ACUTE CARDIOVASCULAR RESPONSE TO MULTIPLE WINGATE EXERCISE

THE ACUTE CARDIOVASCULAR RESPONSE TO MULTIPLE WINGATE EXERCISE IN HEALTHY MALES

By

LINDSAY J. GURR, H.B.KIN

A Thesis Submitted to the School of Graduate Studies in Partial Fulfillment of the Requirements for the Degree of Masters of Science

McMaster University

MASTER OF SCIENCE (2007) McMaster University

Kinesiology Hamilton, Ontario

TITLE: The Acute Cardiovascular Response to Multiple Wingate Exercise in Healthy Males

AUTHOR: Lindsay June Gurr, H. B. Kin (McMaster University)

SUPERVISOR: Professor Maureen Jane MacDonald

NUMBER OF PAGES: ix, 104

ABSTRACT

The Wingate anaerobic test can be used in an exercise training program as a powerful training stimulus for producing metabolic and performance enhancements. Although the acute cardiovascular responses in terms of heart rate (HR), blood pressure (BP), stroke volume (SV), cardiac output (CO), and leg blood flow following a single Wingate have been characterized, the acute cardiovascular recovery pattern in response to multiple Wingate exercise bouts performed in an interval pattern have yet to be described. The purpose of the current investigation was to characterize that acute cardiovascular recovery period following multiple Wingate exercise. We observed the recovery patterns of HR, BP, SV, CO, and leg blood flow for 120 minutes immediately following multiple Wingate exercise. Ten recreationally active males aged 19.8 ± 1.2 years (mean ± SD) years performed a single bout of Wingate exercise, and a session of multiple Wingate exercise in random order, on separate days. Cardiovascular measurements were conducted at rest and after two-minutes of recovery and then continued at 15-minute intervals until 120 minutes of recovery. HR was elevated immediately after exercise compared to rest, and declined towards resting values for the remainder of recovery, although HR failed to return to resting values after 120 minutes of recovery. SV was significantly decreased, compared to rest, immediately following exercise from 87.3 ± 5.7 to 60.0 ± 5.6 ml. CO was increased compared to rest at two-minutes following multiple Wingate exercise, and continued to increase to a maximum

recovery value of 8.1 ± 0.7 L/min at R15. Immediately following exercise systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were all elevated to 155 ± 3, 73 ± 2, and 100 ± 3 mmHg respectively. Although SBP and MAP returned to resting values 15-minutes after exercise, DBP continued to decrease, resulting in a period of hypotension observed from R15 and R30. Leg blood flow was elevated compared to rest following exercise. Common femoral artery (CFA) blood flow was higher after a single Wingate, than after multiple Wingate exercise (1264 ± 109 and 1036 ± 86 ml/min respectively). Superficial femoral artery (SFA) blood flow (616 ± 55 ml/min) immediately following multiple Wingate exercise was not different following a single Wingate, however the time to return to resting values was longer after multiple Wingate exercise. We attribute the sustained elevations in (SFA) blood flow to increased cutaneous flow for thermoregulation purposes following multiple Wingate exercise.

The results of this investigation indicate that the general cardiovascular recovery time is longer after multiple Wingate exercise, than after a single Wingate bout. This type of supramaximal exercise temporarily reduces SV due to elevated SBP and decreases in cardiac preload that are not facilitated by vasodilatation and decreased total peripheral resistance (TPR).

ACKNOWLEDGMENTS

I would like to express my thanks for the assistance given by my supervisor Maureen MacDonald. I would also like to thank the other members of my committee; Martin Gibala, thank you for introducing Wingate exercise to the EMRG lab, and volunteering your time so generously to be on this committee. Neil McCartney, thank you for always providing me with your most forthright opinions and comments. I appreciate that you have always made time in your schedule to listen to the progress I have made, and allowed me access to your extensive cardiovascular knowledge. I would not be at this stage if it were not for your help through this process.

I would like to thank the members of the EMRG lab, in particular Dan West for always volunteering to be a guinea pig during the set up phase of the study. You are a talented group of researchers, and I wish you the best in all of your future endeavors.

Mark Rakobowchuk, Phil Millar, Melanie Stuckey, and Tracy Rerecich, your help was critical for the collection of this data, and the time you so generously put into the project will not be forgotten. I would also like to thank all of my participants who so generously donated their time to this study.

Finally, and most importantly, I would like to express my genuine gratitude towards my family, and my parents. The knowledge of your unequivocal and limitless love, support, and confidence in me is something that I take comfort in every day. I know that you will support me in all of my endeavors, and thank you for supporting me throughout this degree.

TABLE OF CONTENTS

PREFACE

The following is a list of abbreviations and operational definitions used throughout this manuscript:

ARD – aortic root diameter

BP – blood pressure

BF – blood flow

BV – blood velocity

CFA – common femoral artery

CO – cardiac output

DBP – diastolic blood pressure

HR – heart rate

HRV – heart rate variability

MAP – mean arterial pressure

MHz – megahertz

PP – pulse pressure

PEH – post exercise hypotension

SBP – systolic blood pressure

SFA – superficial femoral artery

SD – standard deviation

SEM – standard error of the mean

SV – stroke volume

TPR – total peripheral resistance

TVI – time-velocity integral

VO_{2max} – maximal oxygen consumption

VO_{2peak} – peak oxygen consumption

1.0 LITERATURE REVIEW

1.1 Exercise and Cardiovascular Health

It is well established that the human body responds to regular physical exercise by adapting the muscular (Clarke, 1973; Ikai & Fukunaga, 1970), respiratory (Woolf & Suero, 1969), and cardiovascular system (Seals & Hagberg, 1984; Stratton et al., 1994) to compensate for the increased demands. Regular exercise can also attenuate the decline in cardiovascular fitness inherent to the aging process and help to sustain physical health (Bortz, 1982). A decrease in exercise endurance is linked with a sedentary lifestyle and an increased risk of cardiovascular disease (Abbott et al., 1989). The Framingham study, which involved comprehensive longitudinal analysis, indicated that active women and men tend to live longer lives than their more sedentary peers (Sherman et al., 1994). Some of the positive cardiovascular changes attributed to exercise training are achieved though improved autonomic control of the heart and cardiovascular system, by increasing parasympathetic activity and decreasing sympathetic activity at rest (Carter, Banister, & Balber, 2003).

1.2 Endurance exercise

Endurance exercise is defined as "activity of at least 20 minutes duration in which heart rate is elevated to 60-80% of maximum" (Carter, Banister, & Balber, 2003). The

acute cardiovascular effects of endurance exercise have been examined using a wide range of exercise modalities, intensities, and durations. At the onset of dynamic exercise, central drive from the brain above the medullary centers and feedback from mechanoreceptors in working limbs, cause a withdrawal of parasympathetic tone, and an increase in sympathetic activity (Schibye et al., 1981). These changes in autonomic balance increase heart rate (HR) and myocardial contractility, as well as stimulate whole body vasoconstriction (MacDonald, 2002). Increased systemic vasoconstriction and myocardial contractility act together to increase blood pressure (BP), and preload to the heart; thereby increasing stroke volume (SV) and cardiac output (CO) to match the demands of the working muscles (Baskurt & Meiselman, 2003). As exercise continues, metabolite end products begin to accumulate and the demand for oxygen increases in the working limbs, causing vasodilation and increased perfusion of the working muscles (Baskurt & Meiselman, 2003; Rotstein, Bar-Or, & Dlin, 1982). The rates of metabolite build up, flow requirements, and autonomic adaptation differ according to many factors including the nature, intensity, and duration of activity, and the age, gender, and fitness level of the subject.

Regular physical exercise also acts as a preventative mechanism to reduce the risk of sudden death caused by physical exertion (Albert et al., 2000). Endurance exercise is a highly beneficial method to improve arterial health and autonomic control of the body (Adams et al., 2006; Mourot et al., 2005), an effective therapy for hypertension (Bond et al., 2002; Petrella, 1998), and a safe way to maintain or increase cardiovascular health

and conditioning in healthy and diseased populations (Meyer, 1997; Mourot et al., 2005; Bond et al., 2002; MacDonald, 2002).

1.2.1 Endurance Training

Classically aerobic endurance training has been linked to improvements to VO_{2max} (Hickson, Bomze, & Holloszy, 1977), strength and muscle mass (Timson et al., 1985). Previous studies have indicated that six to twelve weeks of endurance exercise preformed at as little of 50% of VO_{2max} intensity for 30 minutes a day (3 times per week) is effective in increasing VO_{2max} by 5-10% (Blomqvist & Saltin, 1983; Saltin & Rowell, 1980). In many incidences, aerobic improvements are realized through increases in SV (Saltin et al., 1968) and decreases in HR at submaximal exercise intensities (Carter, Banister, & Balber, 2003). Endurance trained athletes typically exhibit a lower HR at rest, and submaximal exercise intensities than untrained individuals. This decrease in resting and submaximal exercise HR is attributed to decreased sympathetic activity, and stronger parasympathetic tone (Carter, Banister, & Balber, 2003). Increases in muscular strength and performance have also been linked to improvements in the oxygen delivery to exercising muscle (Green et al., 1987). Increasing the intensity of endurance exercise yields greater improvements in exercise capacity and cardiovascular conditioning (Laursen & Jenkins, 2002; Oberman et al., 1995).

1.3 Interval exercise

It is difficult to maintain high intensity exercise for a prolonged period, so lower intensity "recovery" periods are interspersed with high intensity intervals in an interval exercise training program. The intention of interval exercise is to overload the physiological regulatory systems by repeating higher intensity bouts without permitting excessive recovery (Green et al., 2006). Åstrand *et al.,* (1960) found that intermittent exercise at the same workload as continuous exercise allows for significantly less lactate production, and less overall oxygen debt. When low intensity exercise intervals separate the higher intensity exercise intervals in intermittent training, it allows participants to exercise for a longer duration before experiencing fatigue (Christensen, Hedman, & Saltin, 1960). Together, the advantages of interval training allow individuals to exercise for a longer duration, at a much higher intensity than they would be able to sustain with continuous exercise.

Adams *et al.,* (2006), found that patients were able to tolerate and benefit from interval exercise that consisted of six intervals where patients walked to their maximum pain threshold, separated by three minutes of rest. Similarly, Warburton *et al.,* (2005) showed that 30 minutes of intense interval exercise consisting of two minute intervals at 90% of heart rate reserve (separated by two minutes at 40% of heart rate reserve) was safe for men with cardiovascular disease who had previously undergone bypass surgery or angioplasty. These results indicate that high intensity interval exercise causes less stress to the cardiovascular system due to their relatively short duration and multiple

recovery periods, indicating that this activity is safe for all participants, including those with cardiovascular problems.

1.3.1 Interval Training

Highly trained athletes may not be able to continue to improve their performance by simply increasing the volume of endurance exercise they are performing (Costill et al., 1988), so for these athletes interval training may provide an alternative and beneficial training stimulus. Four weeks of interval training on a treadmill performed at maximum running speed for 60-75% of the time to exhaustion has been shown to improve the performance of elite middle distance track athletes (Smith, McNaughton, & Marshall, 1999). Not only is aerobic interval training a strong stimulus for improving VO_{2max}, but it has been observed that more intense intervals yield greater improvements in aerobic fitness (Helgerud et al., 2007). In sedentary and recreationally active individuals six weeks of interval training (eight intervals of 20-seconds at 170% maximum power interspersed with 10 second rest intervals) improves endurance performance more than continuous submaximal training (60 minutes at 70% VO_{2max}) when training exercises were performed five times per day (Tabata et al., 1996). It is confirmed that the body is able to respond to a single supramaximal stimulus with higher sympathetic activation after training at higher intensities (Jacob et al., 2004). Given the wide variety of activities that require intense short burst of power, increased sympathetic activation capability is a very important training adaptation.

Following seven weeks of high intensity intermittent kicking exercise with one leg, Krustrup, Hellsten, & Bangsbo, (2004) found that submaximal blood flow and

oxygen extraction were elevated at high, but not at low submaximal intensities, compared to the untrained leg. The authors attributed these differences to metabolic adaptations within the muscle fibers and enhanced capillarization in the trained limb (Krustrup, Hellsten, & Bangsbo, 2004). Various high intensity interval training programs have been shown to not only increase both active muscle oxidative and glycolytic enzyme activity, but also provide improvements in aerobic metabolism (Burgomaster et al., 2005; Gibala et al., 2006; Laursen & Jenkins, 2002; MacDougall et al., 1998; Parra et al., 2000).

High intensity interval training has been introduced to many hospital programs, as an effective and safe way for individuals with peripheral arterial disease, and a variety of other cardiovascular conditions to exercise. Warburton *et al.,* (2005) studied the training effects of traditional endurance exercise versus high intensity interval exercise in 14 men with cardiovascular disease who had previously underwent bypass surgery or angioplasty. The traditional endurance training consisted of 30 minutes of exercise at 65% of heart rate reserve, while the high intensity interval training consisted of alternating two minute intervals at 90% and 40% of heart rate reserve for 30 minutes of total exercise time. The authors found that although both groups improved their performance levels, the high intensity interval training group had increased their time to exhaustion at 90% of heart rate reserve, and anaerobic threshold significantly more than the traditional endurance group. Similar results have been found in various other investigations that have studied populations with compromised cardiac function (Adams et al., 2006; Meyer, Kindermann, & Kindermann, 2004; Warburton et al., 2005).

1.4 The Wingate Anaerobic Test

The Wingate Anaerobic Test was developed to evaluate anaerobic cycling performance using a 30 second bout of all out exercise on a cycle ergometer (Ayalon et al., 1974). Typically, subjects are asked to cycle as fast as possible while 0.075kg/kg body weight of resistance is applied. During the 30 second cycling interval, it is estimated that approximately 80% of energy is derived from anaerobic alactic and lactic acid metabolism (Beneke et al., 2002). The idea behind this particular form of exercise is to push the body beyond the maximal level, as measured by an incremental VO_{2max} test, to permit assessment of anaerobic metabolism capacity and induce large metabolic adaptations in a short duration. When Wingate exercise is performed chronically in an exercise training paradigm it is an effective means of increasing cardiac output (CO), and the muscular demands for fuel and oxygen. Several studies have modified the load of the bike, or the duration of the exercise, but the central theory behind the Wingate exercise remains the same (Sagiv et al., 2005).

1.4.1 Wingate Training

MacDougall *et al.,* (1998) examined multiple Wingate exercise training to show that training does not need to be high in volume to achieve dramatic results. After seven weeks of low volume, high intensity supramaximal training, participants improved their maximum power output during Wingate exercise, as well as their VO_{2max} (MacDougall et al., 1998). Burgomaster *et al.,* (2005) observed similar results in a two-week training study that utilized multiple Wingate intervals separated by 4 minutes of submaximal

cycling. The authors concluded that two weeks of sprint interval exercise is sufficient to induce impressive increases in endurance capacity (mean increase of 100%) as measured by the total time that participants were able to maintain 80% of their predetermined VO_{2peak} (Burgomaster et al., 2005). High intensity interval training is in fact, one of the only ways to continue to realize improvements in endurance performance in highly trained athletes (Laursen & Jenkins, 2002).

Parra *et al.,* (2000) studied the effect of resting periods between training sessions, and found that when participants engaged in sprint interval training every day for a two-week period they failed to experience an increase in peak or mean power compared to pre training. Conversely, when participants completed the same number of training sessions, separated by two days of rest, they experienced a 20% increase in peak power, and 14% increase in mean power in the last training session compared to the first. The authors hypothesized that daily sprint training was so intense that in order to avoid muscle fiber fatigue with this strenuous protocol there must be a minimum of 48 hours separating training sessions. Burgomaster *et al.,* (2005) confirmed that when training sessions are separated by adequate recovery time, 2-weeks of Wingate training resulted in improved aerobic performance during an endurance time trial, but did not improve VO_{2peak}. In contrast, an improvement is VO_{2max} was observed after 8-weeks of submaximal interval training in 40 healthy university students (Helgerud et al., 2007). VO_{2max} is limited by CO and skeletal muscle blood flow so it would be interesting to observe the effect of this strenuous training stimulus on cardiovascular performance or adaptations (Calbet et al.,

2007). Although the cardiovascular effects of sprint interval training have yet to be determined, it is imperative that the acute effects of this form of exercise be examined.

1.5 Acute Cardiovascular Responses to Exercise

1.5.1 Heart Rate

It is well documented in the literature that the initial decline of HR at the termination of exercise is represented by parasympathetic reactivation (Cole et al., 2000; Dimpoulos et al., 2006; Savin, Davidson, & Haskell, 1982). Cole *et al.,* (2000) was able to attribute the HR response pattern during the entire 30-second period directly after exercise to vagal reactivation, independent of age or exercise intensity. Sustained increases in HR are consistent with previous observations following high intensity interval programs (Mourot et al., 2004), and have been attributed to the slow withdrawal of sympathetic tone and restoration of parasympathetic tone back to resting levels. Recovery HR is associated with exercise intensity in such a way that more intense exercise leads to prolonged sympathetic activation, and parasympathetic withdrawal (Forjaz et al., 1998; Mourot et al., 2005). When compared to continuous exercise, the recovery period following interval exercise exhibits a longer duration of sympathetic dominance, and parasympathetic withdrawal (Mourot et al., 2004). Sprint training may also increase the sensitivity of the adrenal medulla to sympathetic input, providing an advantage during situations where brief high intensity activity is required (Zouhal et al., 2001). Mourout *et al.,* (2005) observed the return of HR to resting levels following maximal exhaustive exercise in participants who had performed six weeks of interval

training. The training consisted of nine one minute intervals performed at the maximum workload attained during a previous VO_{2max} test, separated by four minutes of cycling at ventilatory threshold (Mourot et al., 2005). Mourot *et al.,* (2005) determined that HR recovered faster in participants that had completed previous interval training, indicating that parasympathetic tone is better able to resume autonomic control after interval training, than after continuous training. The authors proposed that the repeated changes in intensity, and corresponding adaptations in HR during an interval training session make this a more potent stimulus for training the autonomic nervous system.

1.5.2 Blood Pressure

Hypertension is a disease that affects over 50-million Americans, and is highly correlated with the indices of cardiovascular disease (JNC7). Exercise has been associated with acute and long-term reductions in BP (Hamer, 2006), and represents an effective alternative treatment for hypertension (Petrella, 1998).

Post exercise hypotension (PEH), is defined by a reduction in BP below resting levels immediately following the exercise stimulus (Fitzgerald, 1981). Although endurance exercise frequently results in PEH, the mechanisms and factors that affect this acute drop in BP remain unclear (Forjaz et al., 1998). BP is the product of CO and total peripheral resistance (TPR) so changes in either of these variables will have a direct effect on BP. CO is the amount of blood leaving the heart per minute, and it increases from rest to exercise to meet the metabolic demands of the body. TPR is the resistance in the blood vessels that the blood must overcome to circulate throughout the body.

Investigators suggest that increased perfusion and dilation in the non-working limbs following exercise may decrease TPR at a given CO, and therefore decrease BP (Bond et al., 2002; Hamer 2006; MacDonald, 2002).

Previous research indicates that the intensity of submaximal exercise is not related to the appearance, magnitude, or duration of PEH, and that reduced cardiovascular load in the form of hypotension occurs following dynamic exercise that ranges in intensity from 30-100%VO_{2max} (Cornelissen & Fagard, 2004; Forjaz et al., 1998). The magnitude of BP reduction after exercise is also directly correlated with resting BP values; which makes exercise therapy an attractive treatment for hypertensive individuals (Dujić et al., 2006; Forjaz et al., 2000, Petrella, 1998). In order for exercise to be an effective therapy for hypertension, the decrease in BP has to be significant in magnitude, and sustained for a prolonged period (Cornelissen & Fagard, 2004). A number of studies have examined BP during the extended recovery period following exercise using ambulatory BP measurements (Cornelissen & Fagard, 2004; Forjaz et al., 2000; Pescatello et al., 1991). Preliminary data from these studies indicates that exercise of varying intensities may affect BP to a different degree at different times throughout the day. Using ambulatory BP techniques Pescatello *et al.,* (1991) discovered that higher intensity exercise might elicit small changes in BP during sleeping hours. These results were corroborated by Cornelissen & Fagard (2004), and would explain why acute changes in hypotension have not been observed in previous studies.

Directly following maximal exercise a transient decrease in BP occurs in the first 15 seconds. This immediate drop in BP following dynamic exercise is attributed to the

cessation of the muscle pump, causing an immediate decrease in venous return and preload (Takahashi et al., 2000; Nakahara et al., 2006). The transient drop in BP stimulates baroreceptors in the arterial and carotid bodies to send a strong vasoconstriction signal, and there is a marked rebound in blood pressure which is subsequently followed by a gradual decline towards resting values over six minutes, as the body returns to the resting state (Nakahara et al., 2006).

A single supramaximal bout of exercise causes an abrupt increase in systolic blood pressure (SBP), which then returns to resting values within 10 minutes of supine recovery (Hussain et al., 1996). This SBP response pattern after supramaximal exercise is consistent with SBP trends reported after maximal exercise. Diastolic blood pressure (DBP), immediately following a single supramaximal exercise bout decreases compared to rest, although it returns to resting levels within 30 minutes if the participant is placed in a supine position (Hussain et al., 1996).

Crisafulli *et al.,* (2004) examined the BP response to multiple intervals which were performed at a workload of 150% of maximum watts and observed significant decreases in DBP during the entire recovery period. The majority of studies to date have observed BP responses using auscolometry or oscillometry systems. It has been determined that there are weaknesses in using these methods when detecting acute changes after exercise (MacDonald et al., 1999). As a result, a great deal of caution must be exercised when comparing the results from these studies. Continuous BP measurement provides another means of analysis. It has been reported that this method typically underestimates absolute pressures, but is highly effective at tracking BP changes

(MacDonald et al., 1999). The supine BP response following multiple Wingate exercise has yet to be determined using auscolometry.

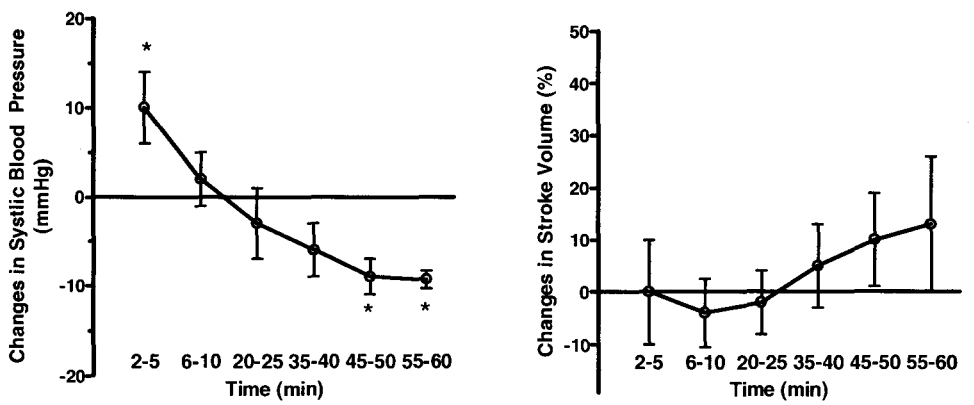
1.5.3 Stroke Volume

Possible factors that affect SV are preload, myocardial contractility, and afterload (Dujić et al., 2006). Takahashi *et al.,* (2005) examined the cardiovascular changes following light exercise in the supine and upright positions, and found that in all cases SV was increased following activity. SV remained higher when participants participated in active recovery, and declined most rapidly when participants underwent a passive upright recovery period. The decreases in SV as compared to the end of exercise observed during upright passive recovery were attributed to venous pooling in the lower limbs (Takahashi et al., 2005). When exercising at higher intensities, the peak and nadirs in SV become increasingly enhanced. These results seem to indicate that more intense activity increases perfusion and dilation in the working limbs. During passive upright recovery from intense exercise blood pools in the legs, decreasing preload and SV, and during passive supine recovery venous return to the heart is enhanced, causing a relative increase in preload, and ultimately SV compared to the supine responses (Dujić et al., 2006).

Calbet *et al.,* (2007) measured SV in nine healthy males with the dye-dilution method during a steady incremental exercise test to exhaustion, and observed a plateau in SV of 147 ml/beat at 64% of maximal workload, as measured in a graded exercise test to exhaustion. In contrast, Mortensen *et al.,* (2005) found that healthy males exhibited a decrease in SV from a maximal value of 160 ml/beat to 142 ml/beat, as exercise

approached maximal effort in an incremental exercise test to exhaustion (SV measured as the quotient of CO derived by the Fick principle and HR). The difference in the two protocols suggests that if workload increased at uneven intervals then SV will decrease after CO max is reached. The authors also suggest that the increased mean arterial pressure (MAP) observed by Mortensen *et al.,* (2005) could have further affected afterload, and decreased SV.

Concerning the end point at maximal exercise, some researchers have failed to observe an increase in SV (Calbet et al., 2007; Mortensen et al., 2005; Piepoli et al., 1993). Although CO was increased at end exercise, due to elevations in HR, the researchers failed to observe any change in SV compared to rest from 2 to 60 minutes following recovery. The authors suggest that SBP may have a greater effect on SV and large artery compliance, and DBP best represents TPR. Figures 1 and 2 from Piepoli *et al.*, (1993) indicate that immediately following exercise SBP is high, yet SV is not elevated from rest.



Figures 1 and 2 Measurements taken in the supine position after maximal exercise. (Piepoli et al., 1993).

All-out supramaximal exercise may present a stimulus quite different from submaximal or maximal exercise to the cardiovascular system. Echocardiograph images of the heart at peak anaerobic effort indicate that older males experience a decrease in left ventricular function immediately following Wingate exercise as compared to younger men (Sagiv et al., 2005). Although in this particular study, the older males were cycling against a lower force, it was determined that the decrease in ventricular function was most likely caused by an increase in afterload on the heart.

It is unknown if SV will plateau during repeated supramaximal interval exercise, or if the changes in workload will have a negative effect on SV. Although the training benefits of interval exercise on SV (Helgerud et al., 2007) have been confirmed, the effect of this unique exercise on the acute recovery following exercise have yet to be determined using accurate measurement techniques.

1.5.4 Cardiac Output

Changes in CO are caused by the interaction of two factors: HR, and SV. For most populations the age predicted HR maximum represents the highest HR attainable. SV, unlike resting HR, does not change when fitness levels improve (Hickson, Bomze, & Holloszy, 1977). CO increases during exercise to respond to the metabolic demands for blood flow throughout the body. When participants recover in the supine position, the rapid increase in SV, attributed to increased venous return, is combined with a rapid decline in HR (Takahaahi et al., 2000; Takahashi et al., 2005). These changes together

ensure that CO is maintained at a consistent level, until the demand for increase flow return to resting levels.

Studies examining central hemodynamics following maximal exercise, have observed that CO is elevated in the supine position during recovery compared to rest due to corresponding increases in SV and HR (Raine et al., 2001). Although CO is elevated both during and following exercise, it may be more appropriate to discuss this variable by examining HR and SV recovery patterns directly.

1.5.5 Leg Blood Flow

At the onset of dynamic exercise, an increase in sympathetic activation vasoconstricts the peripheral resistance arterioles and results in decreased blood flow to inactive vascular beds; concurrently, metabolic vasodilation and increased blood flow in active vascular beds, contribute to a redistribution of blood flow to the active muscle (Pricher et al., 2004). After exercise, there is evidence of greater peripheral vasodilation compared to rest in both the working and non-working limbs for thermoregulatory purposes after maximal exercise (Raine et al., 2001). It has also been shown that the degree of vasoconstriction is directly related to the intensity of the exercise, meaning that more intense exercise will increase perfusion and blood flow to exercising muscles, and constrict flow to non-working muscles to a greater degree (Raine et al., 2001). The mechanisms responsible for the prolonged increase in skeletal blood flow after exercise are poorly understood, but may be due to ongoing metabolic signals being released from previously exercised muscles (Williams, Pricher, &Halliwill, 2005).

Resting common femoral artery (CFA) diameter has been shown to vary quite widely between individuals and increases substantially as a result of training (Huonker, Halle, & Keul, 1996; Rådegran & Saltin, 2000). The adaptations that result in increased conduit artery diameter have been attributed to increased metabolic stress (Rådegran & Saltin, 2000). Although Rådegran & Saltin, (2000) did not detect a change in common femoral artery diameter when participants were required to kick at their maximum power output, the relationship between VO_{2peak} and artery diameter suggests that CFA diameter may be limiting at peak exercise intensities and thus increase with increases in physical fitness. Huonker, Halle, & Keul, (1996) hypothesized that sheer stress caused by increased blood flow during dynamic exercise is the mechanism behind arterial adaptations in conduit arteries. Increased intensity or duration of training would cause even greater elevations to metabolic stress, and would allow for greater adaptations in conduit artery diameter. These increases in conduit diameter would allow for increased blood flow during exercise.

Few studies have examined leg blood flow characteristics during exercise due to the difficulty isolating femoral flow during dynamic exercise. The few studies that have follow CO and femoral flow simultaneously found that, although CO continues to rise with increasing intensity, SV plateaus at 64% of maximum workload as measured in an incremental VO_{2max} test and remains at this level until exhaustion. Additionally, TPR in the legs decreases throughout exercise, the percentage of blood flow being directed to the legs remained constant after participants reached 64% of their maximum workload (Calbet et al., 2007). After maximal exercise leg blood flow is elevated compared to rest

for 105 minutes, and not related to hypotension in the supine position (Williams et al., 2005).

Hussain *et al.,* (1996) observed a seven fold increase over resting values in CFA blood flow two minutes after single Wingate exercise that returned to resting values after 60 minutes of supine recovery. Investigators were also able to detect increases to the profunda femoral artery (PFA), and superficial femoral artery (SFA) using Doppler ultrasound. Although the PFA experienced preferential flow immediately after the Wingate exercise, the SFA exhibited a much longer recovery time constant (Hussain et al., 1996). The investigators speculated that flow to the PFA immediately following exercise was due to the increased demands of the thigh muscles. To date, blood flow in the CFA, SFA, and PFA has yet to be examined after repeated Wingate exercise.

1.6 Conclusion

Although researchers have examined the acute cardiovascular effects of a single Wingate, the cardiovascular effects of a typical multiple Wingate exercise session remain unexplored. Multiple Wingate exercise is a potent exercise stimulus, and may present an alternative way to enhance physical conditioning in considerably less time than traditional endurance exercise training. It is important that an accurate timeline of the cardiovascular changes caused by sprint interval exercise be evaluated. Documenting the changes in HR, BP, SV, CO and leg blood flow will not only provide an explanation to the training effects observed following multiple Wingate training, but will also help

determine the safety of this exercise and the extent to which it stresses the cardiovascular system.

1.7 Purpose

The purpose of the current investigation was to identify the integrated cardiovascular responses in healthy young participants following multiple Wingate exercise. This information will help characterize the effectiveness of sprint interval training as an alternative to endurance type cardiovascular training. The purpose of this study was two fold; the first of which was to follow the recovery pattern of HR, CO, BP and leg blood flow following multiple Wingate exercise. As a secondary goal of the current investigation, the recovery pattern following a single Wingate was followed in an attempt to replicate previous data, and compare the recovery patterns of HR, CO and leg blood flow between the two exercises.

Following multiple Wingate exercise we hypothesized that HR, SV, CO, BP, and leg blood flow would be elevated above resting levels, and experience a steady decline towards resting values for the duration of the recovery period. We also hypothesized that CO would return to resting values before leg blood flow, and that a significant decrease in mean and diastolic BP would exist after exercise. We further hypothesized that these cardiovascular responses would exhibit similar trends, but be attenuated in magnitude and duration following a single Wingate exercise session.

2.0 METHODS

2.1 Participants

Ten recreationally active males aged 19.8 ± 1.2 (mean ± SD) years, with a mean height and body weight of 181.1 ± 2.5 cm and 77.7 ± 2.5 kg respectively were recruited for this study. Participants were healthy non-smokers, and free of cardiac abnormalities. All testing and familiarization sessions took place in the Exercise Metabolism and Research Laboratory in the Department of Kinesiology at McMaster University. Participants visited the lab on three separate occasions. On the first visit participants were familiarized with the testing equipment and multiple Wingate exercise, performed a VO_{2max} test, and resting cardiac images were obtained. On the second and third visit participants performed either a single Wingate, or a typical bout of multiple Wingate exercise in a randomized order. All participants gave informed consent in accordance with the Hamilton Health Sciences/McMaster University Research Ethics Board.

2.2 Preliminary Testing

2.2.1 Preliminary Testing

Familiarization was completed at least 48 hours prior to the first exercise session. During familiarization participants were introduced to the testing procedures and equipment used during all subsequent testing sessions. In addition, during this initial

visit, resting cardiac images were obtained for later analysis, a standard progressive cycling test to exhaustion, and a single familiarization Wingate were also performed.

2.2.2 Aortic Root Measurements

Images of the aortic root were obtained, using ultrasound while participants were at rest in the left lateral decubitus position. Parasternal long axis view, ultrasound images were taken with a 2.5 megahertz (MHz) ultrasound sector probe (GE Vingmed System FiVe, Horten, Norway). Brightness mode was used in combination with a single lead electrocardiogram to obtain images for a minimum of three consecutive cardiac cycles for each participant, which were then stored digitally for later diameter analysis. Commercially available software (EchoPAC V.6.2, GE Vingmed Ultrasound, Horten, Norway) was used to measure the diameter of the aortic annulus at end systole and end diastole of each heart cycle. The aortic root diameter (ARD) was calculated using equation 1.

ARD = (1/3) end systolic diameter + (2/3) end diastolic diameter Equation 1

2.2.3 Maximal Oxygen Uptake (VO_{2max})

Maximal oxygen uptake was determined from a progressive, incremental VO_{2max} test on a cycle ergometer (Excalibur Sport V2.0, Lode, Groningen, The Netherlands). All expired gasses were collected online (Moxus Modular Oxygen Uptake System, AEI Technologies, Pittsburgh, PA). The metabolic cart was calibrated prior to testing with a known composition of standard gasses. Heart rate (HR) was collected continuously

throughout the test (Model XL, Polar USA, Montvale, NJ). The VO_{2max} test consisted of three initial two minute stages, where the resistance increased by 50 Watts at each successive stage. At 150 Watts the resistance continued to increase by 25 Watts each minute until volitional failure. Volitional failure was determined as the point when participants could no longer maintain a cadence of 40 revolutions per minute (RPM). Breath by breath expired gas results were averaged every 15 seconds, and the maximal oxygen uptake was determined to be the highest 30 second average.

2.2.4 Familiarization Wingate

After a two-minute warm-up period, participants completed a single familiarization Wingate consisting of a 30 second all-out exercise bout on a cycle ergometer (Excalibur Sport V2.0, Lode). Participants were instructed to cycle as fast as possible against 0.075kg/kg body weight for the full 30 seconds while receiving continuous verbal encouragement. Resistance was applied to the cycle ergometer by computer software (Wingate Software Version 1.11, Lode).

2.3 Control of Diet and Exercise

In an effort to maintain constant fitness levels during the testing period, participants continued to engage in their normal recreational activities for the duration of the study. However, participants were advised to avoid vigorous physical exertion for 24 hours before testing. All participants were directed to avoid and drug, alcohol, nicotine and caffeine intake twelve hours prior to testing. On the testing days participants

reported to the lab after an eight-hour fast with the exception of a standardized meal replacement drink (Ensure Plus, Laboratories. Abbott Park, Illinois, U.S.A.) which they were instructed to consume four hours before exercise testing. Participants were also asked to record their last meal of the day on the day prior to the first testing day, and repeat that meal on subsequent testing days.

2.4 Sprint Testing Protocol

Temperature was controlled at 22-24°C. Both the single Wingate multiple Wingate exercise occurred at the same time of day, and a minimum of seven days separated the tests to avoid and effects of training or fatigue. On each testing day a 24-gauge catheter was inserted into the acubital vein of the right arm participants while rested in the supine position. Blood samples from this catheter were used to determine pH and lactate levels, as well as packed cell volume. The results of these measurements can be found in Appendix C. A standard oscillometry blood pressure cuff (Dynamap Pro 100 V2, GE Medical Systems, Tampa FLA, USA) was placed on the subject's upper left arm for subsequent blood pressure measurements. Standard one lead heart rate electrodes were placed in the standard CM5 placement. Heart rate was recorded continuously (Powerlab 16sp, ADInstruments, Colorado Springs, CO, USA), while all other measurements were taken at 15-minute intervals. Participants rested in the supine position for 60 minutes prior to exercise. Baseline measurements were taking at B0, B15, B30, B45, and B60. The first and last measurements were discarded and the remaining three measurements were averaged together to obtain a single resting value. Immediately

following exercise participants returned to the supine position and measurements continued every 15 minutes until the recovery period assessment terminated at 120 minutes. During the recovery period, participants were allowed to drink water ad labium.

2.4.1 Exercise

After 60 minutes of supine rest, participants were assisted onto a cycle ergometer (Excalibur Sport V2.0, Lode) and performed two minutes of warm-up cycling at 40 Watts. The warm-up was immediately followed by either a single 30 second all-out Wingate, or a typical bout of multiple Wingate exercise consisting of four Wingates separated by 4 minutes of light cycling at 40 Watts (Burgomaster et al., 2005). Resistance was applied to the cycle ergometer by computer software (Wingate Software Version 1.11, Lode). Participants were instructed to cycle as fast as possible for the entire 30 seconds of resistance while receiving continuous verbal encouragement. Exercise characteristics, including mean power, peak power, minimum power, and fatigue index, were calculated and saved using commercially available software (Wingate Software Version 1.11, Lode). Exercise sessions were separated by at least seven days, and performed at the same time of day. Immediately following the completion of the last 30-second Wingate participants were placed in a supine position for the acquisition of recovery data.

2.5 Heart Rate Analysis

Heart rate was determined from the R-R intervals captured by a 1 lead ECG in the standard CM5 position. The analogue signal from the bioamplifier was converted using a data acquisition board (Powerlab 16sp, ADInstruments, Colorado Springs, CO, USA) and collected using Chart 5 software (ADInstruments, Colorado Springs, CO, USA) and stored on a computer for later off-line analysis (IBM Nevista x86 compatible processor, White Plains, NY, USA). A second set of electrodes provided a second ECG signal for the ultrasound system (System Five, GE Medical Systems, Horten, The Neterlands) to be used when collecting subsequent arterial images. Heart rate was assessed continuously throughout the entire protocol, and calculated as the one minute average starting at the beginning of each measurement time point.

2.6 Cardiac Output Analysis

Stroke velocity was measured using a 2.0 MHz continuous wave Doppler ultrasound probe (GE Vingmed CFM 800, Horten, Norway), positioned at the suprasternal notch while the subject rested in the supine position. The frequency signal was converted to a velocity signal via the internal frequency analysis system (GE Vingmed CFM 800, Horten, Norway) and the peak velocity signal which was generated was converted to a digital signal (Powerlab 16sp, ADInstruments, Colorado Springs, CO, USA). The digital signal was then collected using Chart 5 software (ADInstruments, Colorado Springs, CO, USA) and stored on a computer for off-line analysis (IBM

Nevista x86 compatible processor, White Plains, NY, USA). Stroke velocity was collected for a total of one minute at each measurement time point. The highest quality ten consecutive beats were used for further stroke velocity analysis. Stroke volume (SV) was determined by multiplying the time-velocity integral (TVI) in equation 2 (Ihlen et al., 1984) by the aortic root area. Cardiac output was calculated using the equation 3.

$$SV = TVI \times ((ARD/2)^2 \times \pi) \qquad \text{Equation 2}$$

$$CO = SV \times HR. \qquad \text{Equation 3}$$

2.7 Blood Pressure Analysis

Blood pressure was determined on the left upper arm via automated oscillometry (Dynamap Pro 100 V2, GE Medical Systems, Tampa FLA, USA) while the subject rested in the supine position. Five separate resting measurements were taken (0min, 15min, 30min, 45min, 60min). The first and last blood pressure measurements were excluded, and the remaining three (15min, 30min, 45 min) averaged to represent single resting blood pressure measurement. Following exercise, blood pressure measurements were taken about the 2-minute time point, at the 15 minute time point, and continued at15-minute intervals. Pulse pressure (PP) was determined by subtracting diastolic blood pressure (DBP) from systolic blood pressure (SBP) as described in equation 4. Mean arterial pressure (MAP) was calculated using equation 5.

$$PP = SBP - DBP \qquad \text{Equation 4}$$

$$MAP = (1/3)\ PP + DBP. \qquad \text{Equation 5}$$

2.8 Leg Blood Flow Analysis

2.8.1 Diameter Analysis

Images of the common femoral artery and the superficial femoral artery were obtained in B-Mode ultrasound using a ten MHz linear array Doppler probe (GE Vingmed System FiVe, Horten, Norway), and digitally saved to hard disc for later off-line analysis. Images were taken at the beginning of each measurement time point, and captured at a rate of 11 frames per second. The artery diameter was captured for a total of five consecutive heart cycles, resulting in approximately 52 frames of each artery per time point.

Common femoral artery diameters were typically analyzed 2-4 cm proximal to the bifurcation that divides the common femoral artery into the superficial femoral artery and the profunda femoris artery. The superficial femoral artery and profunda femoris artery diameters were analyzed approximately 2-3 cm distal to the bifurcation.

Automated edge detection software (AMS II, Chalmers University of Technology, Goteborg, Sweeden) was used to determine the average diameter of the vessel of interest for each heart cycle. This software allowed the image to be loaded and visualized. The investigator chose the clearest section of the artery that encompassed both the near and far vessel wall, as the region of interest and kept this region consistent for all subsequent images and measurements on that participant. Measurements were taken from the leading edge of the vessel wall to the trailing edge of the vessel wall excluding the endothelium. Computer software calculated a minimum of 100 diameters for each frame. These diameters were averaged to get a single diameter measurement for

each frame. The average diameters were then graphed to show the change in diameters over five consecutive heart cycles. A single arterial diameter for each measurement time point was calculated as the average diameter over the cleanest 2-5 heart cycles.

2.8.2 Blood Velocity Analysis

Blood velocity (BV) was determined using the same ten MHz Doppler ultrasound probe operating in duplex mode: combined pulsed wave (PW) Doppler and B-Mode imaging in the same region of the artery that all images were taken. The raw quadrature frequency signal from the PW Doppler was connected in series to a spectral analyzer utilizing a fast fourier transform system to convert the signal to an intensity weighted power spectrum (model Neurovision 500M TCD, Multigon Industries, Yonkers, USA). The weighted mean signal was then sampled at 100Hz to obtain continuous tracings of the voltage representing the mean BV simultaneous to heart rate. The angle of insonation was recorded for all testing situations and previously generated regression lines were used to convert mean voltage to MBV. These standard calibrations were used to determine the blood velocity corresponding to each voltage and have been previously validated in our laboratory. An example of this calibration is presented in Figure 3. BV for each artery was obtained for a minimum of 30 seconds and averaged for subsequent analysis. Blood flow (BF) was calculated using equation 6.

$$BF = \pi \text{ (average diameter/2)}^2 \times \text{average BV} \times 60 \text{ sec} \qquad \text{Equation 6}$$

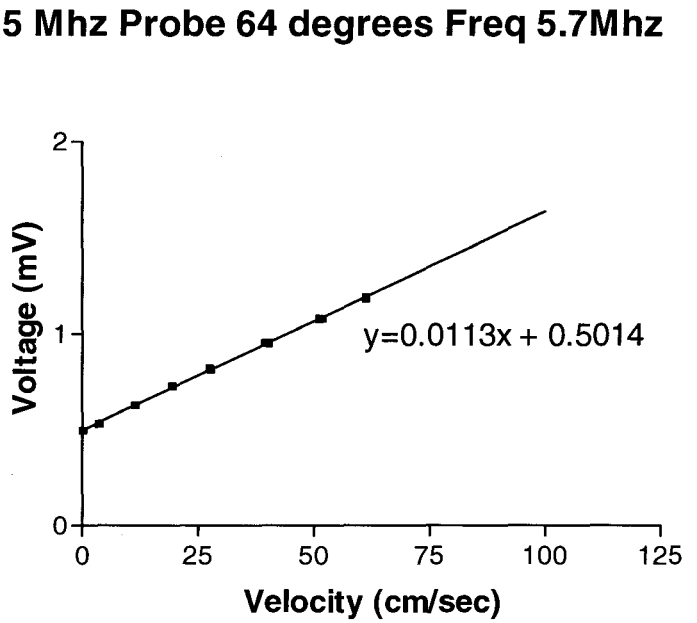


Figure 3 Regression graph of mean voltage against MBV. Equation of regression is inset.

2.9 Statistics Analysis

All data was initially tested for normalcy and equal variance using commercially available software (Sigmastat 3.10, Systat Software Inc., San Jose CA, USA). Data that fulfilled the assumptions of an ANOVA were analyzed with a 1 way repeated measure ANOVA, with trial (single, or multiple Wingate exercise) being compared across 10 measurement time points (R2, R15, R13, R45, R60, R75, R90, R105, R120). To compare the recovery periods between the two exercises a 2 way repeated measures ANOVA with trial (single, or multiple Wingate exercise) being compared across 10 measurement time points. When a significant F ratio was observed, the Tukey HSD post hoc was used for further analysis. Data that violated normalcy or equal variance was analyzed with Wilcoxon matched pairs test (Statistica 5.1, Stat Soft Inc., Tulsa OK,

USA). A p-level ≤ 0.05 was considered to be significant for all variables. All values are represented as mean $\pm$ standard error of the mean (SEM).

3.0 RESULTS

3.1 Exercise Characteristics

The ten recreationally active males that participated in this study were aged 19.8 ± 1.2 years, with a mean height and body weight and VO_{2max} of 181.1 ± 2.5 cm, 77.7 ± 2.5 kg, and 47.0 ± 0.8 ml/kg respectively. Individual participant characteristics can be found in Appendix A.

There was no difference ($p>0.05$) between mean power output during the single Wingate (658.1 ± 2.7 Watts) and the first multiple Wingate (670.8 ± 1.5 Watts). During multiple Wingate exercise, mean power was significantly lower after the first interval, reaching values of 576.0 ± 1.9, 495.1 ± 3.0 and 472.7 ± 1.7 Watts respectively during the second ($p=0.001$), third ($p<0.001$), and fourth Wingate interval ($p<0.001$). Details of exercise characteristics can be found in Appendix A.

3.2 Multiple Wingate Exercise

3.2.2 Heart Rate

Immediately following multiple Wingate exercise heart rate (HR) was increased to 122 ± 6 beats/minute from a resting value of 58 ± 3 bpm. HR then remained significantly elevated above rest for the remainder of the recovery period, and was still elevated at the last assessment time point at R120 ($p<0.05$).

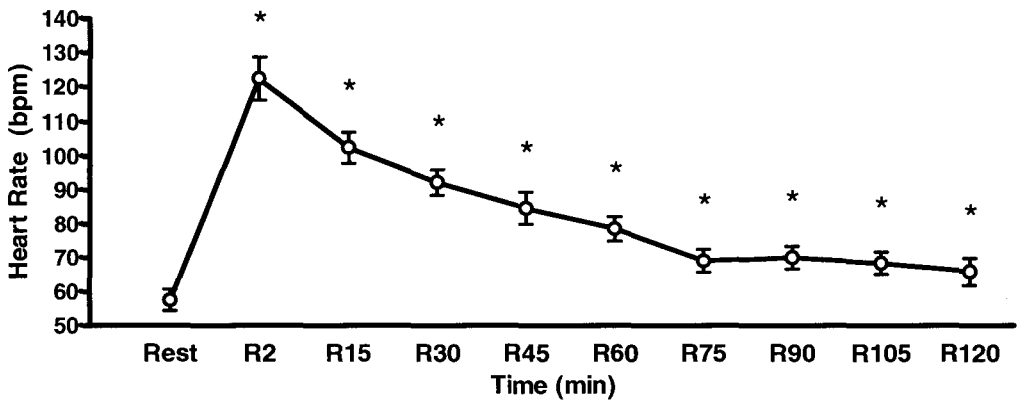


Figure 4 HR response following multiple Wingate exercise (parametric analysis)
* denotes a significant difference from rest ($p<0.05$)

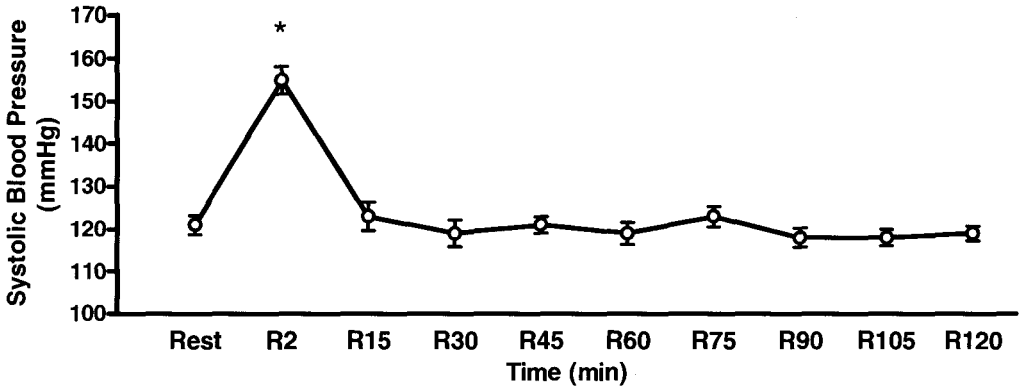
3.2.1 Blood Pressure

Systolic blood pressure (SBP) was elevated above resting levels immediately following exercise to 155 ± 3 mmHg. SBP returned to resting values at R15 and was not significantly different from resting values (121 ± 3 mmHg) at any time during the remainder of the recovery period ($p>0.05$) following multiple Wingate exercise.

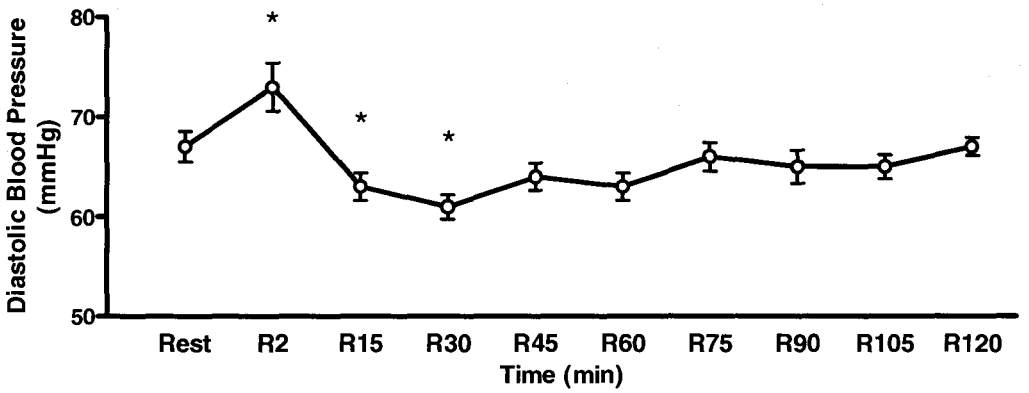
Diastolic blood pressure (DBP) was elevated immediately following exercise to a maximum value of 73 ± 2 mmHg. DBP then decreased below resting values, and was significantly reduced from rest (67 ± 2 mmHg) at R15 and R30 to and 63 ± 1 and 61 ± 1 mmHg respectively ($p=0.02$).

Mean arterial blood pressure (MAP) was elevated immediately following exercise (100 ± 3 mmHg), and decreased non-significantly ($p=0.07$) below resting values (85 ± 2 mmHg) following exercise to reach a minimum value of 80 ± 2 mmHg at R30.

A



B



C

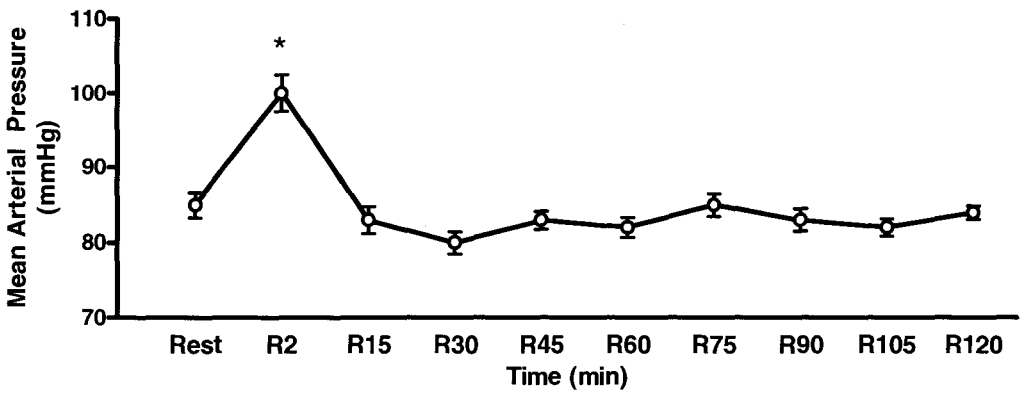


Figure 5 BP response following multiple Wingate exercise (SBP and MAP analysis by non-parametrics, DBP analysis by parametrics) ⋆ denotes a significant difference from rest ($p<0.05$)

3.2.3 Stroke Volume

Compared to rest, stroke volume (SV) decreased sharply immediately flowing exercise to a minimum value of 60.0 ± 5.6 ml. At R15 SV was not significantly different from resting values (87.3 ± 5.7 ml), however was decreased again as compared to rest from R30 to R60 after multiple Wingate exercise.

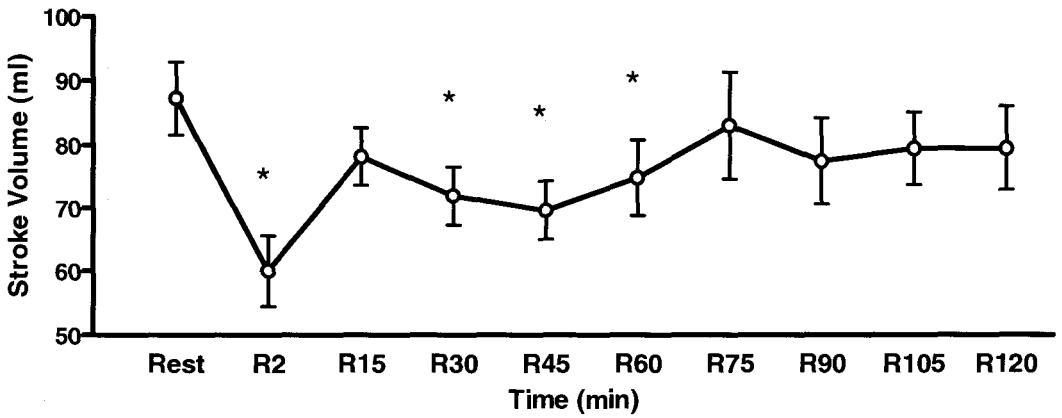


Figure 6 SV response following multiple Wingate exercise (parametric analysis)
* denotes a significant difference from rest ($p<0.05$)

3.2.4 Cardiac Output

Cardiac output (CO) was elevated following exercise from R2 to R30. CO reached a maximum value of 8.1 ± 0.7 L/min 15 minutes after the cessation of exercise and was not different from rest from R45 onward.

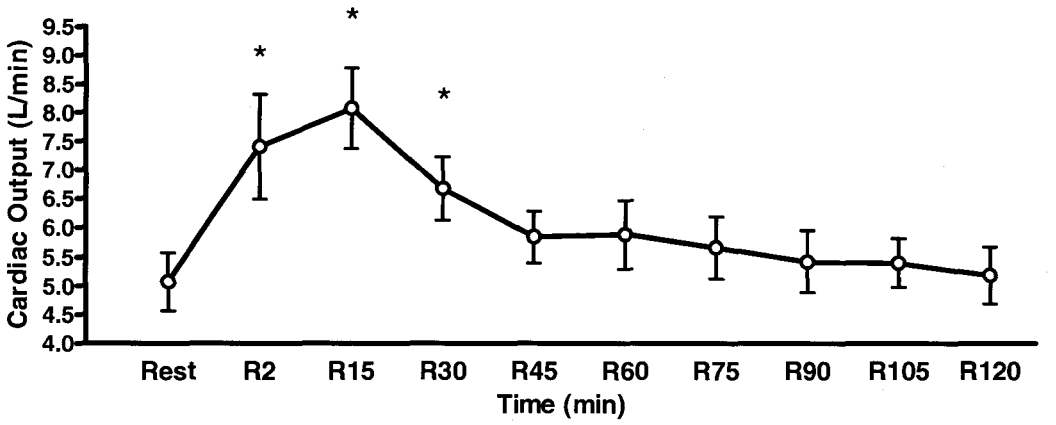


Figure 7 CO response following multiple Wingate exercise (non-parametric analysis)
* denotes a significant difference from rest ($p<0.05$)

3.2.5 Total Peripheral Resistance

Immediately following multiple Wingate exercise total peripheral resistance (TPR) was decreased below the resting values (17.9±1.5 mmHg/L/min) to 15.2 ± 1.7mmHg/L/min. TPR continued to decrease during the recovery period and reached a minimum value of 10.8 ± 0.7mmHg/L/min at R15 ($p<0.001$). For the remainder of the recovery period TPR continued to increase towards resting levels, becoming non-significantly different from resting values at R75 ($p>0.05$)

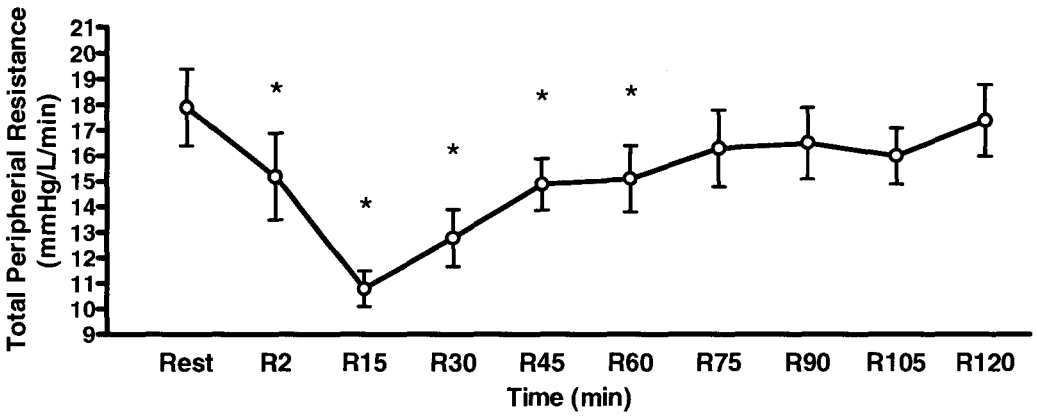


Figure 8 TPR response following multiple Wingate exercise (parametric analysis)
* denotes a significant difference from rest ($p<0.05$)

3.2.6 Leg Blood Flow

3.2.6.1 Common Femoral Arterial Flow

Blood flow through the common femoral artery (CFA) increased significantly from rest to the immediate recovery time point two-minutes after multiple Wingate exercise (130 ± 16 to 1036 ± 86 ml/min). CFA blood flow decreased towards resting values for the remainder of the recovery period, becoming non-significantly elevated above resting values at R90.

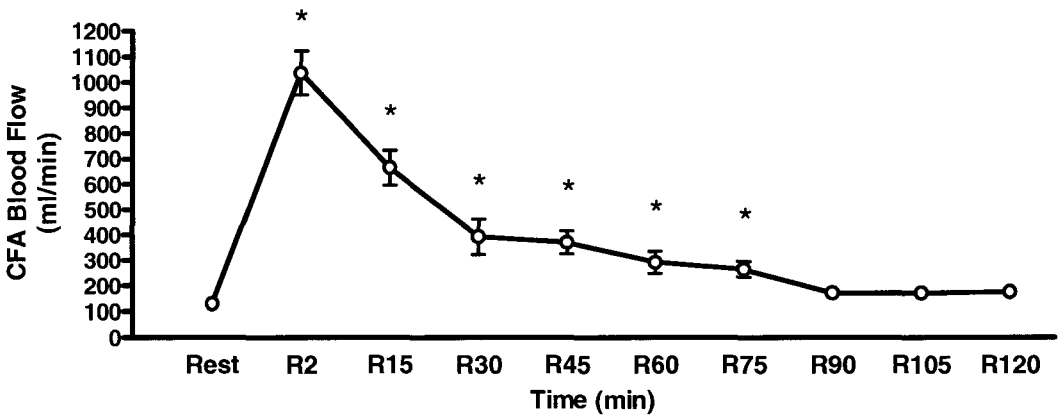


Figure 9 CFA blood flow response following multiple Wingate exercise (non-parametric analysis) * denotes a significant difference from rest ($p<0.05$)

3.2.6.2 Superficial Femoral Arterial Flow

Blood flow through the superficial femoral artery (SFA) increased significantly from rest to immediately following multiple Wingate exercise (98 ± 10 to 616 ± 55 ml/min). Although SFA blood flow decreased towards resting values for the duration of the recovery period, it remained significantly elevated above resting values at the cessation of the recovery period ($p<0.05$).

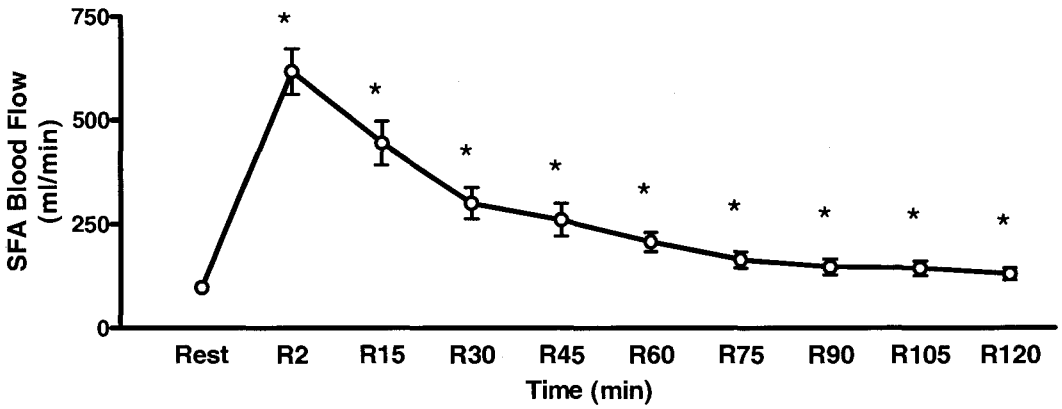


Figure 10 SFA blood flow response following multiple Wingate exercise (non-parametric analysis) * denotes a significant difference from rest ($p<0.05$)

3.3 Single Wingate Results

3.3.1 Heart Rate

HR increased from 56 ± 3 to 105 ± 6 beats/minute immediately following the single Wingate exercise, and continued to remain significantly elevated above rest ($p<0.05$) until the end of the testing period at R120.

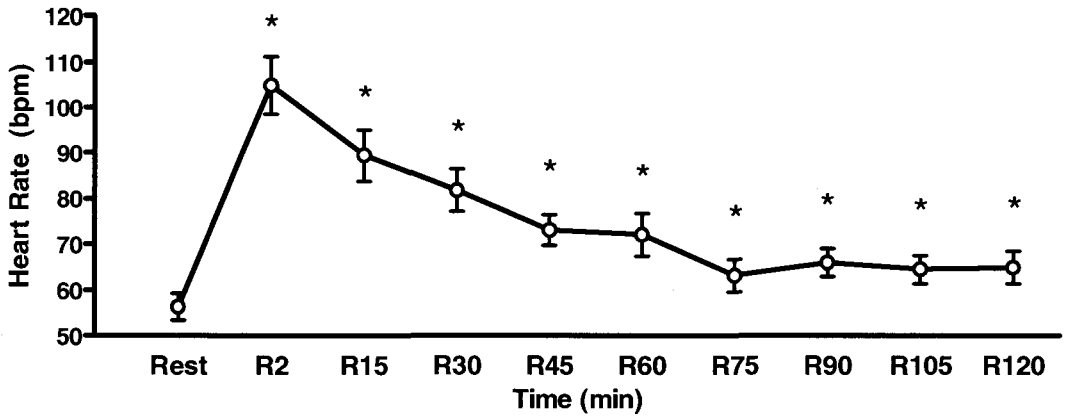


Figure 11 HR response following single Wingate exercise (non-parametric analysis)
* denotes a significant difference from rest ($p<0.05$)

3.2.3 Stroke Volume

Compared to rest, at R30 SV decreased below resting values to reach a minimum value of 68.1 ± 3.4 ml, before returning to resting values at R45. SV decreased again compared to rest from R60 to R120 (except R105) after single Wingate exercise.

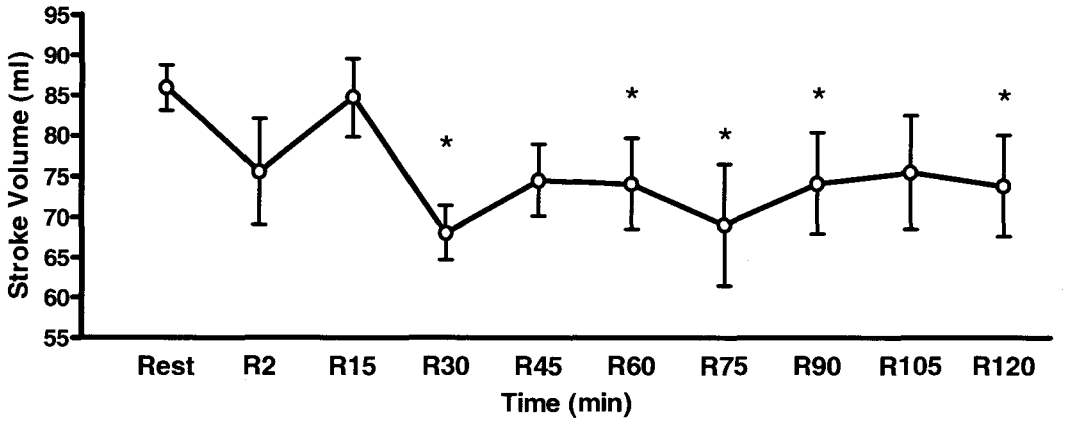


Figure 12 SV response following single Wingate exercise (parametric analysis)
* denotes a significant difference from rest ($p<0.05$)

3.2.3 Cardiac Output

CO was elevated following exercise from R2 to R15. CO reached a maximum value of 7.8 ± 0.7 L/min two-minutes after the cessation of exercise and was not different from rest from R30 onward.

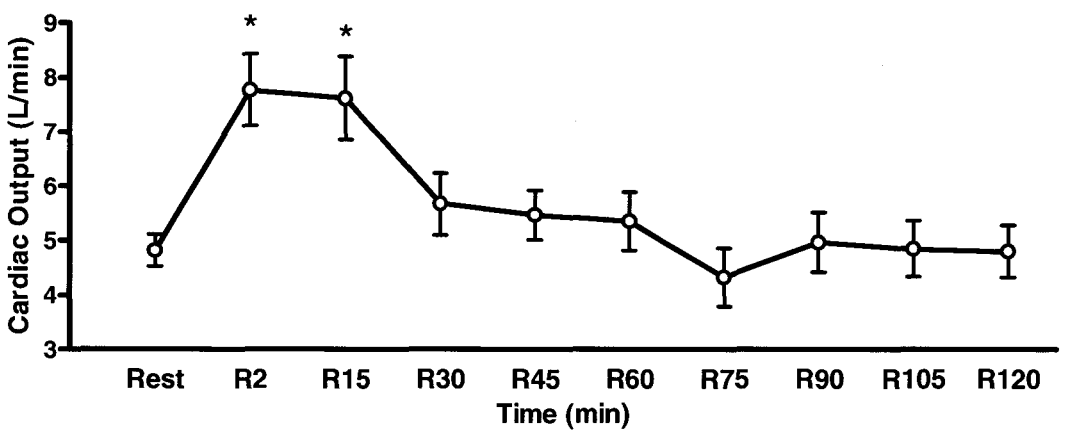


Figure 13 CO response following single Wingate exercise (parametric analysis)
* denotes a significant difference from rest ($p<0.05$)

3.3.4 Leg Blood Flow

3.3.4.1 Common Femoral Arterial Flow

Immediately following the single Wingate exercise CFA blood flow was elevated from 142 ± 18 ml/min at rest, to 1264 ± 109 ml/min at R2. CFA blood flow continued to decrease throughout the recovery period, and became non-significantly elevated above resting values at R60. Although CFA blood flow increased briefly at R75 ($p<0.05$), it quickly fell back to resting values at R90, and remained there for the remainder of the recovery period.

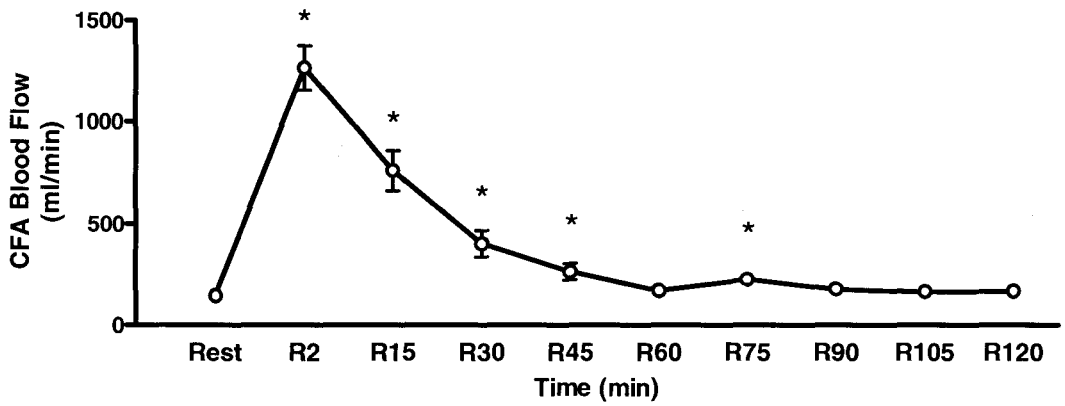


Figure 14 CFA blood flow response following single Wingate exercise (non-parametrics analysis) * denotes a significant difference from rest ($p<0.05$)

3.3.4.2 Superficial Femoral Arterial Flow

After a single Wingate exercise SFA blood flow increased from a resting value of 84 ± 9 ml/min to 579 ± 66 ml/min at R2. SFA blood flow continued to decrease towards resting values, becoming non-significantly elevated above resting values at R60 ($p>0.05$).

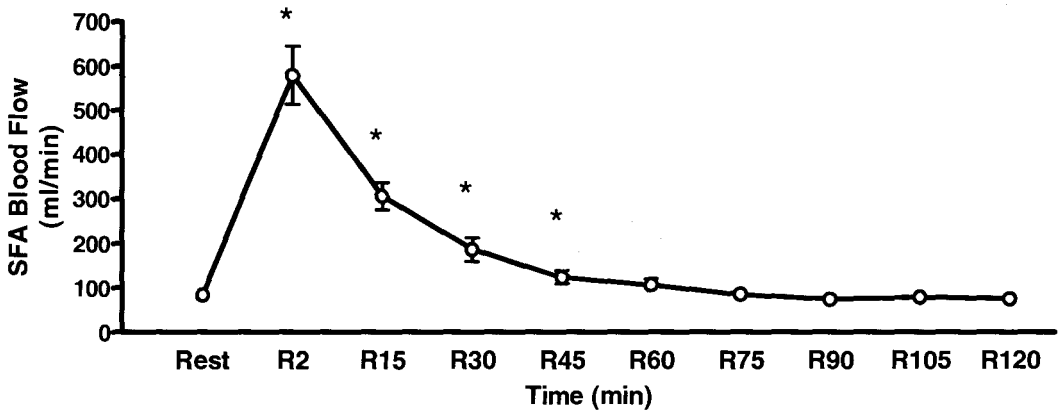


Figure 15 SFA blood flow response following single Wingate exercise (non-parametrics analysis) * denotes a significant difference from rest ($p<0.05$)

3.4 Comparison of Single Wingate and Multiple Wingate Exercise

3.4.1 Heart Rate

When comparing the HR recovery period following both types of exercise, multiple Wingate exercise caused HR to be significantly elevated above the single Wingate HR response at R2, R15, R30, and R45. After 60 minutes of recovery (R60), although HR remained significantly elevated above resting levels for both types of exercise, but there was no statistical difference between the HR following a single Wingate, and the HR following multiple Wingate exercise ($p>0.05$)

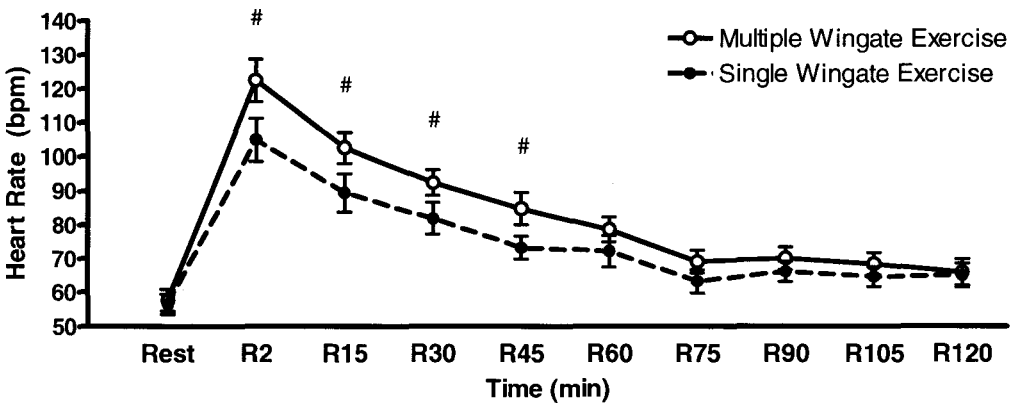


Figure 16 HR response following single Wingate and multiple Wingate exercise (non-parametrics analysis) # multiple Wingate exercise is different from the single Wingate exercise ($p<0.05$)

3.4.1 Stroke Volume

Immediately following exercise, SV after multiple Wingate exercise (60.0 ± 5.6 ml) was lower than the SV after single Wingate exercise (75.6 ± 6.5 ml). For the remainder of the recovery period, there were no significant differences between SV after single or multiple Wingate exercise, with the exception of R75. At R75 SV following

multiple Wingate exercise was briefly higher than SV following single Wingate exercise ($p<0.05$).

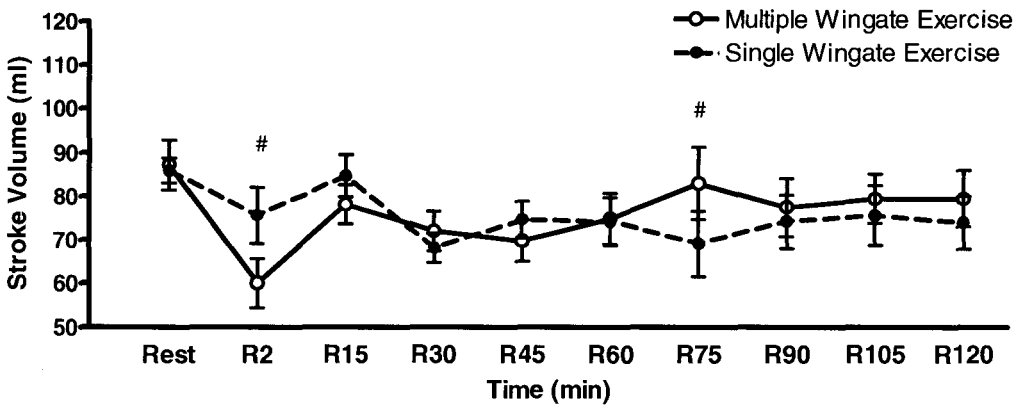


Figure 17 SV response following single Wingate and multiple Wingate exercise (non-parametrics analysis) # multiple Wingate exercise is different from the single Wingate exercise ($p<0.05$)

3.4.1 Cardiac Output

The acute CO recovery pattern following both single and multiple Wingate exercise was very similar. Following multiple Wingate exercise, CO was significantly elevated above single Wingate recovery values at R30 and R75 ($p<0.05$).

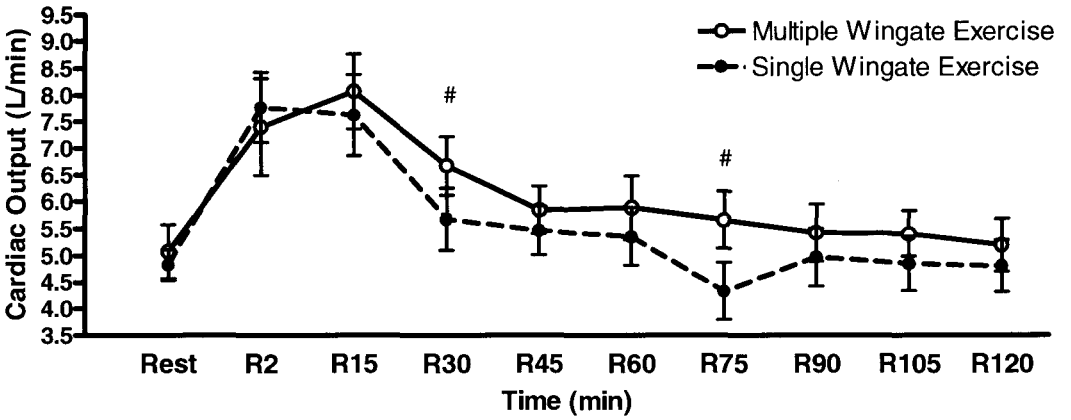


Figure 18 CO response following single Wingate and multiple Wingate exercise (non-parametrics analysis) # multiple Wingate exercise is different from the single Wingate exercise ($p<0.05$)

3.4.2 Leg Blood Flow

3.4.2.1 Common Femoral Arterial Flow

When comparing the individual recovery patterns following single Wingate and multiple Wingate exercise, CFA blood flow remained significantly elevated ($P<0.05$) following both exercises for a similar duration (R2 to R75). Following single Wingate exercise there was a drop below significance at the R60 time point that did not occur following multiple Wingate exercise.

When comparing the pattern of recovery of leg blood flow after single Wingate exercise to multiple Wingate exercise directly, there were several differences in the recovery patterns for CFA blood flow. Although immediately following both single Wingate and multiple Wingate exercise, CFA blood flow was elevated above resting values; the increase after multiple Wingate exercise was not as great as the increase following single Wingate exercise ($p<0.05$). Both recovery values decreased throughout

the recovery period, and remained statistically similar to each other until R45. CFA blood flow was significantly higher than blood flow following a single Wingate from R45 to R75. At R90 both recovery values were not significantly different from resting values, or each other (P>0.05).

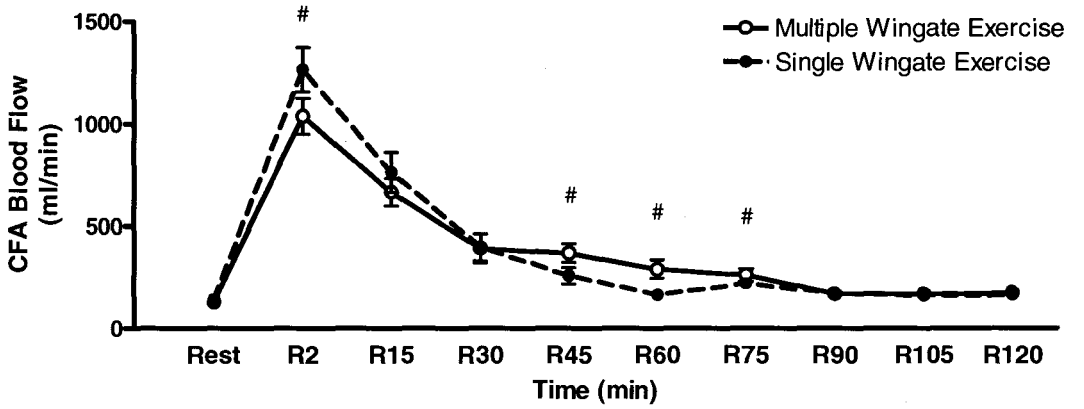


Figure 19 CFA blood flow response following single Wingate and multiple Wingate exercise (non-parametrics analysis) # multiple Wingate exercise is different from the single Wingate exercise (p<0.05)

3.4.2.2 Superficial Femoral Arterial Flow

SFA blood flow remained significantly elevated (P<0.05) following both single Wingate and multiple Wingate exercises from R2 to R45. From R60 and for the remainder of the recovery period only SFA blood flow following multiple Wingate exercise continued to be elevated above resting values (p<0.05). Although immediately following both single Wingate and multiple Wingate exercise, SFA blood flow was elevated above resting values; the increase after multiple Wingate exercise was not significantly different from the increase following single Wingate exercise (p>0.05). Both recovery values decrease throughout the recovery period, although from R15 to the

end of our testing protocol SFA blood flow following multiple Wingate exercise was significantly higher than the blood blow following single Wingate exercise ($P<0.05$).

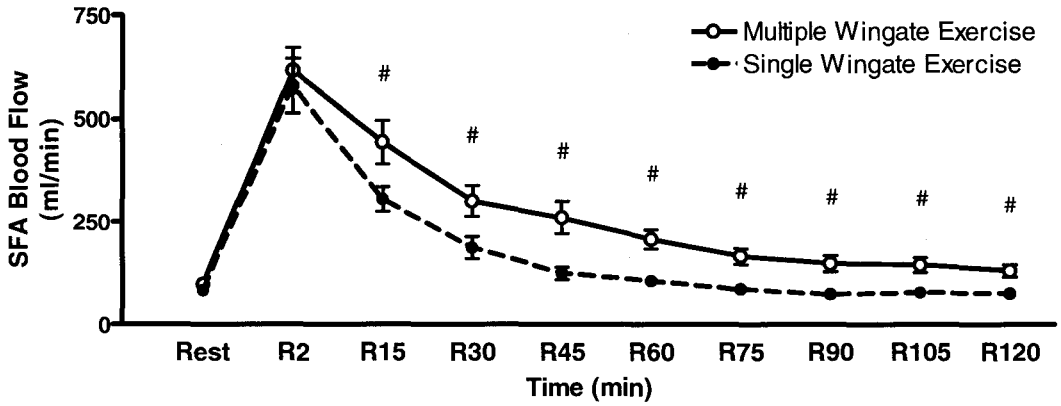


Figure 20 SFA blood flow response following single Wingate and multiple Wingate exercise (non-parametrics analysis), # multiple Wingate exercise is different from the single Wingate exercise ($p<0.05$)

4.0 DISCUSSION

This study was conducted to characterize the cardiovascular recovery period following repeated supramaximal interval exercise. Two-minutes following multiple Wingate exercise HR, BP, CO, and leg blood flow were all elevated compared to resting values, while SV was below resting values. As the recovery period continued, HR and leg blood flow declined toward resting values. BP and SV returned to resting values at the 15-minute measurement time point, allowing CO to increase to a maximum recorded value. Following the 15-minute recovery time point CO declined gradually towards resting values, we also observed decreases in SV and DBP values at various time points during the remainder of the recovery period. The discussion that follows examines the recovery pattern observed in each of the variables collected individually.

4.1 Heart Rate

In the current investigation, participants exhibited a HR of 122 ± 6 and 105 ± 6 beats/minute after two-minutes of passive supine recovery from repeated Wingate and single Wingate exercise respectively. The maximal HR values observed in the current investigation were similar to the 120 beats/minute reported by Hussain *et al.,* (1996) following two-minutes of supine recovery from single Wingate exercise. The HR observed in the current study and by Hussain *et al.,* (1996) were significantly lower then those recorded by Sagiv *et al.,* (2005) at peak anaerobic effort (187 beats/minute) and

Ben-Sira & Sagiv (1997) who recorded HR 15-seconds after a single Wingate in the seated position (188 beats/minute). It is likely that our participants achieved their maximum HR at some point during the 16 minute multiple Wingate exercise session. However, it is not known if the same HR was achieved during each Wingate interval. The similarities in HR observed after two-minutes of recovery in the supine position by Hussain *et al.,* (1996) after a single Wingate, and in the current study after multiple Wingate exercise could indicate that peak HR and acute HR recovery time are similar regardless of the durations of supramaximal interval exercise. Alternately, it could indicate that the peak HR and recovery pattern of the participants differed between the two exercise stimuli. Without knowing the peak HR achieved at the end of multiple Wingate exercise, it is impossible to comment on the recovery pattern between the end of the exercise and the first two-minute measurement.

HR remained elevated above resting values for the entire duration of the recovery period; the steady return towards resting values from R2 to the end of the recovery period (R120) suggests a smooth withdrawal of sympathetic activity, and reactivation of parasympathetic tone. Sustained increases in HR are consistent with previous observations following high intensity interval programs (Mourot et al., 2004), and have been attributed to the slow withdrawal of the sympathetic tone back to resting levels.

Mourot *et al.,* (2004) observed the return of autonomic balance by examining heart rate variability (HRV) following interval exercise. The exercise in the study by Mourot *et al.,* (2004) consisted of nine one-minute intervals at the power output required to achieve their maximum ventilatory threshold, separated by four-minutes of cycling at a

submaximal base workload. The authors observed the presence of enhanced sympathetic tone following a maximal intensity interval exercise protocol persisted for 60 minutes (Mourot et al., 2004). HRV analysis completed after multiple Wingate exercise in the current investigation indicates that sympathetic tone continued to be elevated at the end of the 120-minute recovery period (unpublished results).

4.2 Blood Pressure

The current investigation resulted in two significant observations regarding BP responses following repeated Wingate exercise; the magnitude of the immediate post exercise BP was less than expected following two-minutes of passive supine recovery, and that this period of elevated BP was followed by a subsequent decrease in DBP during the extended recovery period. In the current investigation, we observed a transient increase compared to rest in SBP, DBP and MAP of 34, 6 and 15 mmHg respectively at the two-minute recovery time point.

Sagiv *et al.,* (2005) and Ben-Sira & Sagiv, (1997) observed an increase in SBP of 86 mmHg at maximal anaerobic effort and 87 mmHg 15-seconds after a single Wingate while participants were seated on the cycle ergometer. Two-minutes after single Wingate exercise Hussain *et al.,* (1996) observed an increase in SBP of 49 mmHg in the supine position. Crisafulli *et al.,* (2004) observed a 60 mmHg increase in SBP using a manual sphygmomanometer one-minute after five bouts of 30-second interval exercise at 150% of the participants predetermined maximum power. These results indicate that the 34

mmHg increase in SBP observed in the current investigation is uncharacteristically lower than expected for this type of exercise.

Regarding DBP, Sagiv *et al.* (2005) and Ben-Sira & Sagiv (1997) observed an increase of 21 mmHg at maximal anaerobic effort and 28 mmHg 15-seconds after a single Wingate while participants were seated on the cycle ergometer. Following two-minutes of supine recovery from single Wingate exercise, Hussain *et al.* (1996) observed an increase in DBP of 4 mmHg in the supine position. The 6 mmHg increase in DBP we observed following two-minutes of supine recovery from multiple Wingate exercise is slightly larger, yet consistent with the results reported by Hussain *et al.* (1996). However, using a manual sphygmomanometer Crisafulli *et al.* (2004) observed a 20 mmHg drop in DBP one minute after repeated sprint interval exercise. These results contradict those observed in the current investigation, but the increased age of the participants studied by Crisafulli *et al.* (2004), use of a manual sphygmomanometer for BP measurement, and the upright recovery position used in their study may account for the differences in DBP observations. The authors attribute the decrease in DBP to massive vasodilation in the legs caused by metabolite end-product accumulation. Although we observed an increase rather than a decrease in DBP at the two-minute time point, it is possible that the supine position assumed by our participants increased venous return to the heart, and attenuated any venous pooling regardless of persistent vasodilation in the legs.

Sagiv *et al.* (2005) and Ben-Sira & Sagiv (1997) observed an increase in MAP of 42 mmHg at maximal anaerobic effort and 48 mmHg 15-seconds after a single Wingate

while participants were seated on the cycle ergometer. Two-minutes after single Wingate exercise Hussain *et al.* (1996) observed an increase in MAP of 30 mmHg in the supine position. Although the two-minute reported MAP increase of 15 mmHg in the current investigation is lower than those observed after a single Wingate, they are consistent with the 20 mmHg increase observed by Crisafulli *et al,* (2004) after repeated sprint exercise.

Due to the non-invasive nature of the BP measurement technique using in the current investigation, it was not possible to obtain BP measurements during exercise, so BP values between the end of exercise and two-minutes of recovery remain speculative. However, the significant rise in BP during and immediately following single Wingate exercise observed in previous studies (Sagiv et al., 2005; Ben-Sira & Sagiv, 1997; Hussain et al., 1996), indicates that during and immediately following multiple Wingate exercise BP was most likely elevated much higher than the increases of 34, 7, and 16 mmHg we have reported in the current investigation. Although the short term increase in SBP realized in the current study is consistent with the transient pressure increase following a single Wingate as reported by Hussain *et al.* (1996); it contradicts the decrease in SBP of 7 mmHg reported by Mourot *et al.* (2004) following several maximal exercise intervals. Mourot *et al.* (2004) utilized moderately trained males and recorded BP after 5 minutes of supine recovery. The exercise consisted of nine, one-minute intervals at ventilatory threshold separated by four-minutes of cycling at a submaximal base. In contrast, Crisafulli *et al.* (2004) had participants complete five one-minute intervals at 105% of their maximum power (as measured by an incremental VO_{2max} test) and recorded a SBP increase of 60 mmHg after 1 minute of active recovery in the seated

position. Although in Crisafulli *et al.* (2004) the increase in SBP was very short lived, and SBP was non-significantly elevated above resting values after five-minutes of recovery cycling. This could indicate that the supramaximal intensity of Wingate exercise elicits a much higher increase in sympathetic outflow, causing more pronounced vasodilation and increased BP as compared to maximal exercise, but that this increase is temporary in nature and returns to baseline levels very quickly. The pattern of response likely depends on the mode of recovery, as the results differ depending on the measurement technique, supine or seated body position, and active or passive recovery behavior.

Previous investigators have cautioned the interpretation of BP values obtained using automated auscultatory cuff measurements during and immediately following intense dynamic exercise (Ben-Sira & Sagiv, 1997; Sagiv et al., 2005) as SBP may be artificially elevated by pulse wave amplification (Rowell et al., 1968). However, intra-arterial catheter measurements of BP during maximal exercise (Rådegran & Saltin, 2000) and at near maximal exercise (Ogoh et al., 2007) have obtained similar results to cuff values during maximal exercise (Nakahara et al., 2006). As an alternative to automated auscultatory BP measurement, some previous research has been conducted using continuous non-invasive BP measurement systems such as Finapres, Portapress, or Finometer systems. Continuous non-invasive BP measurement systems have, however, been known to underestimate BP values, particularly DBP, following dynamic exercise (Macdonald, MacDougall, & Hogben, 1999). Post exercise BP measurements obtained using continuous BP (Finapres) in other studies differ quite substantially from other

reported measures (Raine et al., 2001; Rowell et al., 1968), as well as those obtained in the current investigation. This may be due to the effect of pulse wave amplification that increases as BP measurements are obtained further away from the aorta (Rowell et al., 1968).

In most previous research, recovery BP measurements obtained from the cessation of exercise to the two-minute recovery time point were all made while the participants were seated in the exercise position (Ben-Sira & Sagiv, 1997; Crisafulli et al. 2004; Nakahara et al., 2006; Ogoh et al., 2007; Piepoli et al., 1993; Rådegran & Saltin, 2000; Sagiv et al., 2005). In the current investigation, the first recovery measurement was taken following two minutes of recovery while subjects rested in the supine position. The supine position was chosen in the current investigation to facilitate measurement of SV and leg blood flow with Doppler ultrasound. The 34 mmHg increase in SBP and the 15 mmHg increase in MAP we reported are slightly less then the values of 49 and 30 mmHg reported by Hussain *et al.* (1996) for SBP and MAP at the two minute time point following a single Wingate, while the subjects rested in a similar position. When studies were conducted using maximal exercise (Nakahara et al., 2006) the elevations in BP above resting values at exhaustion were quickly followed by a decrease in pressure during the first 20 seconds of recovery, followed by a positive rebound, and then further decreases in pressure back to resting values. The authors attributed the rebound in pressure to activation of the arterial and carotid baroreceptors upon cessation of activity, causing massive vasoconstriction and subsequent increase in pressure. In the current investigation transition from the cycle ergometer to a supine position took no more than

ten seconds, yet it is reasonable to believe that our participants may have experienced a similar pressure reduction and rebound during the time between the cessation of exercise and the two-minute recovery time point.

SBP represents the influence of SV and large artery compliance (Piepoli et al., 1993). During intense dynamic exercise of this nature a massive sympathetic outflow is responsible for elevations in HR, systemic vasoconstriction, and lowering of large artery compliance as a protective mechanism. In the current situation, these factors likely combined to result in the early increase in SBP that was later followed by a decrease in DBP reflecting the vasodilation in the active muscle vascular beds. The effects of increased SBP on SV at the two-minute time point will be addressed later in this discussion.

4.2.1 Post Exercise Hypotension

In the current investigation no significant decreases in SBP or MAP were observed following repeated Wingate exercise, although DBP was significantly reduced below resting values at R15 and R30. The largest recorded decrease in DBP (-6 mmHg) occurred following 15 minutes of supine recovery, although it is possible that a larger decrease in DBP may have occurred at some time between the two minute and 15 minute measurements. A decrease in DBP that lasts for 30 minutes is consistent with the findings of Hussain *et al.* (1996) following a single Wingate, but in that study the largest decrease in DBP (-22 mmHg) was detected after 7 minutes of supine rest. Hussain *et al.* (1996) also noted a decrease in MAP at five minutes post exercise, which returned to

resting levels at the ten-minute time point. We did not detect and changes in MAP at either the two or 15 minutes post exercise, however there may have been undetected decreases in BP values between two and 15 minutes.

MacDonald, MacDougall, & Hogben, (1999) observed a drop in SBP of 8 mmHg below resting values five-minutes after light and moderate exercise in normotensive participants. Participants in the current investigation had average resting DBP, SBP, and MAP that were 3, 11, and 12 mmHg respectively lower than the average resting pressures observed by MacDonald, MacDougall, & Hogben, (1999). As the magnitude of the hypotensive effect has been shown to be proportional to the resting blood pressure (Forjaz et al., 2000; Petrella, 1998), it is possible that the participants in the current investigation were unable to achieve a significant prolonged decrease in DBP and MAP due to low resting BP.

Many studies have noted a substantial decrease in BP following exercise, when the participant remains in a seated or standing position (Nakahara et al., 2006; Takahashi et al., 2000). In many of these cases, the fall in BP has been attributed to the stress of gravity, combined with vasodilation in the legs and attenuation of the skeletal muscle pump (Nakahara et al., 2006; Takahashi et al., 2000). MacDonald, MacDougall, & Hogben, (1999) reported that post exercise hypotension should occur following dynamic exercise so long as the intensity exceeded 40% VO_{2max}. Although some authors have attributed a decrease in BP following exercise to a healthy lifestyle, it is doubtful that the small, transient decrease in BP documented after multiple Wingate exercise would have any long-term health benefits. However, there is evidence to suggest that intense

exercise promotes greater BP reductions during the night, which are not apparent during the immediate recovery period (Forjaz et al., 2000). Further investigations should focus on the long-term effect of multiple Wingate exercise on BP.

4.3 Stroke Volume

The large decrease in SV observed immediately following exercise is contradictory to both the original hypothesis, and recovery patterns observed in previous investigations examining both submaximal and maximal exercise. No previous investigations have documented the SV recovery pattern in the supine position following repeated supramaximal exercise in young healthy individuals.

At the two-minute time point following exercise, we observed a 31% decrease in SV compared to rest. At the next measurement time point SV had returned to resting values, but decreased slightly below resting levels again at the 30, 45, and 60-minute time points. A SV below resting values after exercise contradicts what is typically observed in healthy normotensive males in the supine position (Takahashi et al., 2005). A decrease in SV has been observed when participants remain in the upright and seated position following incremental exercise to exhaustion (Dujić et al., 2006). In the upright position, lower SV values are attributed to the attenuation of the leg muscle pump, combined with the additional stress of gravity causing venous pooling in the legs. When participants engage in an active recovery a SV decline is greatly attenuated or removed altogether (Carter, Watenpaugh, & Smith, 2001; Crisafulli et al., 2004). The possible factors affecting SV are preload, myocardial contractility, and afterload (Dujić et al., 2006).

4.3.1 Preload

Cardiac preload is dependent on both plasma volume and venous capacitance (Aardenburg et al., 2005; Dujić et al., 2006), and affects both venous return, and SV (Aardenburg et al., 2005). While investigators have generally rejected the notion that plasma volume has an effect on PEH, there is a direct documented link between plasma volume and SV (Dujić et al., 2006; Hayes, Lucas, & Shi, 2000; MacDonald, MacDougall, & Hogben, 1999; Mier et al., 1996). We observed a decrease in plasma volume after multiple Wingate exercise (see Appendix C), which could have contributed to the decreases in SV.

The decrease in plasma volume we observed is consistent with the 10-15% decrease in plasma volume following a single Wingate (Rotstien et al., 1982). Although the authors were not able to define the mechanism for this decline, they did suggest that changes in tissue osmolarity, increased capillary and filtration pressure, and the opening of collapsed arteries, and the loss of water due to perspiration could have affected the results (Cheatham et al., 2000; Hayes, Lucas, & Shi, 2000). In the current investigation packed red blood cell to plasma ratio was most significantly increased immediately following exercise. It has also been suggested by Yalcin *et al.* (2003) that red blood cell deformation may conflict with the normal hematological recovery patters following heavy exercise, and red blood cell damage (hemolysis) may be more pronounced in sedentary humans following exhaustive cycling exercise (Sentürk et al., 2005). Both cell

deformation and cell damage could result in increased blood viscosity and altered flow (Baskurt & Meiselman, 2003).

It is also possible that metabolite build up in the working muscles caused massive vasodilation of the legs both during and after exercise (Hussain et al., 1996). Upon assuming the supine position, reports from head up tilt studies indicate that the return of blood back to the heart would have occurred within the first 10 seconds, and possibly even faster due to an elevated heart rate after exercise. It is possible that continued arterial and venous vasodilation in the legs might have prevented full venous return. This theory is consistent with work by Crisafulli *et al.* (2004) who found that repeated bouts of supramaximal exercise impaired venous return more than a single bout due to a more pronounced accumulation of end-products and vasodilation. It can be hypothesized that the pronounced vasodilation that persists for several minutes in the supine position may cause a greater percentage of blood to pool in the legs, and thereby reduce cardiac preload.

4.3.2 Myocardial Contractility

It has been suggested that Wingate exercise may be too strenuous for older, untrained participants (Sagiv et al., 2005). After observing a reduction in myocardial activity following a single Wingate Sagiv *et al.,* (2005) hypothesized that aged myocardium and associated blood vessels are unable to increase contractility enough to overcome increased afterload pressures during Wingate exercise. During multiple Wingate exercise, it is possible that combining the high intensity intervals, with

incomplete recovery periods, caused cardiovascular drift over the 16 minutes of total exercise time (Rowell, L. 1974). This cardiovascular drift could be evaluated by decreases in SV over prolonged exercise and concurrent increases in HR to maintain a consistent CO (Rowell, L. 1974).

4.3.3 Afterload

Afterload is the pressure that exists in the aorta resisting blood flow ejected from the heart (Pouleur et al., 1982). As the body begins exercise there is a massive sympathetic outflow, increased contractility of the heart, increased respiration, and vasoconstriction of the blood vessels. This vasoconstriction of the blood vessels causes increased venous return to the heart, resulting in increased end diastolic volume, a stronger myocardial contraction, and less end systolic volume (Rowell, 1974). In the current investigation, the decrease in SV observed at two-minutes was accompanied by increases in HR, SBP, DBP and MAP; consequently, TPR was also reduced at the two-minute time point. It is reasonable to assume that the increase in MAP was caused by a massive increase in sympathetic activation, causing vasoconstriction in the non-working vascular bed. This vasoconstriction may have caused a spike in BP, and concurrent increase in end diastolic volume; however when combined with the increased exercising vascular vasodilation in the exercising vascular beds, accounted for decreased TPR.

Mortensen *et al.* (2005) found that healthy males exhibited an increase in MAP causing a decrease in SV from a maximal value of 160 ml/beat to 142 ml/beat as exercise neared maximal in an incremental exercise test to exhaustion (SV measured as the

quotient of CO derived by the Fick principle and HR). Similarly, Calbet *et al.* (2007) measured SV in nine healthy males with the dye-dilution method during a steady incremental exercise test to exhaustion, and observed a plateau in SV of 147 ml/beat at 64% of maximal workload, as measured in a graded exercise test to exhaustion. The difference in the two protocols suggests that if workload increased at uneven intervals then SV will decrease after CO max is reached. Although Mortensen *et al.* (2005) hypothesized that alterations in cardiac metabolism are a possible explanation for the decreases in SV. Calbet *et al.* (2007) suggest that the increased MAP observed by Mortensen *et al.* (2005) could have further affected SV through increases in afterload.

After a single Wingate, Doppler ultrasound SV measurements indicate that aortic flow is reduced below resting levels from 83 ml/min to 77 ml/min immediately after exercise (Ben-Sira & Sagiv, 1997). Crisafulli *et al.* (2004) measured SV through impedance cardiography while participants were seated and found that after five bouts of interval exercise at 150% of maximum power output SV was elevated above resting levels immediately after interval exercise, but returned to resting levels after three minutes of passive seated recovery.

4.3.4 Stroke Volume Conclusion

An increase in afterload is the most likely explanation for the immediate decrease in SV after multiple Wingate exercise observed in the current investigation, but there is also support for a decrease in blood or plasma volume affecting SV throughout the recovery period. It is likely that several mechanisms working together produced the SV

response observed in the current investigation. The transient increase in BP we observed had returned to resting levels at the 15-minute time point in the current study, and as soon as eight minutes in Hussain *et al.* (1996). The lack of valid measurement techniques for evaluating SV at maximal exercise, and the minimal research regarding the acute cardiovascular ramifications of supramaximal exercise on SV may explain why a SV decrease of this magnitude has not been previously documented in the literature.

4.4 Cardiac Output

In spite of the large decreases in SV documented at the two-minute time point, HR remained high enough to result in an increase to CO of 7.4 L/min at the two-minute time point compared to resting values of 5.1 L/min. When SV returned to resting values at the 15-minute time point, the corresponding CO was elevated to the maximum recorded post exercise value of 8.1 L/min.

Sagiv *et al.*, (2005) found that during a single Wingate, at peak anaerobic exercise, CO increased in healthy young males from 5.5 to 15.8 L/min. However, they also found that elderly males (mean age 58) were not able to increase CO to the same extent. Elderly males increased their CO from 5.1 L/min at rest to 9.8 L/min at maximal exercise (Sagiv et al., 2005), and the limited increase in CO was attributed to a decrease in left ventricular function and SV. Caution must be taken when comparing the results of the current investigation to those of Sagiv *et al.*, (2005), although our results seem to most parallel the changed observed in elderly males; our values were recorded after two-minutes of recovery in the supine position when HR was much lower (121 beats/minute

compared to 156 beats/minute in elderly males). The increases in CO to 15.8 L/min observed by Sagiv *et al.*, (2005) during a single Wingate in healthy young males is comparable the 14.5 L/min CO observed by Ben-Sira & Sagiv (1997) immediately following a single Wingate. The difference between the two studies is that Ben-Sira & Sagiv, (1997) observed a concurrent decrease in SV values as compared to rest and Sagiv *et al.*, (2005) observed a slight increase in young healthy males during Wingate exercise. Maximum HR in both studies was 188 and 187, and the decrease in SV observed immediately following single Wingate exercise may help to explain why the SV reported in the current investigation had such a pronounced negative effect on CO at the two-minute recovery time point following multiple Wingate exercise.

The results of both Ben-Sira & Sagiv (1997) and Sagiv *et al.* (2005) are contradictory to those reported by Crisafulli *et al.* (2004) who measured CO by impedance cardiography both during and after five 30 second intervals at 150% of the predetermined power to achieve VO_{2max}, separated by one-minutes of light cycling at 40 Watts. Crisafulli *et al.* (2004) observed that CO was increased approximately 11.5 L/min above their resting value of 5.0 L/min after exercise. The rapid decline of this value to approximately 7.5 L/min above the resting value (12.5 L/min) after two-minutes of light recovery cycling mainly attributed the rapid decline of both SV and HR in the first three minutes of recovery. Unfortunately it is difficult to compare these results to our study directly, as the participants in Crisafulli *et al.* (2004) underwent active cycling during their recovery period, whereas the participants in the current investigation recovered passively in the supine position.

Calbet *et al.* (2007) found that during exercise to exhaustion, blood flow to the trunk remained constant at approximately 5 L/min. They also indicated that as exercise progressed the proportional distribution of blood flow to the legs increased. This could explain why we observed a delayed increase in CO following multiple sprint exercise. If the legs were hyper-perfused during the activity, then the lungs, trunk and other limbs would be in a relatively ischemic state. This would explain why, as the effect of afterload on the heart diminished, CO increased so that it could maintain preferential flow to the legs, as well as address the continued metabolic demands of the other body tissues.

4.5 Leg Blood Flow

In the current investigation, CFA flow was increased relative to rest for 75 minutes following multiple Wingate exercise, while SFA flow continued to be elevated at the end of the 120-minute recovery period.

Blood flow is directly correlated to the metabolic conditions of the tissue (Baskurt & Meiselman, 2003), yet it can be hypothesized that the massive increase in leg circulation compared to rest is responsible for a simultaneous reduction in TPR. We have determined that after CO returned to resting values; blood flow to the legs remained significantly elevated for a prolonged period. This may be due to both the redirection of blood away from the organs at the onset of physical activity, the increases in exercising tissue microcirculation for oxygen delivery and metabolite removal, the increase in vascular circulation for thermoregulatory purposes, and the opening of collapsed arteries (Rotstein et al., 1982).

Immediately following multiple Wingate exercise, we observed an eight fold increase in two leg CFA blood flow from a resting value of 260 ml/min to 2072 ml/min (note that single leg blood flow values represented in the results of the current study are doubled to facilitate comparison with previous work). This elevation persisted for 75 minutes of supine recovery, and was no longer above resting values at R90. The CFA blood flow values immediately after multiple Wingate exercise in the current investigation were higher with respect to the magnitude and duration of increase, but similar in terms of absolute peak value, to those reported in similar studies after single Wingate exercise (Hussain et al., 1996). Hussain *et al.* (1996) examined CFA and SFA blood flow using Doppler ultrasound after a single Wingate in the supine position, and observed that CFA blood flow was elevated from a resting value of 314 ml/min to 2150 ml/min two-minutes after exercise. Peak CFA blood flow may have been similar in the untrained participants used in both studies, however the demand for increased blood flow to the leg lasts longer after multiple Wingate exercise, than after a single Wingate. This prolonged demand for flow is most likely attributed to the greater elevations in lactate and pH observed after multiple Wingates compared to single Wingate exercise (Appendix C).

In the current investigation we observed a six fold increase in SFA flow from 196 ml/min to 1232 ml/min. Both the peak absolute SFA value, and peak relative increase were larger in the current investigation than the values observed by Hussain *et al.* (1996) after a single Wingate. Hussain *et al.* (1996) observed a four fold increase in SFA blood flow from 219 ml/min to 1060 ml/min two-minutes after a single Wingate in the supine

position. Our results also indicate SFA flow is elevated for at least twice as long following multiple Wingate exercise compared to single Wingate exercise. The SFA blood flow values observed by Hussain *et al.* (1996) returned to resting values after 60 minutes of passive supine recovery, and in the current investigation SFA blood flow continued to be significantly elevated above resting values at the termination of our recovery period (R120). Since it is known that the PFA supplies the muscles of the thigh, and the SFA supplies the lower leg and skin (Hussain et al., 1996), it would be reasonable to assume that the increased flow to the SFA is most likely due to increased vascular flow associated with thermoregulation.

4.6 Single Wingate versus Multiple Wingate Exercise Comparison

4.6.1 Heart Rate

When comparing multiple Wingate exercise to single Wingate exercise, there are several aspects of particular interest. Both exercise durations elicited an increase in heart rate for the duration of the entire recovery period. When comparing the exercises directly, HR after multiple Wingate exercise was significantly elevated compared to HR following a single Wingate only for the first 45 minutes of recovery. This could indicate that once heart rates had decreased below 80 beats/minute, the influence of the parasympathetic and sympathetic system on the heart was similar. It is well established in the literature that the initial decline in HR at the termination of exercise is represented by parasympathetic reactivation (Cole et al., 2000; Dimpoulos et al., 2006; Savin, Davidson, & Haskell, 1982). Cole *et al.,* (2000) was able to attribute the entire 30-second

period directly after exercise to vagal reactivation, independent of age or exercise intensity. Sustained increases in HR are consistent with previous observations following high intensity interval programs (Mourot et al., 2004), and have been attributed to the slow withdrawal of the sympathetic tone back to resting levels.

Mourot *et al.,* (2004) observed that parasympathetic activity was depressed for a longer duration following nine one-minute intervals at the power output required to achieve their maximum ventilatory threshold, separated by four-minutes of cycling at a submaximal base workload, than it was after continuous exercise of an equal total workload and duration. The authors noted the presence of enhanced sympathetic tone following a maximal intensity interval exercise protocol that persisted for 60 minutes (Mourot et al., 2004).

Comparisons of three exercise types at similar energy expenditures (six minute repeated sprint exercise, 12 minute isocaloric high intensity exercise, and moderate continuous exercise) indicate that parasympathetic activation, measured by HR recovery, is significantly more delayed after repeated sprint exercise due to anaerobic contribution, and is not affected by net energy contribution (Buchheit, Laursen, & Ahmiaidi, 2007). Significantly elevated HR after multiple Wingate exercise is also indicative of increased sympathetic outflow following exercise of a longer duration.

4.6.2 Common Femoral Artery Blood Flow

The most striking comparison we are able to make between single versus multiple Wingate exercise is regarding leg blood flow. CFA blood flow was elevated above

resting values more after single Wingate activity than after multiple Wingate exercise (2528 ml/min after single Wingate exercise as compared to 2072 ml/min after multiple Wingate exercise). Interestingly, after only 15 minutes of recovery, blood flow through in the CFA was similar regardless of the duration of exercise. We have determined that part of the differences observed in CFA blood flow at the two-minute time point can be attributed to increases in whole body blood flow, as CO values following single Wingate exercise (7.8 L/min) are 400 ml higher then those observed after multiple Wingate exercise (7.4 L/min). Although the differences in CO after single and multiple Wingate exercise are not significantly different from each other, it is likely that the 400 ml difference in CO is a major contributor to 456 ml difference in CFA leg blood flow observed at the two-minute time point. We hypothesize that CFA blood flow during multiple Wingate exercise may have been higher during one of the early intervals versus the end of the forth interval due to increased sympathetic outflow and a decrease in power output as the intervals progressed. This pattern in leg blood flow may account for the differences observed between single and multiple Wingate exercise in the current investigation. With Doppler ultrasound it would be impossible to obtain CFA flow values during the exercise, without compromising the position of the participant, and their performance.

CFA blood flow following a single Wingate returned to resting values after 60 minutes of supine recovery, although in the current investigation we observed another transient increase at R90 as well. After multiple Wingate exercise CFA blood flow did not return to resting values until 75 minutes of recovery had passed. Compared to single

Wingate exercise, CFA blood flow after multiple Wingates is not as high, but takes a longer time to return to resting levels.

4.6.3 Superficial Femoral Artery Blood Flow

After single Wingate exercise, SFA blood flow is elevated for 45 minutes of recovery, while following multiple sprint activity it continued to be elevated at the end of the 120-minute recovery period. Since it is known that the PFA supplies the muscles of the thigh, and the SFA supplies the lower leg and skin (Hussain et al., 1996), it would be reasonable to assume that the increased flow to the SFA following the longer exercise is attributed to thermoregulation by cutaneous dissipation The amount of heat produced during a single Wingate is likely much less. It is also interesting to note that although the recovery patterns of SFA blood flow are different between the two activities, peak flow through the SFA at two-minutes post exercise are the same. This could indicate that there is a ceiling effect on SFA blood flow following high intensity exercise, regardless of the duration. Alternately it could indicate that more blood flow was being directed to the muscles of the thigh at this time after multiple Wingate exercise, so less was available to further increase SFA blood flow.

4.7 Limitations

Possible limitations of the present study include an inability to capture haemodynamic variables during the exercise activity. It can be assumed based on previous literature that HR, BP and blood flow would all increase during single and

multiple Wingate exercise. The exercise was performed in the seated position (as it normally would be in practical use), and a possible source of error could have occurred when the subject was being transferred from the bike to the bed. Without measurements taken on the bike as subjects were performing the activity, it is difficult to determine if the positional change from the bike to the bed affected the recovery pattern, and how so.

Following the first measurement time point at two-minutes post exercise, all other recovery measurements were separated by 15 minutes. This may have limited the study by precluding observations during the intervals between measurements. Hussain *et al.* (1996) observed a maximal decrease in DBP following seven minutes of supine recovery. Although in the current investigation we would have missed a transient decrease in BP such as this, it is not likely that hypotension that is sustained for less then 15 minutes would have any long-term benefits to the participant. Nevertheless, the purpose of this investigation was to accurately characterize the recovery period following multiple Wingate exercise, and the time between measurements may have prevented us from accurately satisfying this purpose.

In the PFA, we were unable to obtain accurate Doppler images and velocity tracings due to technical difficulties. Similar difficulties have been experiences by other researches that have attempted to use this artery in their analysis (Hussain et al., 1996). However, since we were able to obtain an visual images of the CFA dividing into the SFA and PFA we can be fairly certain that PFA can be estimated using a simple subtraction method.

During the measurement period, expired gasses were being collected. This may have altered the participant's natural respiratory patterns, and may have had a secondary effect on the cardiovascular system through alteration of the respiratory pump. As stroke velocity measurements were being collected at the sternal notch, a change in airflow pattern may have interfered with the stroke velocity readings.

4.8 Future Directions

The high force of contraction, and extremely high rate of contraction would make it very difficult to accurately evaluate leg blood flow during Wingate exercise, but it would be interesting to capture the rate of leg blood flow at maximal Wingate exertion, and in the acute recovery period. Likewise, it would be fascinating to track the changes in SV during multiple Wingate exercise. We were able to suggest several possibilities for the relatively large reduction in SV we observed at the two-minute time point, but these hypotheses should be confirmed using more direct methods of SV determination. Obtaining a stroke velocity signal using Doppler ultrasound within the first minute after exercise was not possible in this case due to the increased breathing rate.

Future studies should compare the recovery responses of sedentary and recreationally active participants, to the recovery of trained individuals. Previous reports indicate that trained individuals regulate heat dissipation and cardiovascular recovery differently then untrained individuals. It would be interesting to compare the different cardiovascular regulation and recovery patterns between these two groups.

Wingate exercise is extremely physically demanding exercise. It is not surprising that the recovery pattern following this activity would differ from submaximal exercise protocols. It may be beneficial to do a direct comparison between Wingate exercise and less intense exercise protocols. Perhaps the short-term supramaximal load being placed on the cardiovascular system during Wingate exercise is not the most beneficial activity for promoting cardiovascular fitness. The short term and long term cardiovascular risks and benefits of this intense activity need to be examined more closely in the future.

4.9 Conclusion

The purpose of the current investigation was to accurately characterize the acute recovery period following multiple Wingate exercise. As a secondary purpose, we also compared HR and leg blood flow following a single Wingate, to the responses following multiple Wingate exercise. We have determined that HR is increased at the cessation of multiple Wingate exercise, and declines smoothly towards resting values for the remainder of recovery and is higher than HR following single Wingate exercise for the first 45 minutes of recovery. CO is increased two-minutes following multiple Wingate exercise, and increases to a maximum recovery value at 15 minutes of recovery. SV exhibited a significant decrease immediately following exercise that we propose was attributed to increases in afterload. The decreases in SV observed later in the recovery period may be associated with decreases in plasma volume and preload. Immediately following multiple Wingate exercise DBP, SBP, and MAP were all elevated compared to rest. We believe that the increased SBP contributed to an increased afterload, resulting in

reduced SV. Although SBP and MAP returned, to resting values 15-minutes after multiple Wingate exercise, DBP continued to decrease below resting levels, resulting in a period of hypotension from 30 to 60 minutes of recovery. Lastly, as expected, leg blood flow was elevated following exercise. CFA blood flow was higher after a single Wingate, than after multiple Wingate exercise. This could indicate that sympathetic outflow during multiple Wingate exercise caused a greater amount of vasoconstriction regardless of metabolite build up, after multiple Wingate exercise as compared to single Wingate exercise. Peak SFA flow did not differ between the two exercises, although following multiple Wingate exercise the time to recovery was significantly longer than after single Wingate exercise. We attribute the sustained elevations in SFA blood flow to increased cutaneous flow for thermoregulation following exercise of a longer duration

We conclude that the supramaximal intensity of Wingate exercise, combined with the elongated duration during multiple bouts in a single exercise session presents a substantial challenge to the cardiovascular system as assessed by the recovery patterns of HR, BP, SV, CO, and leg blood flow. The nature of this intense activity causes increases in blood pressure, and fatigue on the heart such that SV cannot be maintained in the acute recovery period.

REFERENCES

Aardenburg, R., Spaanderman, M. E., van Eijndhoven, H. W., de Leeuw, P. W., & Peeters, L. L. (2005). Formerly preeclamptic omen with subnormal plasma volume are unable to maintain a rise in stroke volume during moderate exercise. *Journal of the Society for Gynecologic Investigation, 12*(8), 599-603.

Abbott, R.. D., Levy, D., Kannel, W. B., Castelli, W. P., Wilson, P. W. F., Garrison, R. J., & Stokes, J. (1989). Cardiovascular risk factors and graded treadmill exercise endurance in healthy adults: The Framingham offspring study. *American Journal of Cardiology, 63*, 342-346.

Adams, J., Ogola, G., Stafford, P., Koutras, P., & Hartman, J. (2006). High-intensity interval training for intermittent claudication in a vascular rehabilitation program. *Journal of Vascular Nursing, 24*, 46-49.

Albert, C. M., Mittleman, M. A., Chae, C. U., Lee, I. M., Hennekens, C. H., & Manson, J. E. (2000). Triggering of sudden death from cardiac causes by vigorous exertion. *The New England Journal of Medicine, 343*, 1355-1361.

Åstrand, I., Åstrand, P. O-., Christensen, E. H., & Hedman, R. (1960). Intermittent muscular work. *Acta Physiologica Scandinavia, 48*, 448-453.

Ayalon, A., Inbar, O,, Bar, Or, O. (1974). *Relationships among measurements of explosive strength and anaerobic power.* University Park Press, Baltimore. In Nelson & Morehouse International series on Sports Sciences, Vol. I, Bioamechanics IV, pp. 527-532,

Baskurt, O. K., & Meiselman, H. J. (2003) Blood Rheology and Hemodynamics. *Seminars in Thrombosis and Hemostasis, 29*(5), 435-450.

Beneke, R., Pollmann, C., Bleif, I., Leithäuser, R. M., & Hütler, M. (2002). How anaerobic is the wingate anaerobic test for humans? *European Journal of Applied Physiology, 87*, 388-392.

Ben-Sira, D. & Sagiv, M. (1997). The effect of gender on left ventricular function immediately after the wingate test. *European Journal of Applied Physiology, 75,* 549-553.

Blomqvist, C. G., & Saltin, B. (1983). Cardiovascular adaptations to physical training, *Annual Review of Physiology, 45,* 169-189.

Bond, V., Stephens, Q., Adams, R. G., Vaccaro, P., Demeersman, R., Williams, D. et al. (2002). Aerobic exercise attenuates an exaggerated blood pressure response in normotensive young adult African-American men. *Blood Pressure,* 11, 229-234.

Bortz, W. M. (1982). Disuse and aging. *JAMA, 248*(10), 1203-1208.

Buchheit, M., Laursen, P. B., & Ahmaidi, S. (2007). Parasympathetic reactivation after repeated sprint exercise. *American Journal of Physiology. Heart and Circulatory Physiology, 293*, H133-H141.

Burgomaster, K. A., Hughes, S. C., Heigenhaiser, G. J. F., Bradwell, S. N., & Gibala, M. J. (2005). Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. *Journal of Applied Physiology, 98*, 1985-1990.

Calbet, J. A. L., Gonzalez-Alonso, J., Helge, J. W., Søndergaard, H., Munch-Andersen, T., & Boushel, R. (2007). Cardiac output, and leg and arm blood flow during incremental exercise to exhaustion on the cycle ergometer. *Journal of Applied Physiology, 29*, in press.

Carter, R., Watenpaugh, D. E., & Smith, M. L. (2001). Gender differences in cardiovascular regulation during recovery from exercise. *Journal of Applied Physiology, (91)*, 1902-1907.

Carter, J. B., Banister, E. W., & Balber, A. P. (2003). Effect of endurance exercise on autonomic control of heart rate. *Sports Medicine, 33*(1), 33-46.

Cheatham, C. C., Mahon, A. D., Brown, J. D., & Bolster, D. R. (2000). Cardiovascular responses during prolonged exercise at ventilatory threshold in boys and men. *Medicine & Science in Sports & Exercise, 32*(3), 1080-1087.

Christensen, E. H., Hedman, R., & Saltin, B. (1960). Intermittent and continuous running. *Acta Physiologica Scandinavia, 50*, 269-286.

Clarke, D. H. (1973). Adaptations in strength and muscular endurance resulting from exercise. *Exercise and Sport Sciences Reviews, 1*, 73-102.

Cole, C. R., Foody, J. M., Blackstone, E. H., & Lauer, M. S. (2000). Heart rate recovery after submaximal exercise testing as a predictor of mortality in a cardiovascularly healthy cohort. *Annals of Internal Medicine, 132*(7), 552-555.

Cornelissen, V. A., & Fagard, R. H. (2004). Exercise intensity and post exercise hypotension. *Journal of Hypertension. 22*, 1859-1861. (editorial comment)

Costill, D. L., Flynn, M. G., Kirwan, J. P., Houmard, J. A., Mitchell, J. B., Thomas, R., & Park, S. H. (1988). Effects of repeated days of intensified training on muscle glycogen and swimming performance. *Medicine and Science in Sports and Exercise, 20*(3), 249-254.

Crisafulli, A., Carta, C., Melis, F., Tocco, F., Frongia, F., Santoboni, U. M. et al. (2004). Haemodynamic responses following intermittent supramaximal exercise in athletes. *Experimental Physiology, 89*(6), 665-674.

Dimopoulos, S., Anastasiou-Nana, M., Sakelleriou, D., Drakos, S., Kapsimalakou, S., Maroulidis, et al. (2006). Effects of exercise rehabilitation program on heart rate recovery in patients with chronic heart failure. *European Journal of Cardiovascular Prevention and Rehabilitation, 13*(1), 67-73.

Dujić, Ž., Ivančev, V., Valic, Z., Baković, D., Marinović-Terzić, I., Eterović, D. et al. (2006). Postexercise hypotension in moderately trained athletes after maximal exercise. *Medicine & Science in Sports & Exercise, 38*(2), 318-122.

Fitzgerald, W. (1981). Labile hypertension and jogging: a new diagnostic tool or spurious discovery? *British Medical Journal, 282*, 542-544.

Forjaz, C. L. M., Matsudaira, Y., Rodrigues, F. B., Nunes, N., & Negrão, C. E. (1998). Post-exercise changes in blood pressure, heart rate and rate pressure product at different exercise intensities in normotensive humans. *Brazilian Journal of medical and Biological Research, 31*, 1247-1255.

Forjaz,C. L. M., Tinucci, T., Ortega, K. C., Santaella, D. F., Mion, D. Jr., & Negrão, C. E. (2000). Factors affecting post-exercise hypotension in normotensive and hypertensive humans. *Blood Pressure Monitoring, 5*, 255-262.

Gibala, M. J., Little, J. P., van Essen, M., Wilkin, G. P., Burgomaster, K. A., Safdar, A., et al. (2006). Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. *The Journal of Physiology, 575*, 901-911.

Green, H., J., Jones, L. L., Hughson, L. R., Painter, D. C., & Farrance B. W. (1987). Training-induced hypervolemia: lack of an effect on oxygen utilization during exercise. *Medicine and Science in Sports and Exercise, 19*(3), 202-206.

Green, J. M., Mclester, J. R., Crews, T. R., Wickwire, P. J., Pritchett, R. C., & Lomax, R. G. (2006). RPE association with lactate and heart rate during high-intensity interval cycling. *Medicine & Science in Sports & Exercise, 38*(1), 167-172.

Hamer, M., (2006). The anti-hypertensive effects of exercise: integrating acute and chronic mechanisms. *Sports Medicine, 36*(2), 109-116.

Hayes, P. M., Lucas, J. C., & Shi, X. (2000). Importance of post-exercise hypotension in plasma volume restoration. *Acta Physiologica Scandinavia, 169*, 115-124.

Helgerud, J., Høydal, K., Wang, E., Karlsen, T., Berg, P., Bjerkaas, M., et al. (2007). Aerobic high-intensity intervals improve VO2max more than moderate training. *Medicine & Science in Sports & Exercise, 39*(4), 665-671.

Hickson, R. C., Bomze, H. A., & Holloszy, J. O. (1977). Linear increase in aerobic power induced by a strenuous program of endurance exercise. *Journal of Applied Physiology, 42*(3), 372-376.

Hounker, M., Halle, M., Keul, J. (1996). Structural and functional adaptations of the cardiovascular system by training. *International Journal of Sports Medicine, 17*(suppl 3), S164-172.

Hussain, S. T., Smith, R. E., Medbak, S., Wood, R. F., & Whipp, B. J. (1996). Haemodynamic and metabolic responses of the lower limb after high intensity exercise in humans. *Experimental Physiology, 81,* 173-187.

Ikai, M., & Fukunaga, T. (1970). A study on training effect of strength per unit cross-sectional area of muscle by means of ultrasonic measurement. *Internationale Zeitschrift für angewandte Physiologie, einschliesslich Arbeitsphysiologie, 28*(3),173-180.

Jacob, C., Zouhal, H., Prioux, J., Gratas-Delmarche, A., Bentué-Ferrer, D., & Delmarche, P. (2004). Effect of the intensity of training on catecholamine responses to supramaximal exercise in endurance-trained men. *European Journal of Applied Physiology, 91*(1), 35-40.

JNC 7 express: The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure (2003). No. 03-5233). USA: National Institutes of Health.

Krustrup, P., Hellsten, Y., & Bangsbo, J. (2004). Intense interval training enhances human skeletal muscle oxygen uptake in the initial phase of dynamic exercise at high but not at low intensities. *The Journal of Physiology, 559*(1), 335-345.

Laursen, P. B., & Jenkins D.G. (2002). The scientific basis for high-intensity interval training: optimizing training programs and maximizing performance in highly trained endurance athletes. *Sports Medicine, 32*(1), 53-73.

MacDonald, J. R. (2002). Potential causes, mechanisms, and implications of post exercise hypotension. *Journal of Human Hypertension, 16*, 225-236.

MacDonald, J. R., MacDougall, J. D., & Hogben, C. D. (1999). The effects of exercise intensity on post exercise hypotension. *Journal of Human Hypertension, 13*, 527-531.

MacDougall, J. D., Hicks, A. L., MacDonald, J. R., McKelvie, R. S., Green, H. J., & Smith, K. M. (1998). Muscle performance and exzymatic adaptations to sprint interval training. *The Journal of Physiology, 84*(6), 2138-2142.

Meyer, T., Kindermann, M., & Kindermann, W. (2004). Exercise programmes for patients with chronic heart failure. *Sports Medicine. 34*(14), 939-954.

Meyer, K., Samek, L., Schwaibold, M., Westbrook, S., Hajric, R., Beneke, R., et al. (1997). Interval training in patients with severe chronic heart failure: analysis and recommendations for exercise procedures. *Medicine & Science in Sports & Exericse, 29*(3), 306-312.

Mier, C. M., Domenick, M. A., Turner, N. S., & Wilmore, J. H. (1996). Changes in stroke volume and maximal aerobic capacity with increased blood volume in men and women. *Journal of Applied Physiology, 80*(4), 1180-1186.

Morout, L., Bouhaddi, M., Tordi, N., Rouilon, J. -D., & Regnard, J. (2004). Short- and long-term effects of a single bout of exercise on heart rate variability: comparison between constant and interval training exercises. *Eurpoean Journal of Applied Physiology, 92*, 508-517.

Morout, L., Tordi, N., Perrey, S., Bouhaddi, M., Rouilon, J. -D., & Regnard, J. (2005). Overall increase in heart rate variability after the square-wave endurance exercise test training. *Science & Sports, 20*(2), 83-90.

Mortensen, S. P., Dawson, E. A., Yoshiga, C. C., Dalsgaard, M. K., Damsgaard, R., Secher, N. H. (2005). Limitations to systemic and locomotor limb muscle oxygen delivery and uptake during maximal exercise in humans. *The Journal of Physiology, 566*(1), 273-285.

Nakahara, H., Miyamoto, T., Nakanishi, Y., & Kinoshita H. (2006). Time course of blood pressure changes immediately after maximal exercise. *Journal of Sports Medicine and Physical Fitness, 46,* 605-610.

Oberman, A., Fletcher, G. F., Lee, J., Nanda, N., Fletcher, B. J., Jensen, B., et al. (1995). Efficacy of high-intensity exercise training on left ventricular ejection fraction in men with coronary artery disease (the training level comparison study). *American Journal of Cardiology, 76*, 643-647.

Ogoh, S., Dalsgaard, M. K., Secher, N. H., & Raven, P. B. (2007). Dynamic blood pressure control and middle cerebral artery mean blood velocity variability at rest and during exercise in humans. *Acta Physiol.(Oxf),*(in press).

Parra, J., Cadefau, J. A. Rodas, G., Amigo, N., & Cusso, R. (2000). The distribution of rest periods affects performance and adaptations of energy metabolism induced by high-intensity training in human muscle. *Acta Physiologica Scandinavia, 169*, 157-165.

Pescatello, L. S., Fargo, A. E., Leach, C. N., & Scherzer, H. H. (1991). Short-term effect of dynamic exercise on arterial blood pressure. *Circulation, 83*, 1557-1561.

Petrella, R. J. (1998). How effective is exercise training for the treatment of hypertension? *Clinical Journal of Sports Medicine, 8*, 224-231.

Piepoli, M., Coats, A. J. S., Adamopoulos, S., Bernardi, L., Hong Feng, L., Cnway, J. et al. (1993). Persistant peripheral vasodilation and sympathetic activity in hypotension after maximal exercise. *Journal of Applied Physiology, 75*(4), 1807-1811.

Pouleur, H., Rousseau, M. F., van Eyll, C., Van Mechelen, H., Brasseur, L. A., & Charlier, A. A. (1982). Assessment of left ventricular contractility from late systolic stress-volume relations. *Circulation, 65*(6), 1204-1212.

Pricher, M. P., Holowatz, L. A., Williams, J. T., Lockwood, J. M., & Halliwill, J. R. (2004). Regional hemodynamics during postexercise hypotension. I. Splanchnic and renal circulations. *Journal of Applied Physiology, 97*, 2065-2070.

Rådegran, G., & Saltin, B. (2000). Human femoral artery diameter in relation to knee extensor muscle mass, peak blood flow, and oxygen uptake. *American Journal of Physiology: Heart and Circulation Physiology, 278*, 162-167.

Raine, N. M., Cable, N. T., George, K. P., & Campbell, I. G. (2001). The influence of recovery posture on post-exercise hypertension in normotensive men. *Medicine & Science in Sports & Exercise, 33*(3), 404-412.

Rotstein, A., Bar-Or, O., & Dlin, R. (1982). Hemoglobin, hematocrit, and calculated plasma volume changes induced by a short, supramaximal task. *International Journal of Sports Medicine, 3*(4), 230-233.

Rowell, L. B. (1974). Human cardiovascular adjustments to exercise and thermal stress. *Physiology Reviews, 54*(1), 75-159.

Rowell, L. B., Brengelmann, G. L., Blackmon, J. R., Bruce, R. A., & Murray, J. A. (1968). Disparities between aortic and peripheral pulse pressures induced by upright exercise and vasomotor changes in man. *Circulation, 37,* 954-964.

Sagiv, M., Ben-Sira, D., Sagiv, M., & Goldhammer, E. (2005). Left ventricular function at peak all-out anaerobic exercise in older men. *Gerontology, 51*, 122-125.

Saltin, B., Blomqvist, B., Mitchell, J. H., Jonson, R. L., Wildenthal, K., & Chapman, C. B. (1968). Response to submaximal and maximal exercise after bed rest and training. *Circulation, 37/38*(suppl VII), VII1–VII78.

Saltin, B., & Rowell, L. B. (1980). Functional adaptations to physical activity and inactivity. *Federation Proceedings, 39*(5), 1506-1513.

Savin, W.M., Davidson, D.M., & Haskell, W.L. (1982). Autonomic contribution to heart rate recovery from exercise in humans. *Journal of Applied Physioogy,* 53(6), 1572-1575.

Schibye, B., Mitchell, J. H., Payne, F. C., & Saltin, B. (1981). Blood pressure and heart rate response to static exercise in relation to electromyographic activity and foce development. *Acta physiologica Scandinavica, 113*(1), 61-66.

Seals, D. R., Hagberg, J. M. (1984). The effect of exercise training on human hypertension: A review. *Medicine and Science in Sports and Exercise, 16*(3), 207-215.

Sentürk, U. K., Yalçin, Ö., Gunduz, F., Kuru, O., Meiselman, H. J., & Baskurt, O. K. (2005). Effect of antioxidant vitamin treatment on the time course of hematological and hemorheological alterations after an exhausting episode of human subjects. *Journal of Applied Physiology, 98*, 1272-1279.

Sherman, S. E., D'Agostino, R. B., Cobb, J. L., & Cannel, W. B. (1994). Does exercise reduce mortality rates in the elderly? Experience from Framingham heart study. *American Heart Journal, 128*,965-972.

Smith T. P., McNaughton, L. R., & Marshall, K. J. (1999). Effects of 4-wk training using V_{max}/T_{max} on VO_{2max} and performance in athletes. *Medicine and Science in Sports and Exercise, 31*(6), 892-896.

Stratton, J. R., Levy, W. C., Cerqueira, M. D., Schwartz, R. S., & Abrass, I. B. (1994). Cardiovascular responses to exercise. Effects of aging and exercise training in healthy men. *Circulation, 89*, 1648-1655.

Tabata, I., Nishimura, K., Kouzaki, M., Hirai, Y., Ogita, F., Miyachi, M., & Yamamoto, K. (1996). Effects of moderate-intensity endurance and high-intensity intermittent training on anaerobic capacity and VO_{2max}. *Medicine and Science in Sports and Exercise, 28*(10), 1327-1330.

Takahashi, T., Hayano, J., Okada, A., Saitoh., T., & Kamiya, A. (2005). The effects of the muscle pump and body posture on cardiovascular responses during recovery from cycle exercise. *European Journal of Applied Physiology, 94*, 576-583.

Takahashi, T., Okada, A., Saitoh, T., Hayano, J., & Miyamoto, Y. (2000). Difference in human cardiovascular response between upright and supine recovery from upright cycle exercise. *European journal of Applied Physiology, 81*, 233-239.

Timson, B. F., Bowlin, B. K., Dudehoeffer, G. A., & George, J. B. (1985). Fiber number, area, and composition of mouse soleus muscle following enlargement. *Journal of Applied Physiology 58*(2), 619-624.

Warburton, D. E. R., McKenzie, D. C., Haykowsky, M. J., Taylor, A., Shoemaker P., Ignaszewski, A. P., et al. (2005). Effectiveness of high-intensity interval training for the rehabilitation of patients with coronary artery disease. *American Journal of Cardiology, 95*, 1080-1084.

Williams, J. T., Pricher, M. P., & Halliwill, J. R. (2005). Post exercise hypotension related to excess postexercise oxygen consumption through changes in leg blood flow. *Journal of Applied Physiology,98*, 1463-1468.

Woolf, C. R., Suero, J. T. (1969). Alterations in lung mechanics and gas exchange following training in chronic obstructive lung disease. *Chest, 55,* 37-44.

Yalcin, O., Erman, A., Muratli, S., Bor-Kucukatay, M., & Baskurt, O. K. (2003). Time course of hemorheological alterations after heavy anaerobic exercise in untrained human subjects. *Journal of Applied Physiology, 94*, 997-1002.

Zouhal, H., Jacob, C., Rannou, F., Gratas-Delamarche, A., Bentué-Ferrer, D., & Del, P. (2001). Effect of training status on the symapthosdrenal activity during a supramaximal exercise in human. *Journal of Sports Medicine and Physical Fitness, 41*, 330-336.

APPENDIX A
RAW DATA

Participant Characteristics

Subject	Age years	Height cm	Mass kg	V02 Max ml	V02 Max ml/kg
1	21.0	177.8	85.3	4350.5	51.0
2	20.0	180.0	70.0	3552.5	50.8
3	23.0	177.8	78.9	3289.5	41.7
4	21.0	190.5	85.5	3582.5	41.9
5	19.0	177.8	73.3	3871.0	52.8
6	19.0	175.3	81.2	3773.5	46.5
7	20.0	193.0	81.4	4225.0	51.9
8	18.0	185.4	66.5	3524.5	53.0
9	18.0	177.8	74.6	2667.5	35.8
10	19.0	175.3	80.2	3618.5	45.1
Mean	**19.8**	**181.1**	**77.7**	**3645.5**	**47.0**
SD	1.2	2.5	2.5	21.7	2.4
SEM	0.4	0.8	0.8	6.9	0.8

Exercise Characteristics

	Single Wingate		
	Mean	Peak	Fatigue
Subject	Watts	Watts/s	%
1	764.0	1300	32.5
2	633.5	1043	23.6
3	704.4	1001	20.8
4	703.5	1213	27.9
5	630.4	827	12.3
6	636.3	967	18.3
7	677.6	1140	24.0
8	528.6	931	20.3
9	618.8	1011	26.0
10	683.5	1094	28.6
Mean	**658.1**	**1053**	**23.4**
SD	8.0	11.8	2.4
SEM	2.7	0.5	0.1

Subject	Multiple Wingate – Sprint 1			Multiple Wingate – Sprint 2			Multiple Wingate – Sprint 3			Multiple Wingate – Sprint 4		
	Mean Watts	Peak Watts/s	Fatigue %	Mean Watts	Peak Watts/s	Fatigue %	Mean Watts	Peak Watts/s	Fatigue %	Mean Watts	Peak Watts/s	Fatigue %
1	809.9	1469	36.7	748.0	1419	40.1	655.6	1332	37.4	639.1	1175	35.3
2	653.8	1097	26.4	591.4	1121	32.4	547.7	873	21.6	531.5	915	27.9
3	676.4	903	13.7	632.4	730	16.0	418.7	539	14.3	388.3	518	8.8
4	684.9	1222	29.6	514.6	1066	27.6	413.4	849	22.0	365.2	502	10.7
5	628.6	882	15.1	615.0	897	15.8	573.3	883	19.9	536.3	789	16.6
6	637.0	1014	18.7	558.4	881	16.7	515.6	871	18.9	500.6	829	16.6
7	688.6	1134	23.2	607.0	1085	25.6	547.5	1036	22.9	521.4	993	25.1
8	620.2	842	15.4	547.9	917	17.7	474.2	605	10.7	454.5	724	14.6
9	631.0	995	23.5	478.9	722	18.2	430.4	663	16.2	392.1	737	16.8
10	677.8	1152	32.2	466.2	1028	29.3	374.6	879	25.8	397.9	901	25.2
Mean	**670.8**	**1071**	**23. 5**	**576.0**	**987**	**23.9**	**495.1**	**853**	**21.0**	**472.7**	**808**	**19.8**
SD	7.4	13.7	2.8	9.1	14.4	2.9	9.4	15.0	2.7	9.4	14.3	2.9
SEM	1.5	0.5	0.1	1.9	0.7	0.1	3.0	0.6	0.1	1.7	0.5	0.1

Heart Rate – Multiple Wingate Exercise

Subject	Rest	R2		R15	R30	R45		R60	R75	R90		R105	R120
1	45	105		98	89	75		71	54	59		56	46
2	57	111		86	80	70		69	62	63		60	59
3	42	96		86	71	64		59	62	60		58	53
4	54	122		106	92	94		87	80	80		72	70
5	52	112		94	85	66		66	59	58		56	53
6	58	115		92	87	82		77	67	69		74	74
7	68	120		105	106	104		93	88	87		87	79
8	75	148		111	102	95		89	78	85		79	81
9	64	159		132	108	101		87	65	75		72	70
10	63	137		114	102	96		89	75	65		69	73
Mean	**58**	**122**		**103**	**92**	**85**		**78**	**69**	**70**		**68**	**66**
SD	10	20		14	12	15		12	11	11		10	12
SEM	3	6		5	4	5		4	3	3		3	4

Systolic Blood Pressure – Multiple Wingate Exercise

Subject	Rest	R2		R15	R30	R45		R60	R75	R90		R105	R120
1	122	130		125	125	117		120	124	124		124	121
2	111	164		107	103	107		104	111	105		106	113
3	119	154		125	119	119		118	118	117		119	121
4	113	161		136	119	126		111	126	111		115	114
5	117	151		124	129	122		120	117	112		112	113
6	128	159		120	113	117		128	130	121		122	128
7	112	163		132	127	120		132	134	125		125	124
8	131	152		104	101	125		115	120	121		123	117
9	128	163		134	127	123		126	133	128		122	126
10	126	148		127	125	129		115	119	117		115	116
Mean	**121**	**155**		**123**	**119**	**121**		**119**	**123**	**118**		**118**	**119**
SD	7	10		11	10	6		8	8	7		6	5
SEM	2	3		3	3.	2		3	2	2		2	2

Diastolic Blood Pressure – Multiple Wingate Exercise

Subject	Rest	R2		R15		R30		R45		R60		R75		R90		R105		R120	
1	61	59		57		61		63		56		65		59		62		62	
2	66	80		65		64		64		64		61		63		64		65	
3	64	76		66		66		69		63		65		65		61		65	
4	65	82		67		63		65		67		70		65		65		67	
5	63	62		58		59		55		59		57		55		57		65	
6	69	77		62		60		66		66		67		65		66		69	
7	61	73		63		58		60		65		67		69		65		66	
8	75	72		55		53		69		66		72		71		70		69	
9	69	72		66		59		65		66		70		67		66		69	
10	73	80		66		65		67		67		67		71		69		72	
Mean		**67**	**73**		**63**		**61**		**64**		**64**		**66**		**65**		**65**		**67**
SD		5	8		4		4		4		4		4		5		4		3
SEM		2	2		1		1		1		1		1		2		1		1

Mean Arterial Blood Pressure – Multiple Wingate Exercise

Subject	Rest	R2		R15		R30		R45		R60		R75		R90		R105		R120	
1	81	83		80		82		81		77		85		81		83		82	
2	81	108		79		77		78		77		78		77		78		81	
3	82	102		86		84		86		81		83		82		80		84	
4	81	108		90		82		85		82		89		80		82		83	
5	81	92		80		82		77		79		77		74		75		81	
6	89	104		81		78		83		87		88		84		85		89	
7	78	103		86		81		80		87		89		88		85		85	
8	94	99		71		69		88		76		88		88		88		85	
9	89	102		89		82		84		86		91		87		85		88	
10	91	103		86		85		88		83		84		86		84		87	
Mean		**85**	**100**		**83**		**80**		**83**		**82**		**85**		**83**		**82**		**84**
SD		5	8		6		5		4		4		5		5		4		3
SEM		2	2		2		1		1		1		2		1		1		1

Stroke Volume – Multiple Wingate Exercise

Subject	Rest	R2	R15	R30	R45	R60	R75	R90	R105	R120
1	89.9	78.5	61.8	69.7	82.7	84.3	118.7	109.3	109.6	95.7
2	67.2	42.4	62.3	51.7	54.1	59.9	50.9	51.0	60.6	54.2
3	87.3	63.5	101.6	90.2	94.9	91.0	111.8	73.0	89.7	93.1
4	63.1	60.1	87.4	83.0	62.9	62.5	64.1	62.1	57.8	69.8
5	66.5	69.3	70.1	51.3	72.4	68.8	64.0	60.8	71.5	70.4
6	101.7	46.2	77.1	66.8	57.2	52.5	58.7	58.2	65.5	67.0
7	83.5	40.1	73.6	63.2	55.7	95.2	85.8	85.0	81.5	56.7
8	91.6	37.8	76.1	71.1	54.9	49.1	64.5	70.2	76.2	74.8
9	119.3	87.6	101.3	92.7	86.6	103.6	120.4	110.4	107.1	120.7
10	102.3	74.8	70.0	79.8	75.6	81.5	91.2	94.4	74.8	92.0
Mean	**87.2**	**60.0**	**78.1**	**72.0**	**69.7**	**74.8**	**83.0**	**77.4**	**79.4**	**79.4**
SD	18.0	17.7	14.3	14.5	14.9	18.9	26.4	21.3	17.9	20.6
SEM	5.7	5.6	4.5	4.6	4.7	6.0	8.3	6.8	5.7	6.5

Cardiac Output – Multiple Wingate Exercise

Subject	Rest	R2	R15	R30	R45	R60	R75	R90	R105	R120
1	4.1	8.2	6.1	6.2	6.2	6.0	6.4	6.4	6.1	4.4
2	3.8	4.7	5.4	4.1	3.8	4.1	3.2	3.2	3.7	3.2
3	3.6	6.1	8.7	6.4	6.1	5.4	6.9	4.4	5.2	5.0
4	3.4	7.3	9.2	7.7	5.9	5.4	5.1	4.9	4.2	4.9
5	3.4	7.7	6.6	4.3	4.8	4.5	3.8	3.5	4.0	3.7
6	5.9	5.3	7.1	5.8	4.7	4.0	3.9	4.0	4.8	5.0
7	5.7	4.8	7.7	6.7	5.8	8.9	7.5	7.4	7.1	4.5
8	6.8	5.6	8.5	7.2	5.2	4.4	5.0	6.0	6.0	6.1
9	7.6	13.9	13.4	10.0	8.8	9.0	7.9	8.3	7.7	8.5
10	6.4	10.3	8.0	8.2	7.3	7.2	6.9	6.1	5.2	6.7
Mean	**5.1**	**7.4**	**8.1**	**6.7**	**5.8**	**5.9**	**5.7**	**5.4**	**5.4**	**5.2**
SD	1.6	2.9	2.2	1.7	1.4	1.9	1.7	1.7	1.3	1.5
SEM	0.5	0.9	0.7	0.5	0.4	0.6	0.5	0.5	0.4	0.5

Total Peripheral Resistance – Multiple Wingate Exercise

Subject	Rest	R2	R15	R30	R45	R60	R75	R90	R105	R120
1	19.9	10.1	13.2	13.3	13.1	13.0	13.3	12.6	13.5	18.5
2	21.3	22.9	14.7	18.6	20.7	18.8	24.5	24.0	21.3	25.5
3	22.7	16.8	9.8	13.0	14.1	15.2	12.0	18.9	15.4	16.9
4	23.8	14.8	9.7	10.7	14.4	15.1	17.4	16.3	19.6	16.9
5	23.6	11.8	12.1	18.9	16.2	17.6	20.3	21.0	18.8	21.8
6	15.2	19.7	11.5	13.3	17.7	21.5	22.3	21.0	17.5	17.8
7	13.2	21.3	11.1	12.1	13.9	9.9	11.9	11.9	12.0	18.9
8	13.7	17.7	8.4	9.6	16.8	18.9	17.5	14.7	14.6	14.0
9	11.7	7.3	6.6	8.1	9.6	9.6	11.6	10.5	11.0	10.4
10	14.1	10.0	10.8	10.4	12.1	11.4	12.3	14.1	16.2	13.0
Mean	**17.9**	**15.2**	**10.8**	**12.8**	**14.9**	**15.1**	**16.3**	**16.5**	**16.0**	**17.4**
SD	4.8	5.3	2.3	3.6	3.1	4.1	4.8	4.5	3.3	4.3
SEM	1.5	1.7	0.7	1.1	1.0	1.3	1.5	1.4	1.1	1.4

Common Femoral Artery Blood Flow – Multiple Wingate Exercise

Subject	Rest	R2	R15	R30	R45	R60	R75	R90	R105	R120
1	172		529	754	480	391	210	186	200	163
2	96	1191	380	272	256	243	206	86	114	143
3	27	839	585	266	184	31	121	132	167	140
4	101	829	691	462	419	395	482	167	228	198
5	142	954	536	313	146	151	208	123	100	74
6	106	952	708	475	474	268	223	183	107	212
7	183	1033	714	0	530	510	327	167	234	186
8	181	824	574	349	274	193	249	220	134	171
9	111	1061	755	311	488	362	321	284	177	258
10	179	1644	1181	732	456	377	297	170	250	207
Mean	**130**	**1036**	**665**	**394**	**371**	**292**	**264**	**172**	**171**	**175**
SD	51	258	213	225	141	142	99	54	56	50
SEM	16	86	68	71	45	45	31	17	18	16

Superficial Femoral Artery Blood Flow – Multiple Wingate Exercise

Subject	Rest	R2	R15	R30	R45	R60	R75	R90	R105	R120
1	115	729	696	412	341	295	217	225	168	155
2	46		194	111	85	92	79	61	61	68
3	76	564	354	244	187	167	118	141	172	176
4	128	693	598	482	535	302	250	234	223	197
5	85	832	484	272	214	194	214	168	185	138
6	60	267	243	151	149	123	75	57	43	50
7	143	595	491	356	320	297	206	141	162	138
8	107	550	282	236	207	155	139	121	119	89
9	97	753	509	323	298	239	185	177	141	125
10	119	559	590	411	259	202	162	146	165	165
Mean	**98**	**616**	**444**	**300**	**260**	**207**	**165**	**147**	**144**	**130**
SD	31	165	168	119	125	75	60	59	56	48
SEM	10	55	53	38	39	24	19	19	18	15

Heart Rate – Single Wingate Exercise

Subjects	Rest	R2	R15	R30	R45	R60	R75	R90	R105	R120
1	39	84	81	66	57	44	36	48	44	42
2	57	94	69	66	67	66	59	61	57	59
3	46	94	73	70	62	64	70	62	69	65
4	55	112	101	89	75	95	66	68	71	78
5	52	71	61	61	62	60	55	55	57	54
6	67	106	88	82	74	73	65	68	64	70
7	52	112	98	92	83	78	66	72	68	68
8	66	112	96	92	80	79	76	80	78	78
9	63	141	112	103	85	72	67	70	69	63
10	67	124	114	96	86	89	70	76	68	72
Mean	**56**	**105**	**90**	**82**	**73**	**72**	**63**	**66**	**65**	**65**
SD	9	20	18	15	11	15	11	10	10	11
SEM	3	6	6	5	3	5	4	3	3	4

Stroke Volume – Single Wingate Exercise

Subjects	Rest	R2	R15	R30	R45	R60	R75	R90	R105	R120	R135
1	85.3	74.2	74.4	58.6	72.9	70.6	88.5	74.0	92.3	70.4	88.0
2	82.9	80.9	71.8	71.3	80.7	73.9	45.7	59.6	61.2	66.5	46.7
3	98.6	87.7	90.9	61.0	57.8	63.7	63.8	76.1	77.2	59.8	74.0
4	68.2	68.5	80.5	69.2	58.4	69.0	50.0	63.6	59.1	64.8	57.8
5	94.2	119.5	102.2	53.3	92.6	58.3	56.5	49.6	60.3	54.0	76.7
6	87.5	52.1	62.8	60.1	63.7	76.2	42.4	55.0	44.6	63.7	52.0
7	78.0	59.3	89.9	79.6	86.7	81.6	77.8	83.9	88.8	94.1	100.0
8	82.3	59.4	67.9	62.3	64.0	54.5	58.1	72.7	60.8	60.6	43.3
9	95.8	93.5	104.0	84.7	95.0	118.2	116.7	117.0	116.1	116.5	120.2
10	86.3	61.0	102.6	80.5	73.8	75.2	90.9	90.2	94.6	88.3	102.2
Mean	**85.9**	**75.6**	**84.7**	**68.1**	**74.6**	**74.1**	**69.0**	**74.2**	**75.5**	**73.9**	**76.1**
SD	9.0	20.4	15.3	10.7	13.8	17.6	23.9	19.7	22.0	19.6	26.3
SEM	2.8	6.5	4.8	3.4	4.3	5.6	7.5	6.2	7.0	6.2	8.3

Cardiac Output – Multiple Wingate Exercise

Subjects	Rest	R2	R15	R30	R45	R60	R75	R90	R105	R120	R135
1	3.3	6.2	6.0	3.9	4.1	3.1	3.2	3.6	4.0	2.9	3.2
2	4.7	7.6	4.9	4.7	5.4	4.8	2.7	3.7	3.5	3.9	2.6
3	4.5	8.2	6.7	4.3	3.6	4.1	4.5	4.7	5.3	3.9	4.8
4	3.7	7.6	8.1	6.2	4.4	6.6	3.3	4.3	4.2	5.0	4.1
5	4.9	8.5	6.3	3.3	5.7	3.5	3.1	2.7	3.4	2.9	4.4
6	5.8	5.5	5.5	4.9	4.7	5.5	2.8	3.7	2.9	4.5	3.1
7	4.1	6.6	8.8	7.3	7.2	6.4	5.2	6.1	6.0	6.4	6.3
8	5.4	6.7	6.5	5.7	5.1	4.3	4.4	5.8	4.8	4.8	2.9
9	6.0	13.1	11.6	8.7	8.1	8.5	7.8	8.2	8.0	7.4	7.5
10	5.7	7.6	11.7	7.7	6.3	6.7	6.4	6.9	6.4	6.3	6.8
Mean	**4.8**	**7.8**	**7.6**	**5.7**	**5.5**	**5.4**	**4.3**	**5.0**	**4.9**	**4.8**	**4.6**
SD	0.9	2.1	2.4	1.8	1.4	1.7	1.7	1.7	1.6	1.5	1.8
SEM	0.3	0.7	0.8	0.6	0.4	0.5	0.5	0.5	0.5	0.5	0.6

Common Femoral Artery Blood Flow – Single Wingate Exercise

Subject	Rest	R2	R15	R30	R45	R60	R75	R90	R105	R120
1	81	713	598	318	193	98		158	100	134
2	115	1354	657	114	188	92	180	73	132	233
3	65		761	456	214	105		174	117	101
4	124	1203	520	502	143	167	167	123	173	70
5	102	823	374	214	108	69	141	238		172
6	219	1106	570	279	246	157	233	194	160	146
7	220	1533	1240	517	518	308	297	134	170	203
8	212	1537	836	339	252	148	273	212	184	178
9	125	1564	708	386	400	266	251	154	222	195
10	157	1544	1354	867	347	263	233	280	194	207
Mean	**142**	**1264**	**762**	**399**	**261**	**167**	**222**	**174**	**161**	**164**
SD	57	326	311	207	126	84	54	60	39	51
SEM	18	109	98	66	40	26	19	19	13	16

Superficial Femoral Artery Blood Flow – Single Wingate Exercise

Subject	Rest	R2	R15	R30	R45	R60	R75	R90	R105	R120
1	74	712	350	232	123	117		68	94	47
2	70	384	174	79	60	58	49	64	76	59
3	49		248	121	95	74		55	49	32
4	59	829	353	233	140	94	69	39	79	112
5	107	630	266	105	139	82	67	83	92	44
6	86	229	194	132	74	75	57	56	47	63
7	100	588	388	228	145	158	106	89	73	98
8	74	452	238	117	84	72	67	67	58	74
9	67	811	371	282	160	141	128	100	130	91
10	148	578	470	333	221	191	134	116	87	131
Mean	**83**	**579**	**305**	**186**	**124**	**106**	**85**	**74**	**79**	**75**
SD	29	198	95	86	48	44	33	23	25	32
SEM	9	66	30	27	15	14	12	7	8	10

APPENDIX B
STATISTICAL TABLES

Exercise Characteristics – Mean Power Output
Normality Test: Passed (P = 0.590)
Equal Variance Test: Passed (P = 0.587)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	176083.345	19564.816		
Between Treatments	4	329316.380	82329.095	33.463	<0.001
Residual	36	88570.500	2460.292		
Total	49	593970.225			

Exercise Characteristics – Peak Power Output
Normality Test: Passed (P = 0.246)
Equal Variance Test: Passed (P = 0.907)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	1335695.680	148410.631		
Between Treatments	4	559224.280	139806.070	13.258	<0.001
Residual	36	379614.920	10544.859		
Total	49	2274534.880			

Exercise Characteristics – Fatigue Index
Normality Test: Passed (P = 0.500)
Equal Variance Test: Passed (P = 0.595)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	2105.229	233.914		
Between Treatments	4	135.090	33.773	2.534	0.057
Residual	36	479.706	13.325		
Total	49	2720.025			

Heart Rate – Multiple Wingate Exercise
Normality Test: Passed (P = 0.150)
Equal Variance Test: Passed (P = 0.178)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	11505.236	1278.360		
Between Treatments	9	35264.608	3918.290	83.181	<0.001
Residual	81	3815.554	47.106		
Total	99	50585.398			

Systolic Blood Pressure – Multiple Wingate Exercise
Normality Test: Failed (P < 0.050)
Equal Variance Test: Passed (P = 0.395)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	2152.217	239.135		
Between Treatments	9	10962.567	1218.063	25.741	<0.001
Residual	81	3832.953	47.320		
Total	99	16947.737			

Wilcoxon Matched Pairs Test

	Valid N	T	Z	p-level
REST & R0_5	10	0.00000	2.803060	.005065
REST & R15_20	10	20.00000	.764471	.444592
REST & R30_35	10	24.00000	.356753	.721279
REST & R45_50	10	25.00000	.254824	.798861
REST & R60_65	10	16.00000	1.172189	.241130
REST & R75_80	10	16.00000	.770054	.441274
REST & R90_95	10	14.50000	1.325083	.185153
REST & R105_110	10	15.00000	1.274118	.202631
REST & R120_125	10	22.50000	.509647	.610302

Diastolic Blood Pressure – Multiple Wingate Exercise
Normality Test: Passed (P = 0.159)
Equal Variance Test: Passed (P = 0.059)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	962.558	106.951		
Between Treatments	9	998.976	110.997	8.898	<0.001
Residual	81	1010.469	12.475		
Total	99	2972.002			

Mean Arterial Pressure – Multiple Wingate Exercise
Normality Test: Failed (P < 0.050)
Equal Variance Test: Passed (P = 0.218)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	738.454	82.050		
Between Treatments	9	2883.041	320.338	18.136	<0.001
Residual	81	1430.685	17.663		
Total	99	5052.180			

Wilcoxon Matched Pairs Test

	Valid N	T	Z	p-level
REST & R0_5	10	0.00000	2.803060	.005065
REST & R15_20	10	19.00000	.414644	.678405
REST & R30_35	10	15.00000	1.274118	.202631
REST & R45_50	10	15.00000	1.274118	.202631
REST & R60_65	10	10.00000	1.783765	.074471
REST & R75_80	10	27.00000	.050965	.959354
REST & R90_95	10	10.00000	1.783765	.074471
REST & R105_110	10	13.00000	1.477977	.139424
REST & R120_125	10	22.00000	.059235	.952765

Stroke Volume – Multiple Wingate Exercise
Normality Test: Passed (P = 0.380)
Equal Variance Test: Passed (P = 0.257)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	20907.321	2323.036		
Between Treatments	9	5181.758	575.751	4.308	<0.001
Residual	81	10824.441	133.635		
Total	99	36913.520			

Cardiac Output – Multiple Wingate Exercise
Normality Test: Failed (P < 0.050)
Equal Variance Test: Passed (P = 0.230)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	212.486	23.610		
Between Treatments	9	90.274	10.030	8.582	<0.001
Residual	81	94.674	1.169		
Total	99	397.435			

Wilcoxon Matched Pairs Test

	Valid N	T	Z	p-level
REST & R0_5	10	7.00000	2.089554	.036666
REST & R15_20	10	0.00000	2.803060	.005065
REST & R30_35	10	1.00000	2.701130	.006914
REST & R45_50	10	12.00000	1.579906	.114138
REST & R60_65	10	15.00000	1.274118	.202631
REST & R75_80	10	18.00000	.968330	.332887
REST & R90_95	10	20.00000	.764471	.444592
REST & R105_110	10	20.00000	.764471	.444592
REST & R120_125	10	23.00000	.458682	.646465

Total Peripheral Resistance – Multiple Wingate Exercise
Normality Test: Passed (P = 0.248)
Equal Variance Test: Passed (P = 0.208)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	949.686	105.521		
Between Treatments	9	408.967	45.441	6.429	<0.001
Residual	81	572.525	7.068		
Total	99	1931.177			

Common Femoral Artery Blood Flow – Multiple Wingate Exercise
Normality Test: Failed (P < 0.050)
Equal Variance Test: Passed (P = 0.342)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	663736.774	73748.530		
Between Treatments	9	6811845.005	756871.667	44.423	<0.001
Residual	81	1380075.139	17037.965		
Total	99	8855656.919			

Wilcoxon Matched Pairs Test

	Valid N	T	Z	p-level
REST & R0_5	9	0.00000	2.665570	.007690
REST & R15_20	10	0.00000	2.803060	.005065
REST & R30_35	10	4.00000	2.395342	.016611
REST & R45_50	10	0.00000	2.803060	.005065
REST & R60_65	10	0.00000	2.803060	.005065
REST & R75_80	10	0.00000	2.803060	.005065
REST & R90_95	10	12.00000	1.579906	.114138
REST & R105_110	10	9.00000	1.885695	.059345
REST & R120_125	10	11.00000	1.681836	.092610

Superficial Femoral Artery Blood Flow – Multiple Wingate Exercise
Normality Test: Failed (P < 0.050)
Equal Variance Test: Failed (P < 0.050)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	574182.694	63798.077		
Between Treatments	9	2191759.962	243528.885	58.017	<0.001
Residual	80	335803.780	4197.547		
Total	98	3202573.880	32679.325		

Wilcoxon Matched Pairs Test

	Valid N	T	Z	p-level
REST & R0_5	9	0.000000	2.665570	.007690
REST & R15_20	10	0.000000	2.803060	.005065
REST & R30_35	10	0.000000	2.803060	.005065
REST & R45_50	10	0.000000	2.803060	.005065
REST & R60_65	10	0.000000	2.803060	.005065
REST & R75_80	10	0.000000	2.803060	.005065
REST & R90_95	10	3.000000	2.497271	.012520
REST & R105_110	10	3.000000	2.497271	.012520
REST & R120_125	10	6.000000	2.191483	.028424

Heart Rate – Single Wingate Exercise
Normality Test: Failed (P < 0.050)
Equal Variance Test: Passed (P = 0.184)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	11714.172	1301.575		
Between Treatments	9	19190.775	2132.308	38.820	<0.001
Residual	81	4449.141	54.928		
Total	99	35354.088			

Wilcoxon Matched Pairs Test

	Valid N	T	Z	p-level
REST & R0_5	10	0.000000	2.803060	.005065
REST & R15_20	10	0.000000	2.803060	.005065
REST & R30_35	10	0.000000	2.803060	.005065
REST & R45_50	10	0.000000	2.803060	.005065
REST & R60_65	10	0.000000	2.803060	.005065
REST & R75_80	10	5.000000	2.293412	.021831
REST & R90_95	10	0.000000	2.803060	.005065
REST & R105_110	10	4.000000	2.395342	.016611
REST & R120_125	10	0.000000	2.803060	.005065

Stroke Volume – Single Wingate Exercise
Normality Test: Passed (P = 0.630)
Equal Variance Test: Passed (P = 0.051)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	15264.002	1696.000		
Between Treatments	9	2973.203	330.356	2.015	0.048
Residual	81	13281.657	163.971		
Total	99	31518.863			

Cardiac Output – Multiple Wingate Exercise
Normality Test: Passed (P = 0.263)
Equal Variance Test: Passed (P = 0.693)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	193.466	21.496		
Between Treatments	9	126.712	14.079	15.225	<0.001
Residual	81	74.901	0.925		
Total	99	395.078			

Common Femoral Artery Blood Flow – Single Wingate Exercise

Normality Test: Failed (P < 0.050)

Equal Variance Test: Failed (P < 0.050)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	967738.139	107526.460		
Between Treatments	9	11148219.028	1238691.003	65.215	<0.001
Residual	77	1462525.922	18993.843		
Total	95	13595303.826	143108.461		

Wilcoxon Matched Pairs Test

	Valid N	T	Z	p-level
REST & R0_5	9	0.00000	2.665570	.007690
REST & R15_20	10	0.00000	2.803060	.005065
REST & R30_35	10	1.00000	2.701130	.006914
REST & R45_50	10	0.00000	2.803060	.005065
REST & R60_65	10	18.00000	.968330	.332887
REST & R75_80	8	0.00000	2.520504	.011724
REST & R90_95	10	18.00000	.968330	.332887
REST & R105_110	9	17.00000	.651584	.514674
REST & R120_125	10	18.00000	.968330	.332887

Superficial Femoral Artery Blood Flow – Single Wingate Exercise

Normality Test: Failed (P < 0.050)

Equal Variance Test: Failed (P < 0.050)

Source of Variation	DF	SS	MS	F	P
Between Subjects	9	220751.771	24527.975		
Between Treatments	9	2109934.200	234437.133	58.359	<0.001
Residual	78	313336.567	4017.135		
Total	96	2686182.216	27981.065		

Wilcoxon Matched Pairs Test

	Valid N	T	Z	p-level
REST & R0_5	9	0.00000	2.665570	.007690
REST & R15_20	10	0.00000	2.803060	.005065
REST & R30_35	10	1.00000	2.701130	.006914
REST & R45_50	10	4.00000	2.395342	.016611
REST & R60_65	10	11.00000	1.681836	.092610
REST & R75_80	8	12.00000	.840168	.400820
REST & R90_95	10	11.00000	1.681836	.092610
REST & R105_110	10	24.00000	.356753	.721279
REST & R120_125	10	16.00000	1.172189	.241130

Heart Rate – Comparison
Normality Test: Failed ($P < 0.050$)
Equal Variance Test: Passed ($P = 0.447$)

Source of Variation	DF	SS	MS	F	P
Subject	9	21771.310	2419.034		
Trial	1	2815.212	2815.212	17.497	0.002
Trial x Subject	9	1448.098	160.900		
Time	9	53108.333	5900.926	75.902	<0.001
Time x Subject	81	6297.230	77.744		
Trial x Time	9	1347.050	149.672	6.162	<0.001
Residual	81	1967.465	24.290		
Total	199	88754.698	446.004		

Wilcoxon Matched Pairs Test

	Valid N	T	Z	p-level
REST & REST	10	26.00000	.152894	.878483
R2 & R2	10	0.00000	2.803060	.005065
R15_20 & R15_20	10	0.00000	2.803060	.005065
R30_35 & R30_35	10	0.00000	2.803060	.005065
R45_50 & R45_50	10	0.00000	2.803060	.005065
R60_65 & R60_65	10	11.00000	1.681836	.092610
R75_78 & R75_80	10	9.00000	1.885695	.059345
R90_95 & R90_95	10	11.00000	1.681836	.092610
R105_110 & R105_110	10	10.00000	1.783765	.074471
R120_125 & R120_125	10	22.00000	.560612	.575066

Stroke Volume – Comparison
Normality Test: Failed ($P < 0.050$)
Equal Variance Test: Passed ($P = 0.631$)

Source of Variation	DF	SS	MS	F	P
Subject	9	32042.653	3560.295		
Trial	1	16.284	16.284	0.0355	0.855
Trial x Subject	9	4128.670	458.741		
Time	9	5277.164	586.352	2.968	0.004
Time x Subject	81	16000.854	197.541		
Trial x Time	9	2877.797	319.755	3.195	0.002
Residual	81	8105.245	100.065		
Total	199	68448.666	343.963		

Wilcoxon Matched Pairs Test

	Valid N	T	Z	p-level
REST & REST	10	24.00000	.356753	.721279
R2 & R2	10	6.00000	2.191483	.028424
R15_20 & R15_20	10	17.00000	1.070259	.284511
R30_35 & R30_35	10	20.00000	.764471	.444592
R45_50 & R45_50	10	19.00000	.866400	.386277
R60_65 & R60_65	10	27.00000	.050965	.959354
R75_78 & R75_80	10	0.00000	2.803060	.005065
R90_95 & R90_95	10	24.00000	.356753	.721279
R105_110 & R105_110	10	19.00000	.866400	.386277
R120_125 & R120_125	10	15.00000	1.274118	.202631

Cardiac Output – Comparison
Normality Test: Failed ($P < 0.050$)
Equal Variance Test: Passed ($P = 0.448$)

Source of Variation	DF	SS	MS	F	P
Subject	9	385.360	42.818		
Trial	1	12.248	12.248	5.353	0.046
Trial x Subject	9	20.592	2.288		
Time	9	208.041	23.116	16.518	<0.001
Time x Subject	81	113.355	1.399		
Trial x Time	9	8.945	0.994	1.432	0.188
Residual	81	56.220	0.694		
Total	199	804.761	4.044		

Wilcoxon Matched Pairs Test

	Valid N	T	Z	p-level
REST & REST	10	21.00000	.662541	.507629
R2 & R2	10	20.00000	.764471	.444592
R15_20 & R15_20	10	14.00000	1.376047	.168817
R30_35 & R30_35	10	5.00000	2.293412	.021831
R45_50 & R45_50	10	19.00000	.866400	.386277
R60_65 & R60_65	10	18.00000	.968330	.332887
R75_78 & R75_80	10	0.00000	2.803060	.005065
R90_95 & R90_95	10	16.00000	1.172189	.241130
R105_110 & R105_110	10	14.00000	1.376047	.168817
R120_125 & R120_125	10	15.00000	1.274118	.202631

Common Femoral Artery Blood Flow – Comparison
Normality Test: Failed ($P < 0.050$)
Equal Variance Test: Failed ($P < 0.050$)

Source of Variation	DF	SS	MS	F	P
Subject	9	1407977.917	156441.991	3.870	0.006
Trial	1	462.412	462.412	0.0174	0.898
Trial x Subject	9	238824.978	26536.109		
Time	9	16338547.412	1815394.157	73.488	<0.001
Time x Subject	81	2006591.415	24772.734		
Trial x Time	9	436379.560	48486.618	4.485	<0.001
Residual	80	864958.453	10811.981		
Total	198	22707928.392	114686.507		

Wilcoxon Matched Pairs Test

	Valid N	T	Z	p-level
REST & REST	10	20.00000	.764471	.444592
R2 & R2	8	3.00000	2.100420	.035700
R15_20 & R15_20	10	13.00000	1.477977	.139424
R30_35 & R30_35	10	27.00000	.050965	.959354
R45_50 & R45_50	10	3.00000	2.497271	.012520
R60_65 & R60_65	10	2.00000	2.599201	.009348
R75_80 & R75_80	8	3.00000	2.100420	.035700
R90_95 & R90_95	10	25.00000	.254824	.798861
R105_110 & R105_110	9	12.00000	1.243933	.213533
R120_125 & R120_125	10	23.00000	.458682	.646465

Superficial Femoral Artery Blood Flow – Comparison

Normality Test: Failed ($P < 0.050$)

Equal Variance Test: Failed ($P < 0.050$)

Source of Variation	DF	SS	MS	F	P
Subject	9	719493.720	79943.747	4.649	0.003
Trial	1	291663.130	291663.130	23.683	<0.001
Trial x Subject	9	110943.340	12327.038		
Time	9	4115193.921	457243.769	70.122	<0.001
Time x Subject	81	530145.965	6545.012		
Trial x Time	9	90493.039	10054.782	6.087	<0.001
Residual	80	132154.193	1651.927		
Total	198	6337968.638	32009.943		

Wilcoxon Matched Pairs Test

	Valid N	T	Z	p-level
REST & REST	10	11.00000	1.681836	.092610
R2 & R2	8	15.00000	.420084	.674427
R15_20 & R15_20	10	0.00000	2.803060	.005065
R30_35 & R30_35	10	0.00000	2.803060	.005065
R45_50 & R45_50	10	0.00000	2.803060	.005065
R60_65 & R60_65	10	0.00000	2.803060	.005065
R75_80 & R75_80	8	0.00000	2.520504	.011724
R90_95 & R90_95	10	2.00000	2.599201	.009348
R105_110 & R105_110	10	4.00000	2.395342	.016611
R120_125 & R120_125	10	2.00000	2.599201	.009348

APPENDIX C
SUPPLEMENTARY BLOOD DATA

Lactate – Multiple Wingate Exercise

Subject	Rest	R2	R15	R30	R45	R60	R90	R120
1	1.1	16.2		15.5	8.8	5.7	2.4	2.0
2								
3	1.5	12.7	9.0	5.5	3.7	3.0	2.2	1.4
4	1.5	16.0	12.7	9.7	7.4	5.0	3.9	2.8
5	1.5	13.2	12.7	8.7	6.7	4.6	3.0	2.3
6	1.3	14.1	13.1	10.2	7.3	3.8	3.0	1.9
71.4		13.4	16.7	11.6	7.3	4.3	2.8	2.0
8	1.4	15.4	14.1	11.0	8.6	6.4	3.5	2.6
9	1.1	13.8	12.1	8.7	5.2	3.9	2.3	1.8
10	1.3	13.9	9.8	7.9	6.2	4.3	3.2	2.0
Mean	**1.3**	**14.3**	**12.5**	**9.9**	**6.8**	**4.6**	**2.9**	**2.1**
SD	0.2	1.3	2.4	2.8	1.6	1.0	0.6	0.4
SEM	0.1	0.4	0.8	0.9	0.5	0.3	0.2	0.1

Lactate – Single Wingate Exercise

Subject	Rest	R2	R15	R30	R45	R60	R90	R120
1	1.5	4.5	6.4	6.0	3.7	2.9		
2	1.6	7.2	7.0	5.2	3.7	3.0	2.5	2.2
3	1.2	11.5	10.2	7.4	4.9	3.2		
4	1.5	8.7	10.7	7.6	5.4	3.6	2.5	2.2
5	1.6	4.8			3.8	2.7	1.5	1.8
6	1.5	11.8	9.6	7.0	4.8	3.6	2.6	1.8
7	1.4	6.3	7.7	5.0	4.0	2.7	2.2	2.0
8	1.1	8.2	8.9	6.9	5.1	4.0	2.4	2.0
9	1.5	13.7	10.7	7.1	4.7	3.3	1.9	1.6
10	1.5	7.0	10.1	8.0	5.3	3.4	3.0	1.9
Mean	**1.4**	**8.4**	**9.0**	**6.7**	**4.5**	**3.2**	**2.3**	**1.9**
SD	0.2	3.1	1.6	1.1	0.7	0.4	0.5	0.2
SEM	0.1	1.0	0.5	0.4	0.2	0.1	0.2	0.1

pH – Multiple Wingate Exercise

Subject	Rest	R2	R15	R30	R45	R60	R90	R120
1	7.33	7.05	7.15	7.26	7.37	7.37	7.36	7.36
2								
3	7.32	7.13	7.25	7.31	7.33	7.34	7.34	7.35
4	7.38	7.12	7.19	7.31	7.34	7.38	7.35	7.37
5	7.34	7.10	7.15	7.30	7.33	7.35	7.37	7.36
6	7.30	6.96	7.03	7.16	7.24	7.28	7.31	7.31
7	7.36	7.07	7.12	7.25	7.33	7.37	7.38	7.39
8	7.36	7.03	7.07	7.15	7.19	7.24	7.30	7.30
9	7.33	7.10	7.17					
10	7.38	7.12	7.19	7.30	7.36	7.38	7.37	7.36
Mean	**7.34**	**7.08**	**7.15**	**7.26**	**7.31**	**7.34**	**7.35**	**7.35**
SD	0.03	0.06	0.07	0.07	0.06	0.05	0.03	0.03
SEM	0.01	0.02	0.02	0.02	0.02	0.02	0.01	0.01

pH – Single Wingate Exercise

Subject	Rest	R2	R15	R30	R45	R60	R90	R120
1	7.36	7.26	7.24	7.32	7.34	7.39		
2	7.32	7.20	7.23	7.26	7.31	7.34	7.35	7.33
3								
4	7.33	7.13	7.24	7.30	7.34	7.34	7.35	7.38
5	7.337.18				7.33	7.34	7.35	7.34
6	7.34	7.05	7.10	7.26	7.29	7.32	7.32	7.32
7	7.34	7.16	7.19	7.26	7.31	7.32	7.33	7.34
8	7.35	7.16	7.16	7.24	7.28	7.31	7.31	7.35
9	7.34	7.10	7.16	7.27	7.34	7.36	7.37	7.34
10	7.38	7.19	7.23	7.32	7.37	7.37	7.37	7.38
Mean	**7.34**	**7.16**	**7.19**	**7.28**	**7.32**	**7.34**	**7.34**	**7.35**
SD	0.02	0.06	0.05	0.03	0.03	0.03	0.02	0.02
SEM	0.01	0.02	0.02	0.01	0.01	0.01	0.01	0.01

Packed Cell to Plasma Ratio – Multiple Wingate Exercise

Subject	Rest	R2	R15	R30	R45	R60	R90	R120
1	37.67	47.50	46.50	43.50	43.00	41.67	39.67	39.33
2								
3	35.67	42.33	43.67	40.33	38.33	37.00	36.00	34.50
4	42.50	51.00	46.67	46.00	45.00	40.00	45.33	40.00
5	39.67	46.00	45.67	44.00	39.00	41.50	43.50	40.00
6	44.00	52.67	49.00	47.00	46.33	44.67	45.00	44.00
7	45.00	52.00	50.50	48.50	47.00	46.00	47.67	46.00
8	42.00	49.50	47.67	42.00	44.50	43.33	43.33	42.67
9	41.67	51.33	49.33	47.67	48.33	43.33	45.00	45.00
10	43.67	51.00	47.00	46.50	45.50	45.67	45.67	46.33
Mean	**41.31**	**49.26**	**47.33**	**45.06**	**44.11**	**42.57**	**43.46**	**41.98**
SD	3.09	3.38	2.07	2.75	3.44	2.88	3.55	3.87
SEM	1.03	1.13	0.69	0.92	1.15	0.96	1.18	1.29

Packed Cell to Plasma Ratio – Single Wingate Exercise

Subject	Rest	R2	R15	R30	R45	R60	R90	R120
1	41.67	50.33	46.00	43.67	42.33	44.33		
2	44.33	48.33	44.50	43.33	44.50	44.67	43.50	46.00
3	36.50	43.00	41.00	40.00	39.00	37.50		
4	44.67	49.67	49.67	46.00	45.50	44.67	46.00	46.67
5	43.00	45.00			43.33	43.67	41.33	42.67
6	46.33	53.67	52.00	48.33	47.67	46.00	44.00	45.67
7	46.00	50.67	51.00	49.00	47.50	48.00	45.00	47.50
8	40.00	47.50	47.50	45.67	43.00	44.00	40.67	43.00
9	43.00	48.67	48.00	44.67	41.50	42.00	43.00	43.00
10	41.33	50.50	45.00	47.50	44.50	46.00	47.00	47.00
Mean	**42.68**	**48.73**	**47.19**	**45.35**	**43.88**	**44.08**	**43.81**	**45.19**
SD	2.97	3.04	3.47	2.81	2.66	2.81	2.18	1.99
SEM	0.94	0.96	1.16	0.94	0.84	0.89	0.77	0.70