20	8.20 ± 2.40	9.90 ± 2.51	0.016	0.000
ESTA (cm^2)	33.48 ± 3.96	30.37 ± 5.00	36.27 ± 13.28	33.48 ± 5.09	35.46 ± 3.91	31.80 ± 3.64	32.80 ± 4.32	34.58 ± 3.60		0.015
ESMA (cm^2)	23.27 ± 4.86	25.17 ± 5.75	28.00 ± 14.24	24.30 ± 5.85	24.56 ± 4.00	25.19 ± 3.63	24.60 ± 4.65	24.69 ± 4.34		
EDCA (cm^2)	20.64 ± 2.59	16.48 ± 2.44	19.74 ± 2.75	18.72 ± 2.89	22.82 ± 2.17	18.81 ± 2.43	19.53 ± 5.80	21.19 ± 3.54	0.018	0.000
EDTA (cm^2)	41.01 ± 3.63				45.49 ± 4.44				0.006	
EDMA (cm^2)	20.36 ± 4.40				22.66 ± 4.11				0.040	

SA (cm^2)	10.43 ± 1.66	10.32 ± 1.30	11.95 ± 1.81	9.45 ± 1.61	11.93 ± 1.63	12.20 ± 2.32	12.03 ± 2.48	11.30 ± 2.44	0.030	0.012
FAC	0.5 ± 0.06	0.63 ± 0.09	0.61 ± 0.06	0.51 ± 0.06	0.52 ± 0.05	0.65 ± 0.07	0.59 ± 0.10	0.53 ± 0.10		0.000
σ ($dyn \cdot cm^2$)	63.27 ± 15.88	55.70 ± 31.50	54.08 ± 22.73	50.10 ± 16.62	63.09 ± 15.09	55.94 ± 15.74	55.65 ± 5.05	50.58 ± 15.25		
SBP/ESCA Ratio	10.77 ± 3.14	27.27 ± 10.38	17.01 ± 5.56	10.38 ± 2.22	9.97 ± 2.78	23.99 ± 5.12	16.54 ± 8.32	10.05 ± 3.47	0.023	0.000

HR- heart rate, SBP- systolic blood pressure, DBP- diastolic blood pressure, ESCA- end-systolic cavity area, ESTA- end-systolic total area, EDCA- end-diastolic cavity area, SA- stroke area, FAC- fractional area change, σ- wall stress

Table 3-7. Changes in left ventricular systolic function during recovery from a 2000-m rowing trial following 10-weeks of combined strength and endurance training. (Areas). Values are means ± SD

	Rest	Immediate After	5-min After	45-min After
HR (beats·min^{-1})	72.10 ± 14.62 [b,c,d]	142.20 ± 13.16 [a,c,d]	116.30 ± 14.31 [a,b,d]	93.50 ± 14.42 [a,b,c]
SBP (mmHg)	105.60 ± 18.03 [b,c]	158.22 ± 26.86 [a,c,d]	124.7 ± 25.06 [a,b,d]	91.80 ± 13.05 [b,c]
DBP (mmHg)	58.00 ± 13.98	61.11 ± 10.54 [d]	56.00 ± 11.74	45.60 ± 9.08 [b]
ESCA (cm^2)	10.89 ± 1.56 [b,c]	6.61 ± 1.20 [a,c,d]	8.20 ± 2.40 [a,b,d]	9.90 ± 2.51 [b,c]
ESTA (cm^2)	35.46 ± 3.91 [b,c]	31.80 ± 3.64 [a,d]	32.80 ± 4.32 [a,d]	34.58 ± 3.60 [b,c]
ESMA (cm^2)	24.56 ± 4.00	25.19 ± 3.63	24.60 ± 4.65	24.69 ± 4.34
EDCA (cm^2)	22.82 ± 2.17 [b]	18.81 ± 2.43 [a]	19.53 ± 5.80	21.19 ± 3.54
SA (cm^2)	11.93 ± 1.63	12.20 ± 2.32	12.03 ± 2.48	11.30 ± 2.44
FAC	0.52 ± 0.05 [b,c]	0.65 ± 0.07 [a,c,d]	0.59 ± 0.10 [a,b,d]	0.53 ± 0.10 [b,c]
σ (dyn·cm^2)	63.09 ± 15.09	55.94 ± 15.74	55.65 ± 15.05	50.58 ± 15.25
SBP/ESCA	9.97 ± 2.78 [b,c]	23.99 ± 5.12 [a,c,d]	16.54 ± 8.32 [a,b,d]	10.05 ± 3.47 [b,c]

HR- heart rate, SBP- systolic blood pressure, DBP- diastolic blood pressure, ESCA- end-systolic cavity area, ESTA- end-systolic total area, EDCA- end-diastolic cavity area, SA- stroke area, FAC- fractional area change, σ- wall stress

a- significantly different from rest ($p<0.05$)

b- significantly different from immediate post ($p<0.05$)

c- significantly different from 5-min post ($p<0.05$)

d- significantly different from 45-min post ($p<0.05$)

Table 3-8. Changes in left ventricular systolic function during recovery from a simulated 2000-m rowing race following 10-weeks of combined strength and endurance training. (Volumes) Values are means ± SD.

	Rest	Immediate After	5-min After	45-min After
HR (beats·min^{-1})	72.10 ± 14.62 [b,c,d]	142.20 ± 13.16 [a,c,d]	116.30 ± 14.31 [a,b,d]	93.50 ± 14.42 [a,b,c]
SBP (mmHg)	105.60 ± 18.03 [b,c]	158.22 ± 26.86 [a,c,d]	124.7 ± 25.06 [a,b,d]	91.80 ± 13.05 [b,c]
DBP (mmHg)	58.00 ± 13.98	61.11 ± 10.54 [d]	56.00 ± 11.74	45.60 ± 9.08 [b]
ESV (mL)	52.03 ± 10.86	24.69 ± 6.73	34.71 ± 14.81	45.75 ± 16.30
EDV (mL)	157.12 ± 22.45	117.84 ± 22.76	128.19 ± 47.25	141.51 ± 35.15
SV (mL)	105.09 ± 17.41	93.15 ± 21.62	93.47 ± 41.53	95.76 ± 18.85
Q (L·min^{-1})	7.467 ± 1.368	13.091 ± 2.466	11.113 ± 5.309	4.558 ± 1.764
EF	0.67 ± 0.06	0.79 ± 0.06	0.66 ± 0.25	0.68 ± 0.10
Emax	2.15 ± 0.78	5.91 ± 2.67	4.55 ± 2.77	2.39 ± 1.25

HR- heart rate, SBP- systolic blood pressure, DBP- diastolic blood pressure, ESV- end-systolic volume, EDV- end-diastolic volume, SV- stroke volume, Q- cardiac output, EF- ejection fraction, Emax- contractility

a- significantly different from rest
b- significantly different from immediate post
c- significantly different from 5-min post
d- significantly different from 45-min post

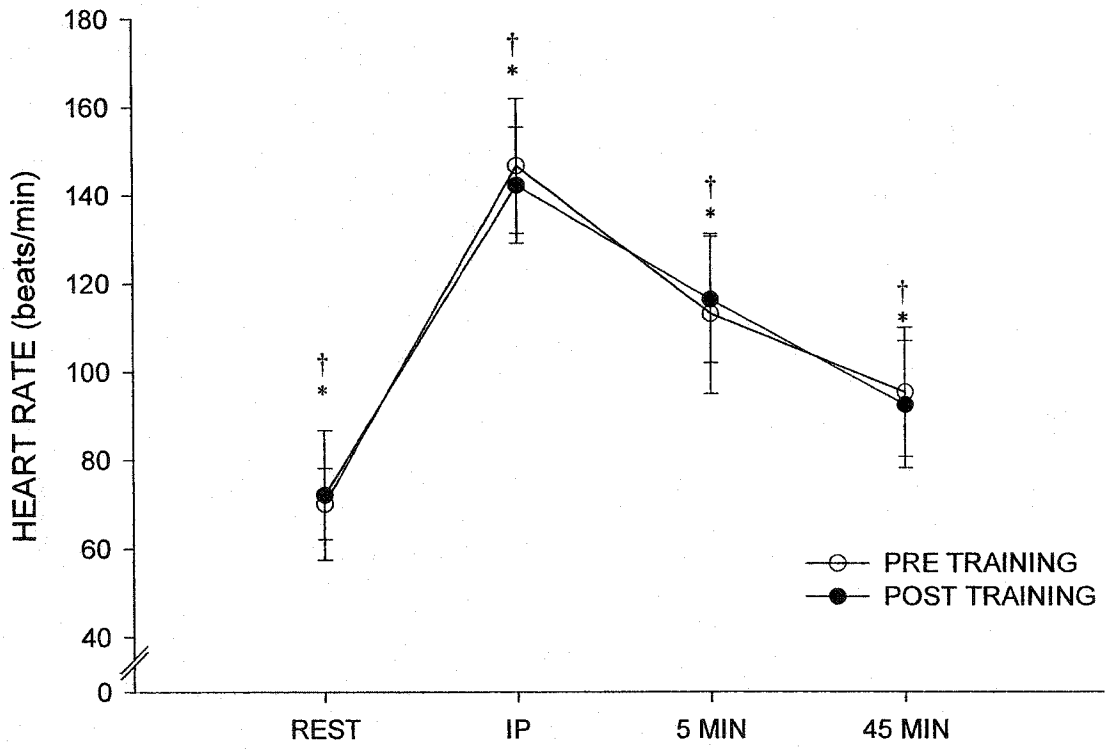

Figure 3-1. Effects of combined strength and endurance training on heart rate following a 2000-m rowing trial. (Error bars = SD)

*- significantly different from all measurement times (PRE)
†- significantly different from all measurement times (POST)

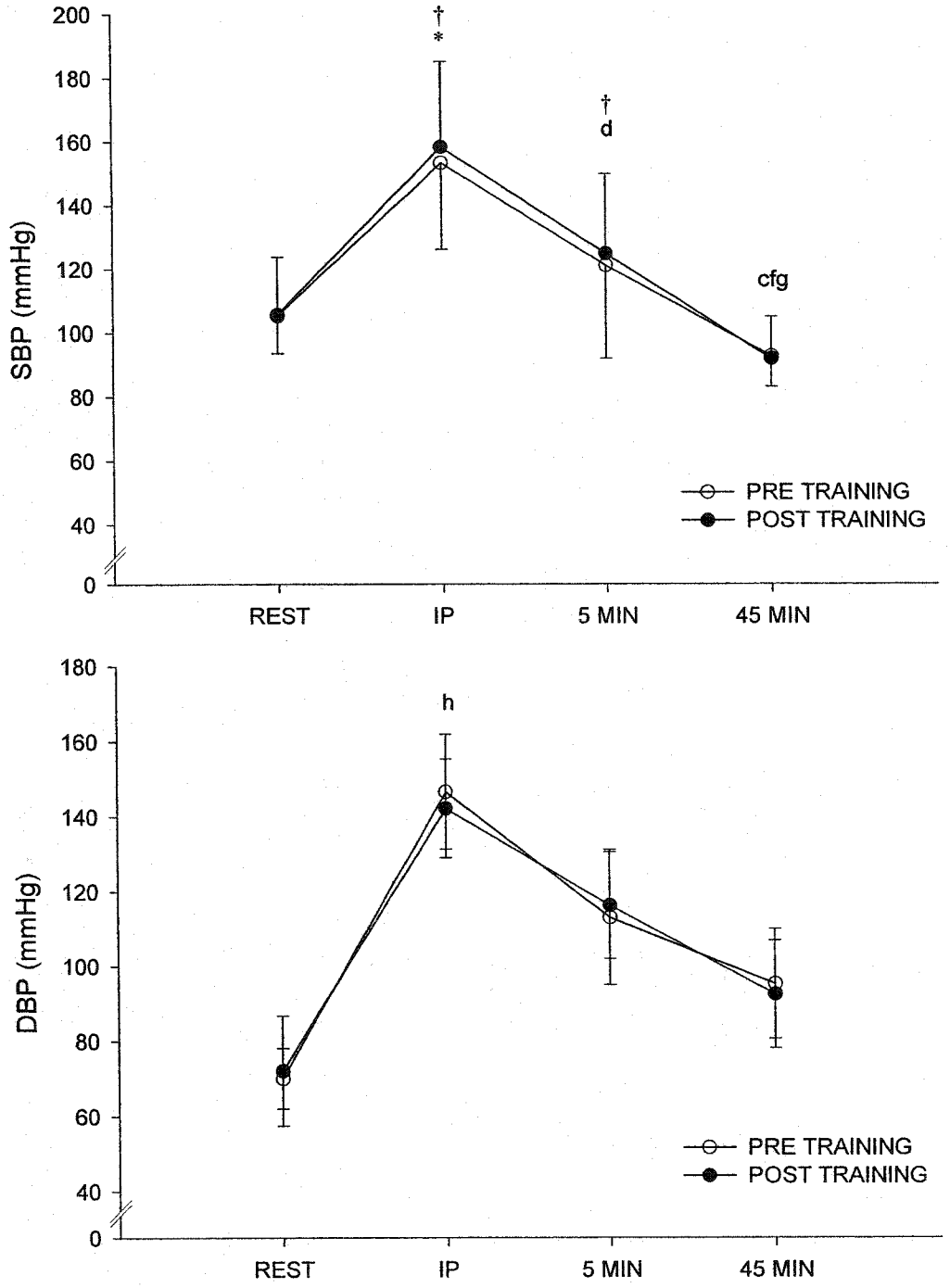

Figure 3-2. Effects of 10-weeks of combined strength and endurance training on blood pressure following a 2000-m rowing race (Error bars = SD)

c- significantly different from 5-min (PRE)
d- significantly different from 45-min (PRE)
*- significantly different from all measurement times (PRE)
f- significantly different from IP (POST)
g- significantly different from 5-min (POST)
h- significantly different from 45-min (POST)
†- significantly different from all measurement times (POST)

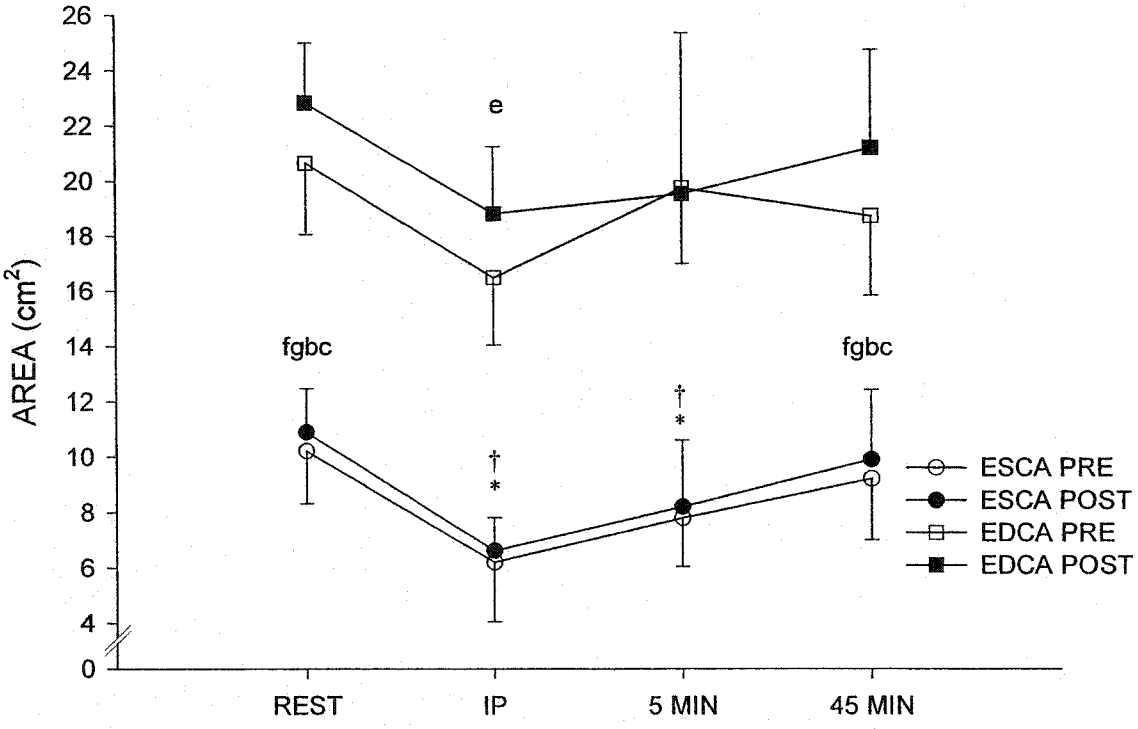

Figure 3-3. Effects of combined strength and endurance training on cavity cavity areas following a 2000-m rowing trial. (Error bars = SD)

b- significantly different from IP (PRE)
c- significantly different from 5-min (PRE)
*- significantly different from all measurement times (PRE)
e- significantly different from rest (POST)
f- significantly different from IP (POST)
g- significantly different from 5-min (POST)
†- significantly different from all measurement times (POST)

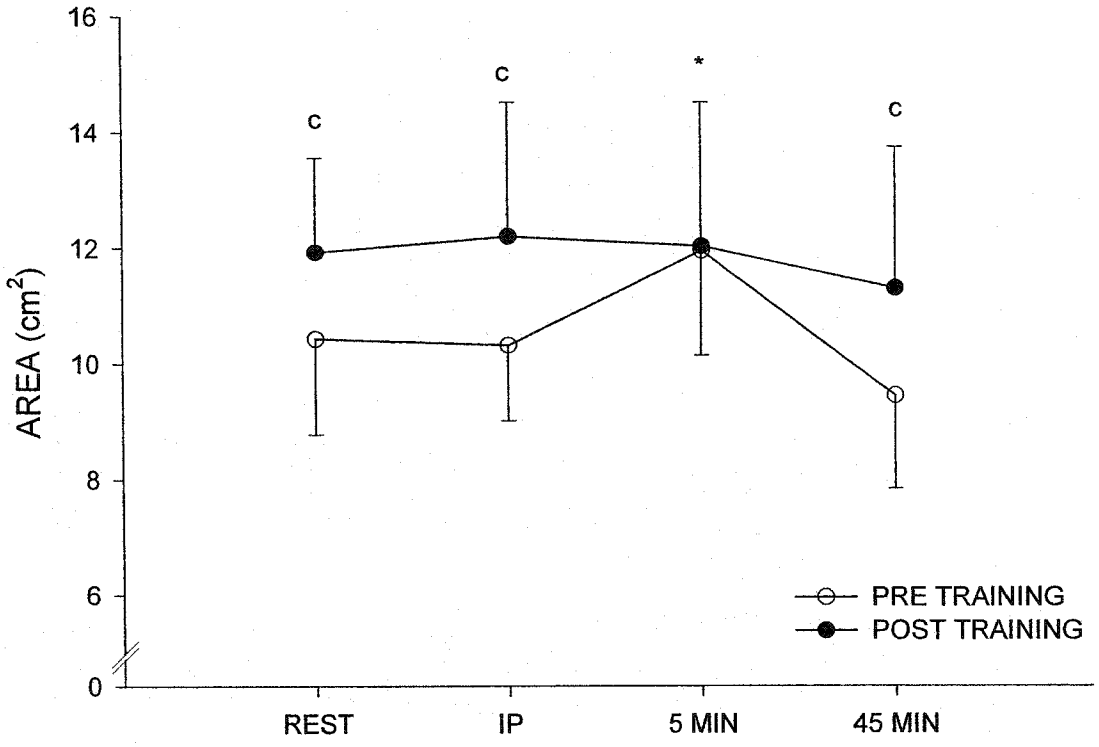

Figure 3-4. Effects of combined strength and endurance training on stroke area following a 2000-m rowing trial. (Error bars = SD)

c- significantly different from 5-min (PRE)
*- significantly different from all measurement times (PRE)

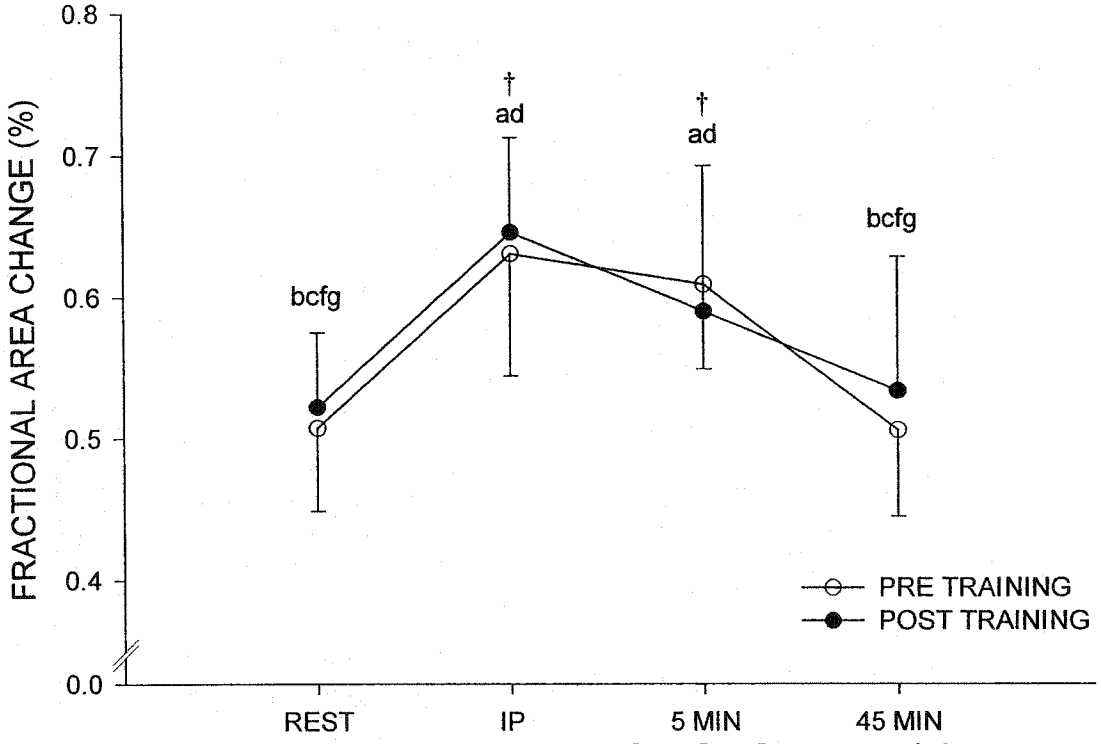

Figure 3-5. Effects of combined strength and endurance training on fractional area change following a 2000-m rowing trial. (Error bars = SD)

a- significantly different from rest (PRE)
b- significantly different from IP (PRE)
c- significantly different from 5-min (PRE)
d- significantly different from 45-min (PRE)
f- significantly different from IP (POST)
g- significantly different from 5-min (POST)
†- significantly different from all measurement times (POST)

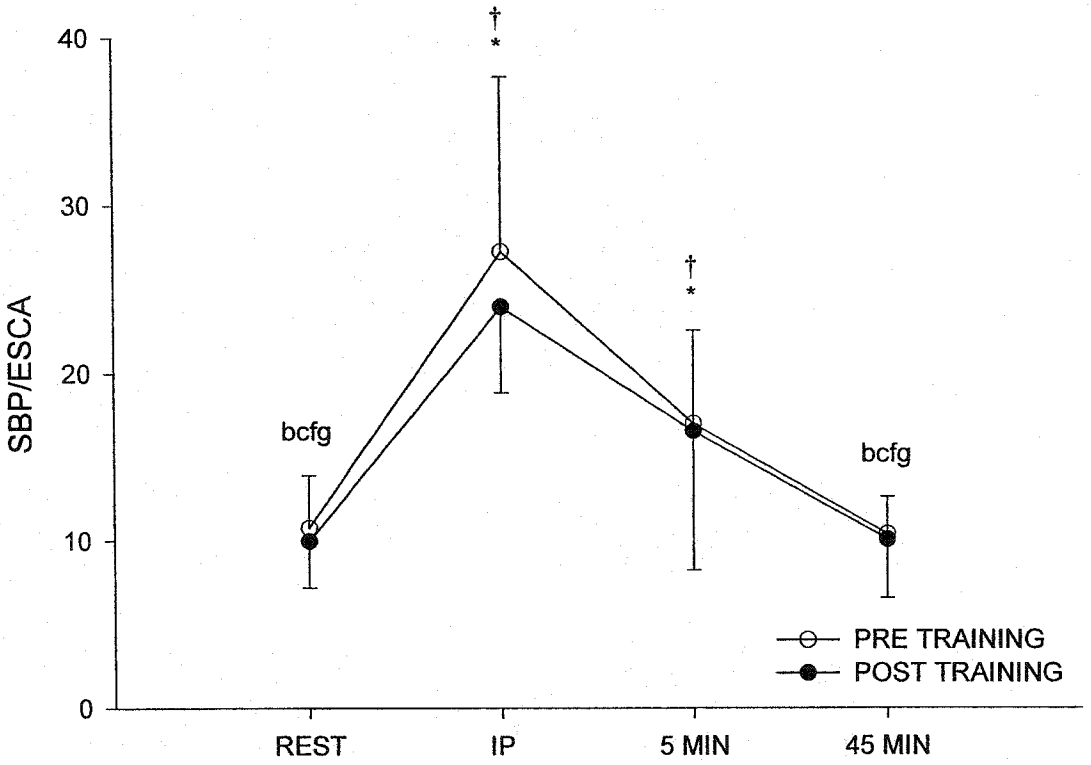

Figure 3-6. Effects of combined strength and endurance training on SBP/ESCA RATIO following a 2000-m rowing trial. (Error bars = SD)

b- significantly different from IP (PRE)
c- significantly different from 5-min (PRE)
*- significantly different from all measurement times (PRE)
f- significantly different from IP (POST)
g- significantly different from 5-min (POST)
†- significantly different from all measurement times (POST)

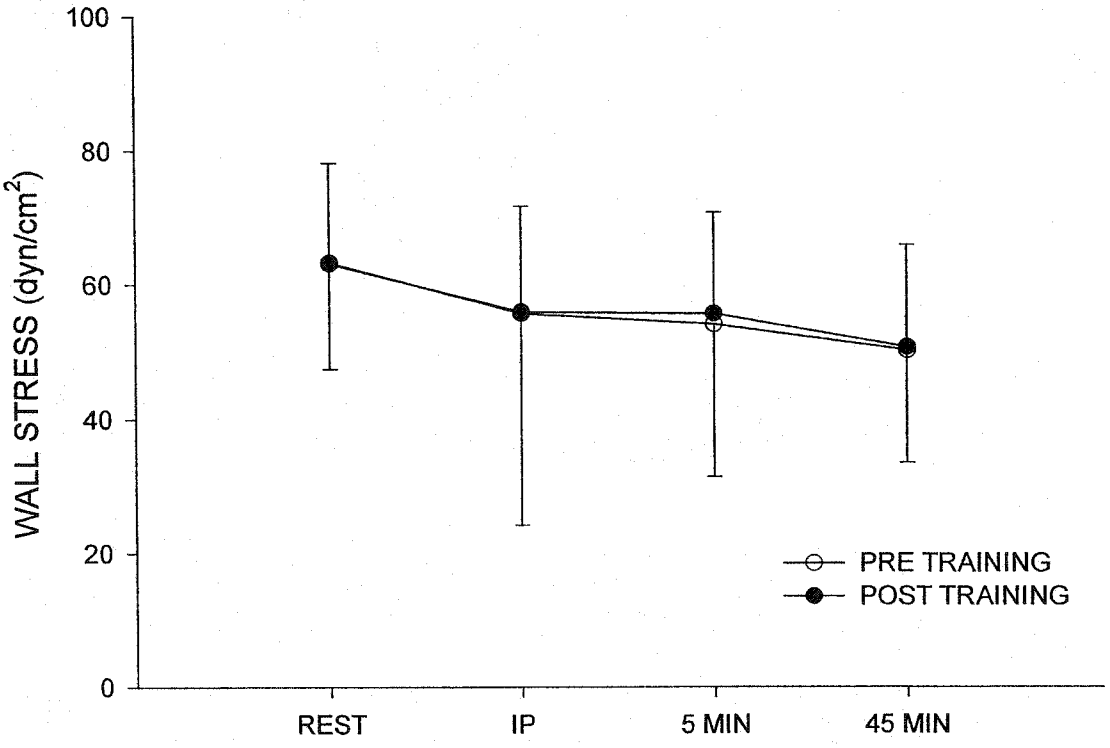

Figure 3-7. Effects of combined strength and endurance training on end-systolic wall stress following a 2000-m rowing trial. (Error bars = SD)

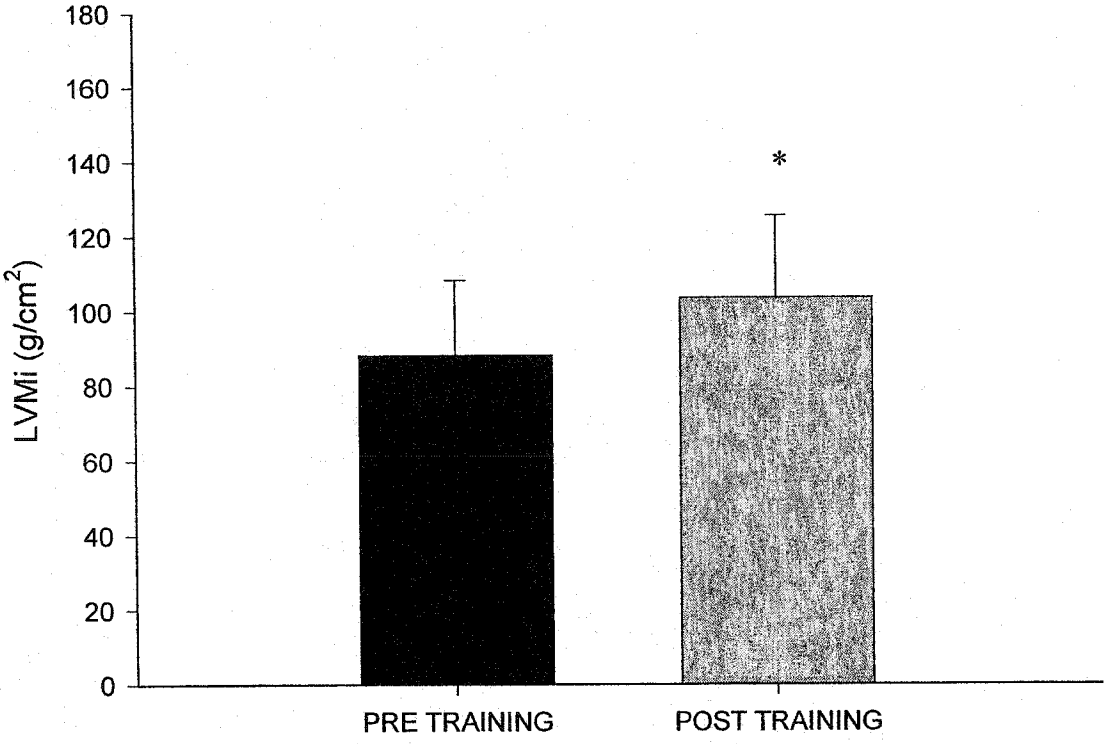

Figure 3-8. Effects of combined strength and endurance training on LVMi (Error bars = SD)
*- significantly different from PRE TRAINING

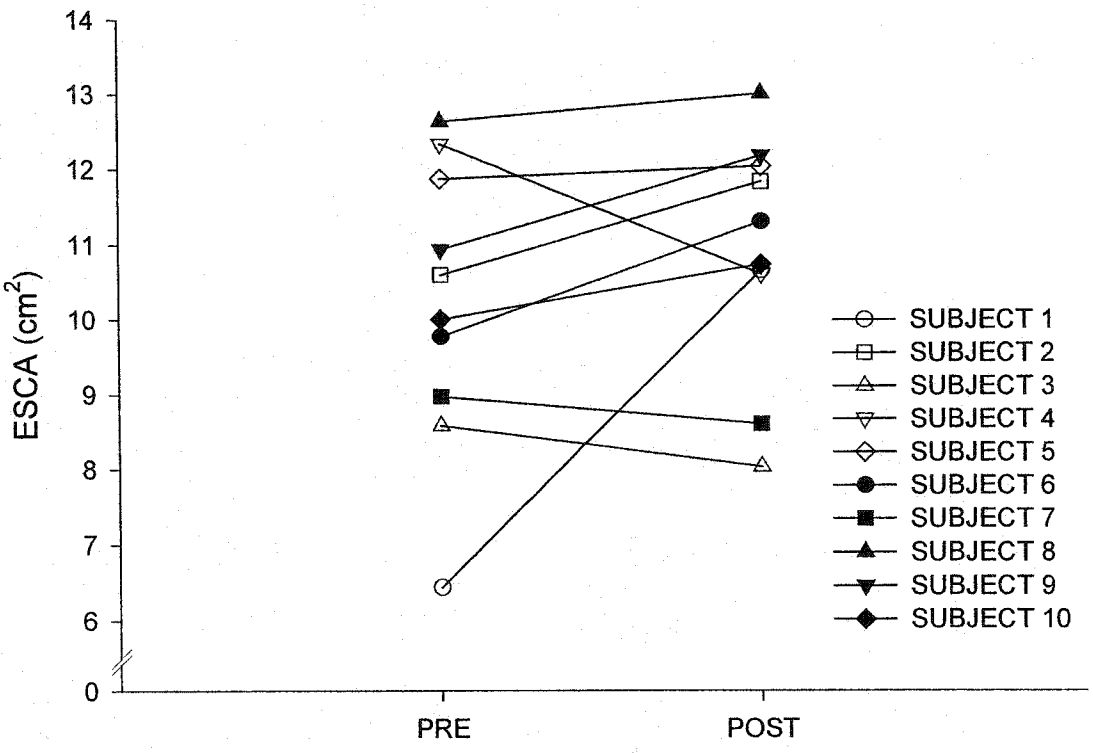

Figure 3-9. Effect of 10-weeks of combined strength and endurance training on ESCA at rest.

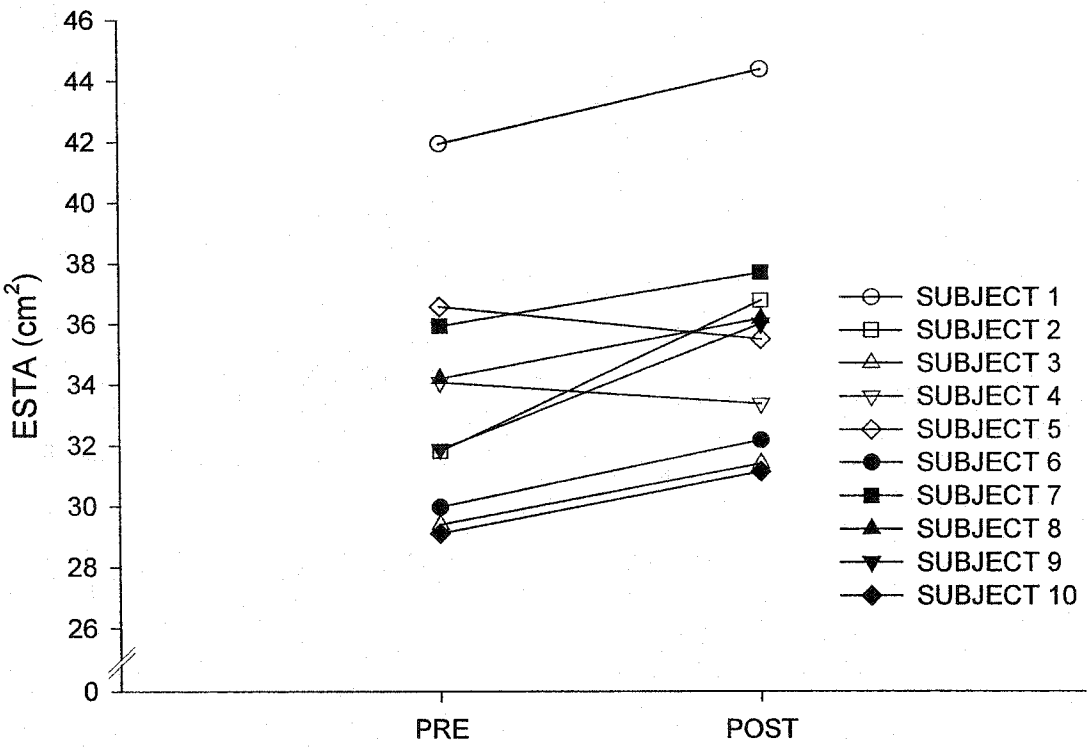

Figure 3-10. Effects of 10-weeks of combined strength and endurance training on ESTA at rest.

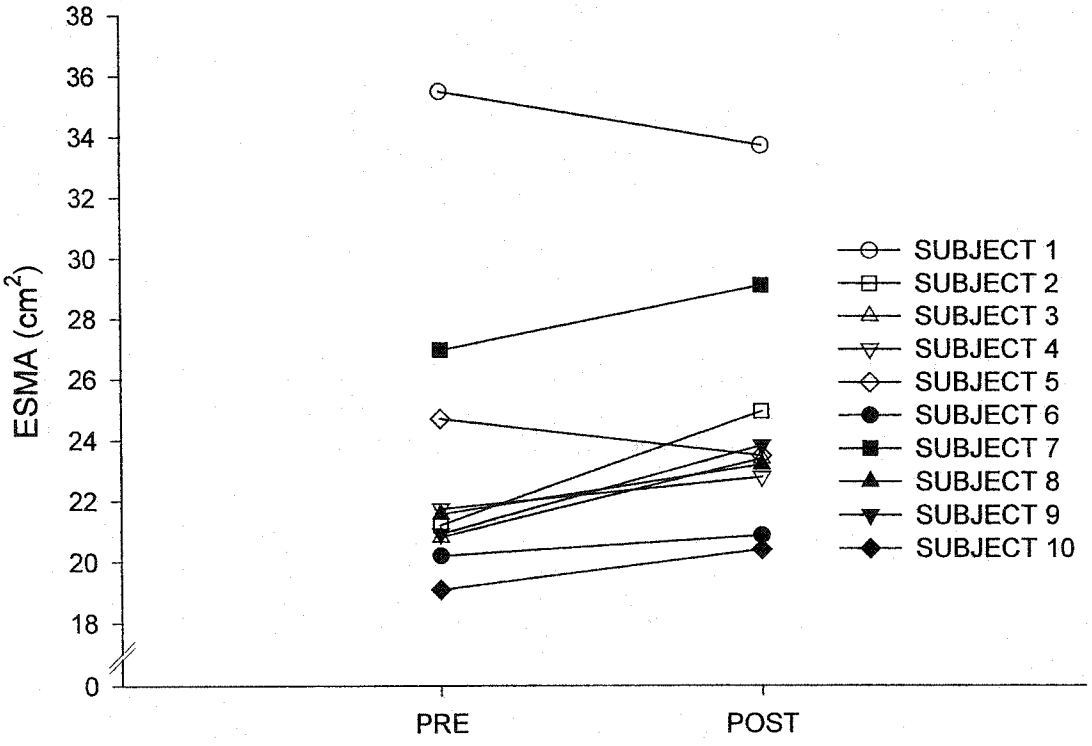

Figure 3-11. Effects of 10-weeks of combined strength and endurance training on ESMA at rest.

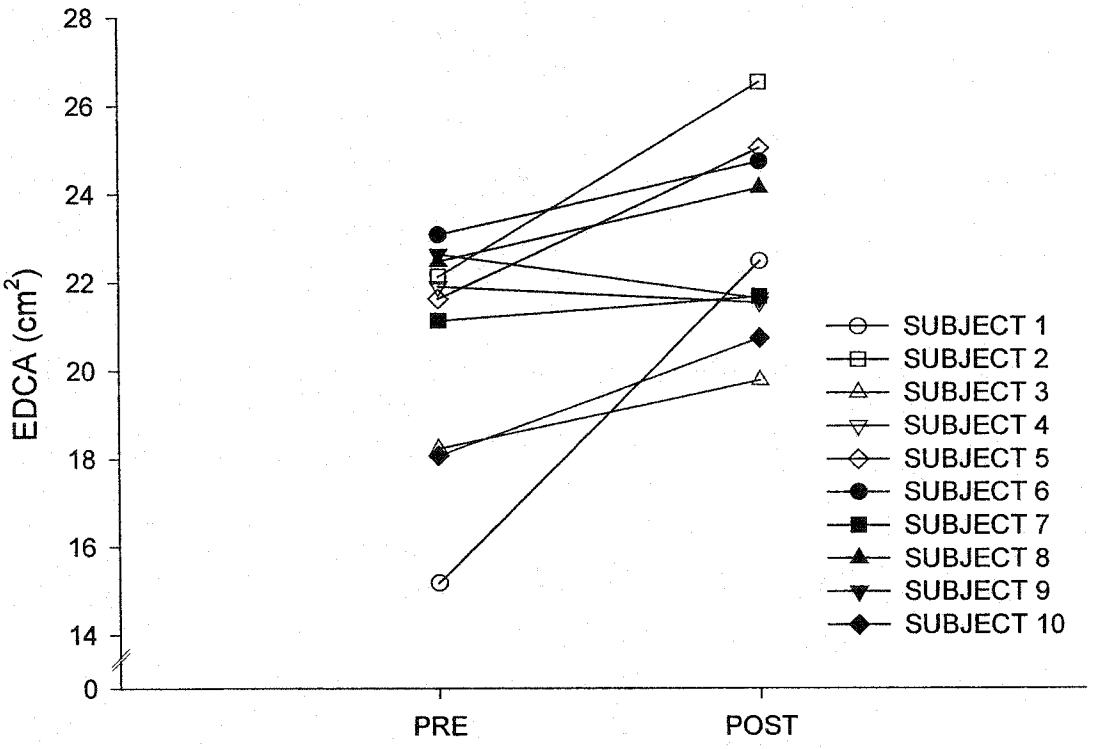

Figure 3-12. Effect of 10-weeks of combined strength and endurance training on EDCA at rest.

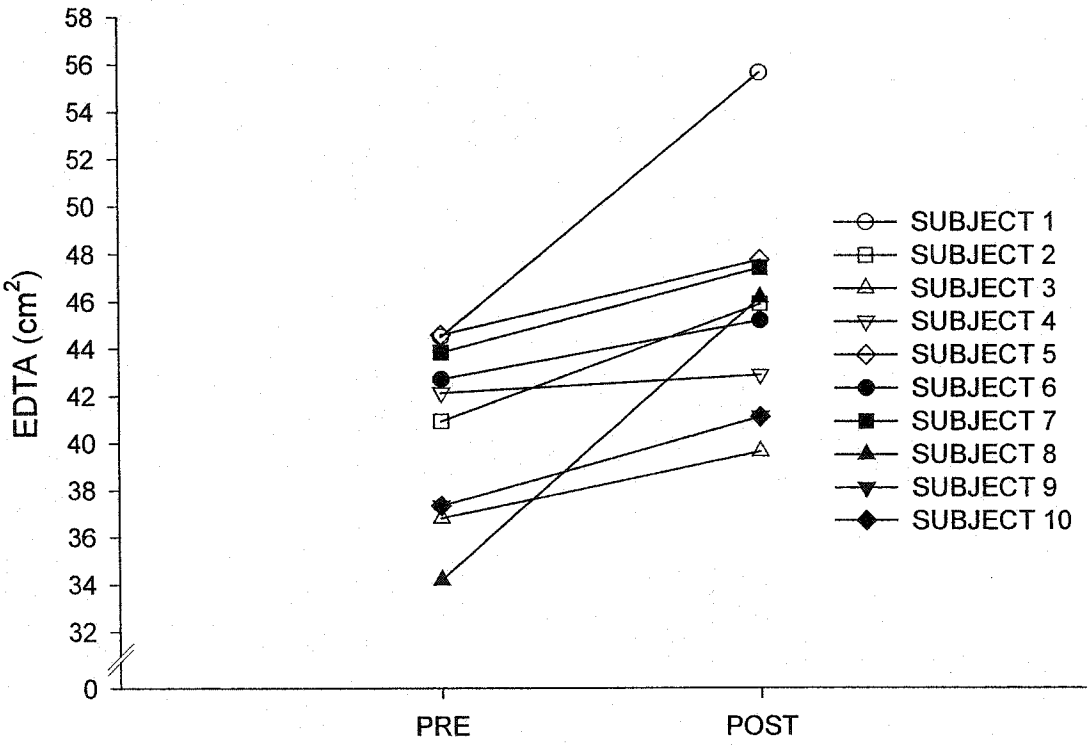

Figure 3-13. Effects of 10-weeks combined strength and endurance training on EDTA at rest.

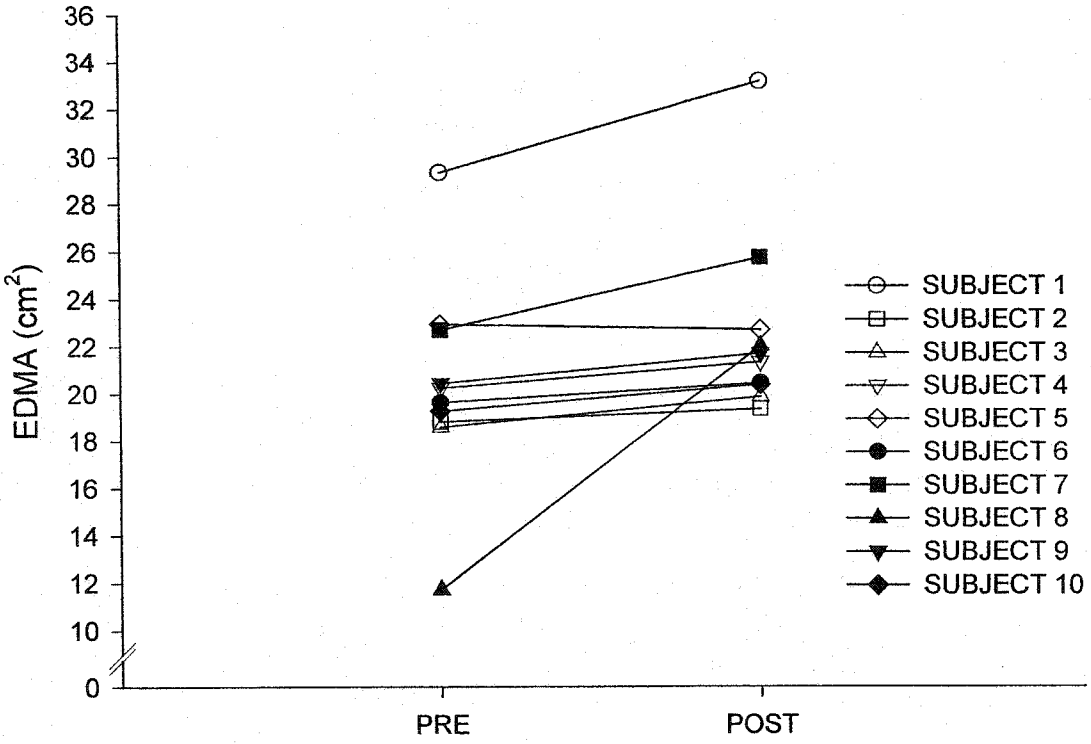

Figure 3-14. Effect of 10-weeks of combined strength and endurance training on EDMA at rest.

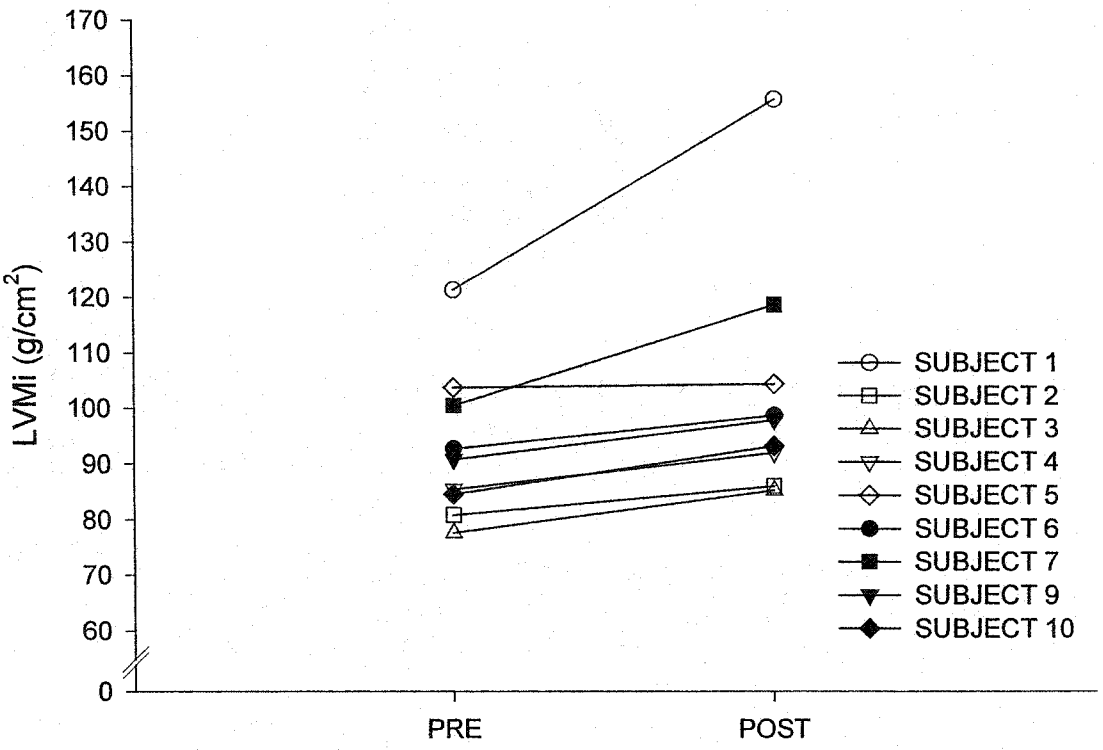

Figure 3-15. Effect of 10-weeks of combined strength and endurance training on LVMi at rest.

CHAPTER 4

GENERAL DISCUSSION AND CONCLUSIONS

4.1 Discussion

Previous research has shown that high blood lactate and low blood pH results in a decrease in contractile function of perfused hearts (Samaja et al., 1999). It has also been shown that rowers produce some of the highest reported blood lactate levels and lowest blood pH following a 2000-m rowing race (Neilson, 1999). It has also been shown that rowers possess some of the largest reported hearts (both LVM and wall thickness) and that these greater masses and wall thicknesses are a result of the combined strength and endurance training that are associated with rowing training (Pelliccia et al., 1991). With larger than average hearts, high blood lactate production and low pH levels following a 2000-m rowing trial, and the potential negative effects on LV contractile function by high blood lactate and low pH levels, rowers may provide an appropriate population for the investigation of high-intensity rowing exercise on LV systolic function.

High-intensity rowing exercise on LV systolic function immediately following and during recovery from a 2000-m rowing trial has not been previously investigated. It was hypothesized that a 2000-m rowing trial would have a negative effect on the LV systolic function immediately following and during recovery manifested as a decrease in the SBP/ESCA ratio that could be attributed to an increase in blood lactate and a decrease in blood pH. During recovery from the 2000-m rowing race there was a significant increase in blood lactate as compared to rest, however following the 2000-m trial there

was an increase in LV systolic function, shown by an improvement in SBP/ESCA relation and during recovery function returned to resting levels. The improvement in contractility (SBP/ESCA relation) may be due to a number of mechanisms. Although blood lactate levels increased significantly following the 2000-m trial, the levels obtained in this study are not as high as those seen by Neilson (1999). If high lactate and low pH play a role in decreasing contractility there may be a critical value that needs to be attained that is greater than that seen in this study. The increase in circulating catecholamines during exercise increases the contractile function of the LV and may have other protective mechanisms that outweigh the negative effects of high lactate and low pH.

The results of this investigation support the work of others suggesting that following high-intensity exercise, LV systolic function is improved and that this improvement could be attributed to an increase in contractility (Di Bello et al., 1996 and Goodman et al., 1991). These authors also suggest that at lower intensities improvements in LV systolic function could be a result of improvements in loading conditions (improvement in the Frank-Starling mechanism). However, there was no improvement in loading conditions immediately following the 2000-m trial. The lack of decrease in LV systolic function following a 2000-m rowing trial combined with an increase in blood lactate and a decrease in blood pH suggest the there may be some mechanism that protects the heart from the negative effects of increased blood lactate and decreased blood pH.

It was also hypothesized that 10-weeks of combined strength and endurance training would result in an increase in resting LV morphology. Following training there

was a significant increase in both LVM and LVMi at rest, however these were not as dramatic as those seen by Pelliccia et al. (1991). It has also been shown that the type of training that is followed can have an effect on the type of 'cardiac hypertrophy' that occurs. Training that employs a dynamic component results in an increase in LV cavity area, as well as an increase in LV wall thickness, which decreases wall stress (Maron, 1986). In this investigation there was an increase in cavity area combined with an increase in wall thickness at rest, suggesting eccentric hypertrophy. Therefore, 10-weeks of rowing training may provide the essential stimuli for increasing heart size.

Finally, the effects of the 10-week combined strength and endurance training on LV systolic function immediately following and during recovery from a 2000-m rowing race were investigated. It was hypothesized that the training program would improve LV systolic function, however there was no significant difference in any variable between the pre-training test and the post-training test. Although there was no change in any variable there was significant improvement in VO_{2max}, 2000-m time and leg-press strength, suggesting that the combined strength and endurance program improved performance. These improvements could be attributed to an increase in peripheral oxidative capacity and an improvement in resting SA, however peripheral changes following training and during recovery from the 2000-m trial and LV systolic function during exercise were not evaluated.

Following training there was a main effect for ESCA, EDCA, SA, and SBP/ESCA. There were significant increases in ESCA, EDCA, and SA suggesting a larger cavity area and improvements in filling. This can be attributed to the increased cavity area due to training and a greater reliance on the Frank-Starling mechanism

following training. There was also a decrease in the SBP/ESCA relation suggesting a decrease in contractility following training. This decrease in contractility was a result of increased reliance on the Frank-Starling mechanism as opposed to increases in blood lactate. Following training, improved LV function was a result of the LV became more reliant on the less energy demanding Frank-Starling mechanism as opposed to increases in contractility.

It appears that there were no negative effects of increased blood lactate and decreased pH on LV systolic function in this investigation contrary to the work of Samaja et al. (1999) and Berger et al. (1999). The use of human subjects in an exercise setting vs. isolated perfused hearts and skinned muscle fibres may be the major cause of these differences. Samaja et al (1999) were able to use a controlled solution to perfuse the hearts; while in this investigation the complexity of circulating blood (plasma, increased hormone levels, increased catecholamines) may attenuate any negative effect of blood lactate generated during exercise.

4.2 Limitations

The use of resting and recovery echocardiographic data limits the ability to discuss exercise responses. It has been shown previously that recovery measurements do not equal those obtained during exercise although heart rate and blood pressure were still elevated (Pokan et al., 2000). There also exists a measurement error with echocardiography (Perrualt and Turcotte, 1994), however this was minimized as the same technician obtained all images both pre-training and post-training and an experienced cardiologist performed all analysis. A final limitation of this investigation was the use of blood lactate to estimate acidosis. Despite an established relationship between lactate

and H^+ production; blood lactate accumulation is not the most valid indication of pH levels.

Future investigations should evaluate changes in LV systolic function during exercise as opposed to immediately following exercise and during recovery. Focus should also be placed on determining the mechanisms that prevent high levels of blood lactate and low blood pH to have a negative effect on LV systolic function.

This investigation showed an improvement in LV systolic function immediately following and during recovery from a 2000-m rowing race despite an increase in blood lactate. It was also shown that 10-weeks of combined strength and endurance training increased resting LV morphology (increased LVM and LVMi). Finally, 10-weeks of combined training did not improve LV systolic function immediately following and during recovery from a 2000-m rowing trial despite an improvement in performance.

4.3 References

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APPENDIX 1- VALIDITY AND RELIABILITY OF ACCUSPORT LACTATE ANALYZER

Known Concentration	Read Concentration
20.0 mmol·L^{-1}	20.3 mmol·L^{-1}
10.0 mmol·L^{-1}	10.3 mmol·L^{-1}
5.0 mmol·L^{-1}	4.9 mmol·L^{-1}
2.5 mmol·L^{-1}	2.7 mmol·L^{-1}
1.25 mmol·L^{-1}	1.0 mmol·L^{-1}

Ear lobe	1.1 mmol·L^{-1}
Finger tip	2.8 mmol·L^{-1}

APPENDIX 2- AEROBIC TRAINING PROGRAM

	Week 1-3	Week 4-6	Week 6-8	Week 8-10
Monday	**WU:1000m** WO: 5000m cont. CD: 1000m SR: 22-28 **Total: 7000m**	WU:1000m WO: 6000m cont. CD: 1000m SR: 22-28 **Total: 8000m**	WU:1000m WO: 7000m cont. CD: 1000m SR: 22-28 **Total: 9000m**	WU:1000m WO: 8000m cont. CD: 1000m SR: 22-28 **Total: 10000m**
Wednesday	WU:1000m WO: 2x 2500m cont. CD: 1000m SR: 22-28 **Total: 7000m**	WU:1000m WO: 2x 3000m cont. CD: 1000m SR: 22-28 **Total: 8000m**	WU:1000m WO: 3500m cont. 3000m cont. CD: 1000m SR: 22-28 **Total: 8500m**	WU:1000m WO: 4000m cont. 3000m cont. CD: 1000m SR: 22-28 **Total: 9000m**
Friday	WU:1000m WO: 4x 500m CD: 1000m SR: 28-34 **Total: 6000m**	WU:1000m WO: 5x 500m CD: 1000m SR: 28-34 **Total: 7000m**	WU:1000m WO: 3 sets of: 1x 250 1x 500 1x 250 CD: 1000m SR: 28-34 **Total: 8000m**	WU:1000m WO: 6x 500m 2x 250m CD: 1000m SR: 28-34 **Total: 9000m**

APPENDIX 3- STRENGTH TRAINING PROGRAM

Schedule 1- Bench Press an Leg Press							
Day	**Set 1**	**Set 2**	**Set 3**	**Set 4**	**Set 5**	**Set 6**	**Total Reps/ Average intensity**
Week 1 and 2							
Tuesday	6 x 65%	6 x 70%	6 x 75%	6 x 80%	6x 85%		30/75.0%
Thursday	8 x 65%	8 x 70%	8 x 75%	8 x 80%			32/72.5%
Saturday	8 x 65%	8 x 70%	8 x 75%	8 x 80%			32/72.5%
Week 3 and 4							
Tuesday	10 x 65%	6 x 80%	6 x 82%	4 x 84%	4 x 88%		30/79.8%
Thursday	10 x 65%	6 x 80%	6 x 82%	4 x 84%	4 x 88%		30/79.8%
Saturday	8 x 65%	8 x 70%	8 x 75%	8 x 80%			32/72.5%
Week 5							
Tuesday	10 x 65%	6 x 80%	6 x 82%	4 x 84%	4 x 88%		30/79.8%
Thursday	10 x 65%	6 x 80%	6 x 82%	4 x 84%	4 x 88%		30/79.8%
Saturday	10 x 80%	10 x 80%	10 x 80%				30/80.0%
Week 6							
Tuesday	10 x 65%	6 x 80%	6 x 82%	4 x 84%	4 x 88%		30/79.8%
Thursday	10 x 65%	6 x 80%	6 x 82%	4 x 84%	4 x 88%		30/79.8%
Saturday	10 x 65%	6 x 80%	6 x 80%	6 x 80%			28/76.3%
Week 7							
Tuesday	10 x 55%	8 x 65%	6 x 72%	4 x 82%	3 x 88%	2 x 92%	33/75.8%
Thursday	10 x 55%	8 x 65%	6 x 72%	4 x 82%	3 x 88%	2 x 92%	33/75.8%
Saturday	10 x 65%	8 x 80%	6 x 80%	6 x 80%			30/76.3%
Week 8							
Tuesday	10 x 55%	8 x 65%	6 x 72%	4 x 82%	3 x 88%	2 x 92%	33/75.8%
Thursday	10 x 55%	8 x 65%	6 x 72%	4 x 82%	3 x 88%	2 x 92%	33/75.8%
Saturday	10 x 60%	8 x 70%	6 x 80%	4 x 85%			28/73.8%
Week 9							
Tuesday	6 x 75%	6 x 75%	6 x 75%				18/75.0%
Thursday	6 x 75%	6 x 75%	6 x 75%				18/75.0%
Saturday	10 x 60%	8 x 70%	6 x 80%	4 x 85%			28/73.8%
Week 10							
Tuesday	6 x 65%	6 x 70%	6 x 75%	6 x 85%			30/75.0%
Thursday	6 x 65%	6 x 70%	6 x 75%	6 x 85%			30/75.0%
Saturday	8 x 65%	8 x 70%	8 x 75%	6 x 80%			30/72.5%

Schedule 2- Lateral Pull Down and Seated Row						
Day	**Set 1**	**Set 2**	**Set 3**	**Set 4**	**Set 5**	**Total Reps/ Average intensity**
Week 1 and 2						
Tuesday	6 x 65%	6 x 70%	6 x 75%	6 x 80%	6 x 85%	30/75.0%
Thursday	8 x 65%	8 x 70%	8 x 75%	8 x 80%		32/72.5%
Saturday	10 x 65%	10 x 70%	10 x 75%			30/70.0%
Week 3 and 4						
Tuesday	6 x 65%	6 x 70%	6 x 75%	6 x 80%	6 x 85%	30/75.0%
Thursday	8 x 65%	8 x 70%	8 x 75%	8 x 80%		32/72.5%
Saturday	10 x 80%	10 x 80%	10 x 80%			30/80.0%
Week 5						
Tuesday	6 x 65%	6 x 70%	6 x 75%	6 x 80%	6 x 85%	30/75.0%
Thursday	10 x 60%	8 x 70%	6 x 80%	4 x 85%		28/75.3%
Saturday	8 x 65%	8 x 70%	8 x 75%	8 x 80%		32/72.5%
Week 6						
Tuesday	10 x 60%	8 x 70%	6 x 89%	4 x 85%		28/73.8%
Thursday	6 x 65%	6 x 70%	6 x 75%	6 x 80%	6 x 85%	30/75.0%
Saturday	8 x 65%	8 x 70%	8 x 75%	8 x 80%		32/72.5%
Week 7						
Tuesday	8 x 70%	8 x 70%	8 x 70%			32/70.0%
Thursday	8 x 78%	8 x 78%	8 x 78%			32/78.0%
Saturday	10 x 65%	6 x 80%	6 x 80%	6 x 80 %		28/76.3%
Week 8						
Tuesday	10 x 65%	6 x 80%	6 x 80%	6 x 80 %		28/76.3%
Thursday	10 x 65%	6 x 80%	6 x 80%	6 x 80 %		28/76.3%
Saturday	10 x 65%	6 x 80%	6 x 80%	6 x 80 %		28/76.3%
Week 9 and 10						
Tuesday	10 x 65%	6 x 82%	6 x 82%	6 x 88 %		28/79.3%
Thursday	10 x 65%	6 x 80%	6 x 80%	6 x 80 %		28/76.3%
Saturday	10 x 65%	6 x 75%	6 x 75%			28/71.7%

Schedule 3- Leg extention and Leg Curl						
Day	**Set 1**	**Set 2**	**Set 3**	**Set 4**	**Set 5**	**Total Reps/ Average intensity**
Week 1 and 2						
Tuesday	12 x 66%	10 x 75%	8 x 80%			30/73.7%
Thursday	10 x 70%	8 x 80%	6 x 85%			24/78.3%
Saturday	10 x 70%	10 x 75%				20/72.5%
Week 3 and 4						
Tuesday	10 x 70%	8 x 80%	6 x 85%			24/78.3%
Thursday	12 x 66%	10 x 75%	8 x 80%			30/73.7%
Saturday	8 x 77%	8 x 80%				16/78.5%
Week 5						
Tuesday	8 x 85%	8 x 85%	8 x 85%			24/85.0%
Thursday	8 x 70%	8 x 70%	8 x 70%			24/70.0%
Saturday	12 x 66%	12 x 70%				24/68.0%
Week 6						
Tuesday	5 x 77%	4 x 88%	3 x 88%			12/84.3%
Thursday	5 x 77%	4 x 88%	3 x 88%			12/84.3%
Saturday	10 x 70%	8 x 80%	6 x 85%			24/78.3%
Week 7 and 8						
Tuesday	8 x 70%	6 x 72%	4 x 80%			18/74.0%
Thursday	8 x 70%	6 x 72%	4 x 80%			18/74.0%
Saturday	8 x 70%	6 x 72%	4 x 80%			18/74.0%
Week 9 and 10						
Tuesday	6 x 80%	6 x 90%	6 x 90%			18/86.7%
Thursday	6 x 80%	6 x 90%	6 x 90%			18/86.7%
Saturday	8 x 80%	6 x 90%	4 x 95%			20/86.7%

Schedule 4- Upright Row, Calf Raise, Biceps Curl, Triceps Extension						
Day	**Set 1**	**Set 2**	**Set 3**	**Set 4**	**Set 5**	**Total Reps/ Average intensity**
Week 1 and 2						
Tuesday	10 x 70%	8 x 80%	6 x 85%			24/78.3%
Thursday	10 x 70%	8 x 80%	6 x 85%			24/78.3%
Saturday	10 x 65%	8 x 70%	6 x 75%			24/70.0%
Week 3 and 4						
Tuesday	10 x 65%	8 x 70%	8 x 70%			26/68.3%
Thursday	10 x 65%	6 x 78%	6 x 80%	6 x 82%		28/76.3%
Saturday	10 x 70%	6 x 82%				16/76.0%
Week 5 and 6						
Tuesday	8 x 65%	8 x 70%	8 x 75%	8 x 80%		32/72.5%
Thursday	10 x 70%	8 x 80%	6 x 85%			24/78.3%
Saturday	12 x 66%	10 x 75%	8 x 80%			30/73.7%
Week 7 and 8						
Tuesday	10 x 70%	8 x 75%	8 x 75%			26/73.3%
Thursday	10 x 70%	8 x 75%	8 x 75%			26/73.3%
Saturday	8 x 78%	8 x 78%	8 x 78%			24/78.0%
Week 9 and 10						
Tuesday	10 x 65%	6 x 78%	6 x 80%	6 x 82%		28/76.3%
Thursday	10 x 65%	6 x 78%	6 x 80%	6 x 82%		28/76.3%
Saturday	10 x 65%	10 x 70%	10 x 75%			30/70.0%