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Cardiovascular regulation during passive cycle exercise in trained and untrained males

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Cardiovascular Regulation During Passive Cycle Exercise in Trained and Untrained Males

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Abstract

Cardiovascular responses of trained male Cyclists (20.30 ±1.11 years; n=10), trained Runners (26.20 ±1.10 years; n=10), and fit but untrained Controls (22.00 ±0.75 years; n=10) were examined at supine and upright rest, and during passive cycle exercise. Mean maximal aerobic power of Cyclists (76.25 ±1.44 $ml.kg^{-1}.min^{-1}$) and Runners (65.50 ±2.16 $ml.kg^{-1}.min^{-1}$) was significantly higher ($p<0.05$) than that of Controls (46.31 ±2.36 $ml.kg^{-1}.min^{-1}$). Resting heart rate of Cyclists (58.43 ±2.69 $b.min^{-1}$), Runners (57.21 ±3.47 $b.min^{-1}$), and Controls (65.38 ±2.93 $b.min^{-1}$) was not significantly different ($p>0.05$).

Impedance cardiography was used to examine the response of heart rate, stroke volume, cardiac output, contractility of the heart, and arteriolar resistance, during passive cycle exercise at three different intensities (30 rpm, 60 rpm, and 80 rpm). Blood pressure and rate pressure product were measured throughout exercise. The vagal influence on the heart was assessed through time series analysis of heart rate variability at low (0.07-0.11 Hz) and high frequencies (0.12-0.40 Hz). Electromyography was used to ensure that passively moved muscles did not contract. Cardiovascular response was compared for all subjects combined and between the three groups (Cyclists, Runners, and Controls).

Results indicated that during passive cycle exercise all subjects combined showed a significant increase in heart rate, stroke volume, stroke index, cardiac output, cardiac index, contractility of the heart, blood pressure, and decreased

arteriolar resistance and vagal influence on the heart ($p < 0.001$). However, during passive cycle exercise no differences in cardiovascular response were found between Cyclists, Runners, and Controls.

The increase in heart rate during passive cycle exercise may be due to the stimulation of mechanoreceptor located in the exercising muscles, joints, and tendons. The absence of EMG activity during passive cycle exercise support the view that central command did not contribute to the heart rate increase to passive exercise. The small and similar stroke volume response during passive cycle exercise of Cyclists, Runners, and Controls indicates that the muscle pumps may not be effective during this form of passive exercise.

Dedicated to

The memory of my Father,

My Mother,

My Brothers, and

My Sisters, for all their support.

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CHAPTER ONE: INTRODUCTION

1.1 RATIONALE

One of the major factors underlying the high aerobic power of trained athletes is their ability to increase stroke volume during exercise (Rerych *et al.*, 1980). The primary mechanism underlying the enhanced stroke volume of the trained appears to be enhanced left ventricular end-diastolic volume (Schairer *et al.*, 1992). Thus, exercise training appears to increase cardiac performance by inducing cardiac dilatation during exercise. This dilatation is also present during rest as trained aerobic athletes typically possess significantly larger resting stroke volumes and lower resting heart rates compared to sedentary individuals (Convertino, 1991). Therefore, untrained compared to trained individuals typically possess smaller stroke volumes at rest and fail to significantly increase stroke volume during aerobic exercise. The large increase in exercise stroke volume of trained subjects and the failure to increase stroke volume in untrained males was verified by a recent study from our laboratory (Boutcher, McLaren, Cotton, & Nurhayati, 1994). The results of this study are shown in Figure 1.1. The increased stroke volume of the trained during exercise implies that, in contrast to the untrained, they are able to increase venous return to the heart. We have shown that this increase in stroke

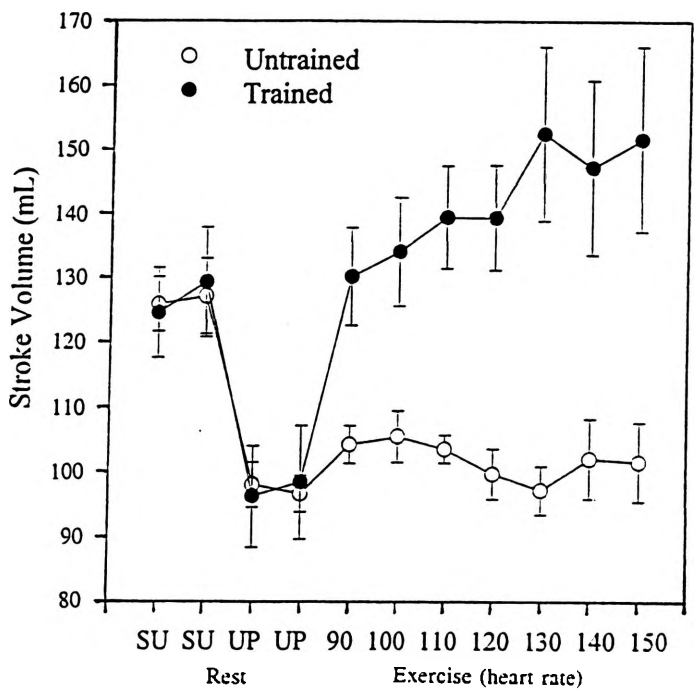

Figure 1.1 Stroke volume response of trained and untrained during exercise

volume occurs much earlier on during exercise than previously thought (below 20% of $\dot{V}o_{2max}$; Boutcher *et al.*, 1994). How trained individuals increase venous return so early on during exercise, however, is undetermined. It is feasible that the enhanced blood volume accompanying training may be the basis of the greater cardiac dilatation of trained subjects during exercise (Convertino, 1991). For instance, a number of studies have shown that trained males possess greater blood volume compared to untrained subjects (Brotherhood *et al.*, 1975; Dill *et al.*, 1974). Importantly, Convertino (1991) has demonstrated that with elevated blood volume comes an increase in central venous pressure. Thus, one of the factors underlying the inability of untrained subjects to enhance stroke volume during exercise appears to be their smaller blood volumes that result in a venous return that is insufficient to increase central venous pressure and stroke volume during exercise.

The ability of the exercise muscle pumps to effectively return blood to the heart may also contribute to the enhanced stroke volume of the trained subjects (Folkow *et al.*, 1971; Laughlin, 1987; Stegall, 1966). For instance, it has been shown that cycling results in substantial adaptations in the leg muscles (Gardner & Fox, 1993; Ludbrook, 1966). Every major muscle group in the legs acts as a muscle pump by compressing the veins and forcing blood back to the heart (Folkow *et al.*, 1970; Gauer & Thorn, 1965). *Thus, more efficient muscle pumps, or what Rowell (1986, p.144) has termed the athletes' "second heart" together with an increased central venous pressure through enhanced blood volume may be the foundation for the early increase in stroke volume of aerobically trained subjects. Consequently, mechanical rather than neural or metabolic factors may be the major determinant of stroke volume increase during light exercise.*

Thus, the examination of the factors that determine the increase in stroke volume during initiation of exercise are crucial in explaining how the trained are able to expand their hearts so dramatically during aerobic exercise. Unfortunately, the initiation of stroke volume increase during exercise is extremely difficult to assess because subjects tend to exhibit a sympathetic response to exercise even at light work loads. The increase in sympathetic activity either through muscular activity or pre-exercise anxiety typically results in elevated heart rates. One method of reducing the autonomic and metabolic response to exercise is passive exercise. During passive exercise subjects make no voluntary movements and sit passively while their limbs are moved through some outside agency. If the increase in stroke volume during exercise is driven by mechanical factors (as suggested above) then the enhancement of venous return during passive exercise should increase stroke volume when the muscle pumps are passively initiated. If individuals possess greater blood volume then it would follow that passive stimulation of their muscle pumps would result in greater venous return and, therefore, greater increases in stroke volume. Although infrequently used preliminary work using passive exercise supports the notion that passive movement of the lower limbs results in an increase in stroke volume.

For instance, Morikawa *et al.* (1989) compared the cardiovascular response of healthy subjects and paraplegic patients. Interestingly, their results indicated that both healthy subjects and paraplegic patients recorded a similar cardiac response to passive exercise. They showed that during light passive exercise, cardiac output and stroke volume increased for both groups. The mechanism underlying the stroke volume increase in paraplegic patients is likely to be the activation of the muscle

pump as they lack afferent feedback.

Also in the Morikawa *et al.* (1989) study during passive exercise both groups displayed a decrease in heart rate. The decrease in heart rate underlies the lack of neural input to the heart during passive exercise and also reflects its unique cardiovascular regulation. It is likely that heart rate decreases during light and moderate passive exercise because increased venous return results in elevated cardiac output, which in turn causes an increase in mean arterial pressure. Thus, heart rate decrease may be the likely response to decrease cardiac output, resulting in decreased blood pressure. It is feasible that increased parasympathetic influence on the heart may be responsible for the decreased heart rate during moderate passive exercise.

In contrast, Nóbrega and Araújo (1993) have shown that heart rate increases during the first four seconds of high intensity passive exercise. They suggest that the increase in heart rate occurring through high intensity passive cycle exercise is likely to have been brought about by mechanical stimuli in the exercising limbs. Thus, heart rate response to high intensity passive exercise might depend on central command or mechanoreceptor activation in the exercising limbs without interference from peripheral metaboreceptors. Collectively, these data suggest that heart rate may decrease during moderate passive exercise but can accelerate during more intensive passive exercise.

In summary, one of the major factors underlying the high aerobic power of trained athletes is their ability to increase stroke volume during exercise. The primary mechanism underlying the enhanced stroke volume of athletes appears to be mechanical, namely enhanced venous return. The mechanical effects on the heart

can be studied through passive exercise which substantially reduces neural and metabolic response to exercise.

It is predicted that the cardiovascular response to moderate intensity passive exercise will be an increase in stroke volume and a decrease in heart rate. There are several mechanisms underlying the increase of stroke volume. One of the factors influencing stroke volume is the action of the muscle pumps. However, the action of the muscle pumps may vary among individuals. In paraplegics, passive exercise also results in a decreased heart rate and increased stroke volume emphasizing the importance of mechanical factors influencing the return of blood to the heart. As trained athletes possess significantly greater blood volume it is likely that passively moving the lower limbs, will result in greater venous return to the heart and consequently a greater stroke volume.

To examine the influence of aerobic exercise on stroke volume response to passive exercise the cardiovascular response in trained cyclists (Cyclists), trained runners (Runners), and healthy untrained males (Controls) during passive cycle exercise will be examined. The pattern of stroke volume, cardiac output, heart rate, blood pressure, total peripheral resistance, and parasympathetic response during passive cycle exercise will be assessed in all three groups.

1.2 SIGNIFICANCE

An important factor that influences the performance of endurance athletes is their ability to increase stroke volume. The increase in stroke volume can be brought about by an increase in end-diastolic volume or a decrease in end-systolic volume. The increase in end-diastolic volume or increase in venous return has been shown to be a particularly important influence on stroke volume increase during

early or light exercise. Unfortunately, the initial increase in stroke volume is difficult to examine during actual exercise because of pre-exercise anxiety and metabolic demands on the heart. Passive exercise may provide a way to examine cardiac dynamics during light exercise. Understanding how athletes are able to expand stroke volume is important as their larger stroke volumes are the basis for their superior cardiovascular performance during aerobic exercise. Also passive cycle exercise provides a non-metabolic method of examining cardiovascular response to movement of large muscle groups.

1.3 AIMS

The specific aims of this study were to:

i) determine the cardiovascular response during passive cycle exercise of Cyclists, Runners, and Controls combined.

ii) determine the possible differences in stroke volume, heart rate, and vascular response as well as parasympathetic influence on the heart during passive cycle exercise between Cyclists, Runners, and Controls.

1.4 HYPOTHESES

It is hypothesized that,

(i) compared to Controls, trained Cyclists and Runners will display significantly greater stroke volume, stroke index, and lower heart rate, and total peripheral resistance at rest.

(ii) passive cycle exercise for all thirty subjects combined will result in significantly greater stroke volume, heart rate, stroke index, cardiac output, cardiac index, contractility of the heart, blood pressure, rate pressure product, and decreased total peripheral resistance and vagal influence on the heart.

(iii) during passive cycle exercise trained Cyclists and Runners will display significantly greater increase of stroke volume, reduction of heart rate, and decrease of vagal tone compared to Controls.

(iv) trained Cyclists and Runners will display significantly shorter pre-ejection period, left ventricular ejection time, and pre-ejection period/left-ventricular ejection time (PEP/LVET ratio) compared to Controls during passive cycle exercise.

CHAPTER TWO: LITERATURE REVIEW

This literature review describes the cardiovascular response to exercise and to passive exercise and in particular focuses on the stroke volume response to aerobic exercise. In the first section, cardiovascular response to aerobic exercise is described, followed by cardiac adaptation to aerobic exercise (second section). Stroke volume response to exercise is then discussed together with the factors underlying stroke volume changes during exercise (e.g., contractility of the heart, venous return, the effectiveness of the muscle pumps, and blood volume). The passive cycle exercise paradigm as a method of assessing the effect of venous return on the stroke volume response to exercise is described in the next section. Finally, the influence of passive cycle exercise on cardiovascular response and the possible mechanisms underlying these cardiovascular changes are described in the last section.

2.1 CARDIAC RESPONSE TO AEROBIC EXERCISE

Acute aerobic exercise (e.g., treadmill, cycle ergometry exercise) brings about rapid cardiovascular changes. Braunwald and Ross (1979) have suggested that during exercise, the cardiac response is complex, and involves an interaction of changes in heart rate, contractility of the heart, end-diastolic volume, and end-systolic volume. During aerobic exercise, the major determinants of cardiac performance are heart rate and stroke volume (Brooks & Fahey, 1984, p.313). It is well established that during exercise heart rate and cardiac output increase linearly with work (Rosiello *et al.*, 1987). Heart rate is also correlated to oxygen consumption and metabolic demands (Fagraeus & Linnarson, 1976; Petro *et al.*, 1970). Also, maximal oxygen consumption or aerobic power is correlated with

diastolic filling (Levy *et al.*, 1993). The increase of heart rate during exercise is closely correlated with plasma renin activity which reflects increased sympathetic activity to the kidneys (Finberg & Berylne, 1977). The rate of heart rate increase depends on the type of exercise, age, sex, and fitness of the individual (e.g., sedentary or trained). For instance, Rosiello *et al.* (1987) found that heart rate response was significantly higher during rowing than cycle exercise. This result indicates that the increase of heart rate depends on the muscle groups that are involved in different types of exercise.

The mechanism underlying the increase of heart rate at the onset of exercise is brought about by vagal withdrawal (Nóbrega & Araújo, 1993; Petro *et al.*, 1970); during harder exercise the increase of heart rate is attributed to an increase in cardiac sympathetic activity (Christensen & Galbo, 1983). A study by Maciel *et al.* (1986) attempted to examine the heart rate response during dynamic exercise under pharmacological blockade of the parasympathetic and sympathetic nervous systems. They found that the increase of heart rate in the first thirty seconds of exercise was due to vagal withdrawal. Furthermore, they suggested that the activation of sympathetic nervous system plays an important role in increasing heart rate during hard dynamic exercise.

Vagal withdrawal from the heart can also be assessed through time series analysis of heart rate variability. Heart rate variability as a measure of vagal influence on the heart is one noninvasive method of obtaining information about cardiovascular autonomic nerve function (Niklasson *et al.*, 1993; Saul, 1990; Yamamoto *et al.*, 1991). The measurement of heart rate variability, which will be used in this study, can be used both as a marker of parasympathetic and sympathetic

influence under various conditions (De Meersman, 1993; Malik & Camm, 1993). Heart rate variability can be assessed through time series analysis that identifies different frequencies within the heart rate variability spectrum. The low frequency (0.05-0.15 Hz) usually is modulated by both parasympathetic and sympathetic activity, whereas high frequency (0.15-0.50 Hz) is modulated by parasympathetic activity (Akserold *et al.*, 1985; Malik & Camm, 1993; Saul *et al.*, 1990). The increase of heart rate variability at the high frequency indicates increased modulation of vagal activity, whereas the decrease of heart rate variability at the high frequency reflects reduction of vagal activity modulation (Malik & Camm, 1993). Maciel *et al.* (1986) showed that during moderate dynamic exercise the vagal nerves have a greater influence on increase in heart rate compared to the sympathetic pathways. Furthermore, a cross-sectional study by DeMeersman (1993) has shown that habitual exercise resulted in augmented heart rate variability.

2.1.1 Cardiac Output and End-Diastolic Volume Response To Aerobic Exercise

The regulation of cardiac output during exercise is complex (Christensen & Galbo, 1983). Cardiac output is regulated by multiple mechanisms that include heart rate and factors that affect stroke volume (Lakatta, 1993). A study by Thadani and Parker (1978) has shown that during upright cycle exercise cardiac output was increased about 160% from upright rest values. Furthermore, Poliner *et al.* (1980) have suggested that an increase of cardiac output is caused by tachycardia and an increase in myocardial contractility that is brought about by greater sympathetic activity and also by the Frank-Starling mechanism. However, during maximal exercise, different factors affect the cardiac output of trained athletes and sedentary individuals. A study by Ogawa *et al.* (1992) showed that the effect of exercise

training on maximal cardiac output in trained subjects was due to a larger stroke volume. During maximal exercise, the trained can increase cardiac output by almost twice as much as that of the untrained with similar maximal heart rate (Gledhill *et al.*, 1994). Therefore, the sedentary can increase cardiac output mainly by increasing heart rate, whereas trained athletes can increase cardiac output by increasing both heart rate and stroke volume (Schairer *et al.*, 1991). Similarly, Tanaka *et al.* (1986) showed that the increase of cardiac output in trained subjects during cycle exercise was attributed to an increase in stroke volume, because the heart rate response did not differ significantly between trained and untrained subjects. Thus, the greater stroke volume was due to lesser intravascular resistance in peripheral circulation and/or greater myocardial contractility. Supine exercise resulted in an increase in cardiac output that was probably caused by an increase in left ventricular end-diastolic volume as a result of increased central venous pressure (Kanstrup *et al.*, 1992). Supine exercise also resulted in an increased heart rate with no change in end-systolic volume, whereas stroke volume was unchanged or slightly increased (Miyamoto *et al.*, 1983). Furthermore, the increase of cardiac output during exercise is primarily caused by the ability of the left ventricle to dilate, resulting in increased end-diastolic volume that leads to increased stroke volume (Rerych *et al.*, 1980).

The role of diastolic filling during exercise has not been studied extensively in humans (Schulman *et al.*, 1992). A study by Levy *et al.* (1993) showed that the increase of diastolic filling during exercise is caused by an increased pressure gradient between the left ventricle and left atrium during early diastole. Furthermore, Levy *et al.* (1993) have suggested that two mechanisms may be

responsible for the enhancement of diastolic filling during exercise; that is an increase in diastolic suction of the left ventricle and an increase in left atrial pressure.

End-diastolic volume increases during both upright and supine exercise. A study by Rubal *et al.* (1980), using radionuclide ventriculography, showed that during upright exercise the higher work achieved by trained subjects was caused by an increase in end-diastolic volume and stroke volume. Their results indicated that the average increase in end-diastolic volume of athletes was about 33%, whereas the increase in the control group was about 14%. Similarly, Adams *et al.* (1992) also found that left ventricular end-diastolic volume was related to stroke volume changes during progressive exercise in the supine position. Furthermore, Marmor *et al.* (1993) showed that supine cycle exercise in healthy subjects resulted in a significant increase in end-diastolic volume.

2.1.2 Stroke Volume Response To Aerobic Exercise

Stroke volume is one of the most important factors underlying the cardiac performance of aerobic athletes (e.g., runners, cyclists, swimmers). The ability to increase stroke volume during exercise is a major factor in the enhancement of cardiac output. Stroke volume in the trained increases during cycle and run exercise in the upright position. A study by Ogawa *et al.* (1992) showed that the effect of exercise training on stroke volume was greater in male than female subjects. Plotnick *et al.* (1986) have shown that upright exercise resulted in increased stroke volume by 31-35%. They suggested that the increase of stroke volume was mainly due to the Frank-Starling mechanism. A recent study from our laboratory (Boutcher *et al.*, 1994) has shown that during light cycle exercise trained male cyclists can

increase stroke volume by about 20%. Heart rate during light exercise in this study was about 90 $b.min^{-1}$. Thadani and Parker (1978) have shown that the plateau of stroke volume can be reached at 40% of $\dot{V}o_{2max}$. In contrast, Gledhill *et al.* (1994) have shown that stroke volume of endurance trained athletes does not plateau during submaximal exercise, but progressively increases to maximal exercise. These data indicate that endurance athletes have the ability to increase ventricular filling and ventricular emptying even at high heart rates.

A study by Rosiello *et al.* (1987), which attempted to investigate the stroke volume response in different forms of exercise, found that stroke volume during rowing was significantly lower than cycling. They concluded that the different stroke volume response during rowing and cycling was due to the different response in ventricular contractility. There are several potential mechanisms to explain the increase of stroke volume of the trained during aerobic exercise. Some studies indicate that the increase of stroke volume during exercise is caused by the increase of ventricular filling pressure and end-diastolic volume (Hopper *et al.*, 1988; Levine, 1993; Spina *et al.*, 1992). The enhancement of inotropic state and/or reduction in afterload which leads to increased ventricular emptying may also play a role in enhanced stroke volume response to exercise training (Goodman & Plyley, 1991; Spina *et al.*, 1992). Furthermore, some studies have suggested that stroke volume changes are caused by the Frank-Starling mechanism and increased contractility (Goodman & Plyley, 1991; Poliner *et al.*, 1980; Sullivan *et al.*, 1991). Thus, myocardial contractility becomes a primary mechanism in enhancing left ventricular stroke volume which leads to increased cardiac output (Goodman & Plyley, 1991). A study that examined the stroke volume response in men and

women (Sullivan *et al.*, 1991) showed that during exercise the increase of stroke volume (about 32 to 41%) was due to an increase in left ventricular end-diastolic volume. They also suggested that the increase of left ventricular contractility during exercise was indicated by a decrease of left ventricular end-systolic volume and an increase of left ventricular ejection fraction which maintained the stroke volume changes and end-diastolic volume.

2.1.3 Blood Pressure Response To Aerobic Exercise

Aerobic exercise also affects vasculature adjustment, such as blood pressure response. Blood pressure is a product of cardiac output and total peripheral resistance. The increase of blood pressure is very important to maintain the blood flow to critical areas such as the heart, brain, and exercising muscles (Åstrand & Rodahl, 1977, p.170; Brooks & Fahey, 1984, p.337). Furthermore, the response of blood pressure to exercise can vary greatly, depending on the dynamic and isometric characteristics of the exercise (Franz, 1991). Typically, systolic blood pressure increases during both static or dynamic exercise (MacDougal, 1994). During aerobic exercise, systolic blood pressure increases almost linearly to values of 200 mmHg or more at maximal exercise (MacDougal, 1994). Franz (1991) has shown that systolic blood pressure and diastolic blood pressure are lower in dynamic exercise than in isometric exercise. During dynamic exercise, the increase of systolic blood pressure is accompanied by decreased peripheral resistance which is caused by progressive vasodilatation that occurs in the vessels of the exercising muscles (MacDougal, 1994). Furthermore, Melcher and Donald (1981) and Ludbrook *et al.* (1978) have suggested that the carotid sinus reflex is a major factor in increasing blood pressure during exercise. A study by Penny *et al.* (1981) has

shown that 14-week of jogging program resulted in decreased operational systolic blood pressure.

The decrease of peripheral resistance during exercise results in a slight increase or no increase in diastolic blood pressure. Toska and Eriksen (1994) have shown that the decrease of total peripheral resistance immediately at the initiation of exercise reflects the dramatic increase in muscular vascular conductance caused by increased blood flow to the exercising muscles. Furthermore, Toska and Eriksen (1994) have suggested that during moderate supine dynamic exercise the vasoconstriction of non-exercising muscles is caused by the baroreflex response as a result of decreased mean arterial pressure.

2.2 CARDIAC ADAPTATIONS TO CHRONIC AEROBIC EXERCISE

It has been established that regular aerobic exercise at a moderate to high intensity will change the cardiovascular and metabolic response to acute exercise. However, it has been argued that the major changes as a consequence of aerobic exercise occur in heart function (Rowell, 1986, p.257). Levine (1993) suggested that endurance training results in central and peripheral adaptations that include an increase in the ability to distribute and accommodate blood flow through an increase in maximal cardiac output and stroke volume. Similarly, McArdle *et al.* (1978) suggested that exercise which activates a relatively large muscle mass (e.g., running or cycling) produces general adaptations in heart rate, resulting in an inherent change in cardiac function which is brought about by central and peripheral factors. Most studies have shown that $\dot{V}o_{2max}$ increases as a result of exercise training over a long period of time. Mutton *et al.* (1993) have suggested that five weeks of exercise (e.g., running and cycling) can significantly improve $\dot{V}o_{2max}$ and run performance.

The range of $\dot{V}o_{2max}$ increase varies between male and female and young and old individuals. Van Handel *et al.* (1976) showed that ten weeks of jogging resulted in an increase of $\dot{V}o_{2max}$ about 19.7% for male subjects and 14.8% for female subjects. Stratton *et al.* (1994) showed that six months of exercise training in older and young groups, which consisted of walking, jogging, and cycling, resulted in an increase in $\dot{V}o_{2max}$ by 21% in the older group, whereas the young group increased $\dot{V}o_{2max}$ by about 17%. Hickson *et al.* (1977) have shown that the average of $\dot{V}o_{2max}$ increases by 44% after ten weeks of exercise (e.g., cycling and running). Rowell (1974) has shown that endurance athletes showed little or no increase in $\dot{V}o_{2max}$ with years of exercise training.

What mechanism underlies the increase of maximal oxygen transport to exercising muscle? There are several possibilities underlying the mechanism of the increased $\dot{V}o_{2max}$. These possibilities are highlighted by the Fick principle:

$$\dot{V}o_{2max} = HR_{max} \times SV_{max} \times a\text{-}\bar{v}o_2diff_{max}$$

Based on the Fick equation, an increase in $\dot{V}o_{2max}$ could be achieved by an increase in heart rate (HR), stroke volume (SV), and arteriovenous oxygen difference ($a\text{-}\bar{v}o_2$difference). The increase of $\dot{V}o_{2max}$ with exercise training is primarily due to an increase in maximal stroke volume and cardiac output (Kilbom, 1971). On the other hand, Rodeheffer *et al.* (1984) suggested $\dot{V}o_{2max}$ is determined by maximum cardiac output and maximum $a\text{-}\bar{v}o_2$difference. Furthermore, Ekblom *et al.* (1968) has shown that exercise training resulted in an increase of $\dot{V}o_{2max}$ of about 40% which was accompanied by a 32% increase in cardiac output and stroke volume and only 8% increase in $a\text{-}\bar{v}o_2$difference. Levy *et al.* (1993) have shown that six months of exercise training in young and old subjects, which consisted of jogging, walking

and cycling, resulted in an increased maximal oxygen uptake of about 19%, whereas resting heart rate decreased by about 14%.

The slow heart rate at rest occurs as a consequence of aerobic exercise carried out over a long period of time (Blomqvist & Saltin, 1983; Scheuer & Tipton, 1977). Furthermore, it is well established that the phenomenon of resting bradycardia is present in endurance trained humans and animals (Smith *et al.*, 1989). However, the mechanism underlying the decreased heart rate is unclear. The mechanism may be mediated by a training-induced increased cardiac parasympathetic (vagal) tone and decreased sympathetic activity (Seals & Chase, 1989) or reduced intrinsic heart rate (Katona *et al.*, 1982; Lewis *et al.*, 1980; Negrao *et al.*, 1992). De Meersman (1993) has suggested that parasympathetic activity can be maintained by habitual aerobic exercise which is reflected by augmented heart rate variability. A study by Smith *et al.* (1989) has found that endurance trained subjects had greater parasympathetic influence compared to the untrained. Furthermore, they concluded that exercise training bradycardia which is present in endurance trained subjects was due to lower intrinsic heart rate and enhanced parasympathetic predominance. Van Handel *et al.* (1976) showed that several days of exercise training (jogging) resulted in a reduction of heart rate in male and female subjects.

Cross-sectional studies in humans have shown that improvement of diastolic filling occurred through exercise training, whereas longitudinal studies have shown that exercise training in young subjects resulted in no improvement in diastolic filling (Levy *et al.*, 1993). Furthermore, their study showed that the increase of end-diastolic volume of about 13% occurring in the six months of exercise training may have contributed to the increase in maximal stroke volume, cardiac output, and

maximal oxygen consumption. Stratton *et al.* (1994) showed that the stimulation of beta-adrenergic activity through isoproterenol during exercise training did not enhance the beta-adrenergic diastolic filling responses in old and young subjects. A study by Forman *et al.* (1992) has shown that endurance training over a long period of time is correlated with ventricular filling dynamics.

2.2.1 Blood Volume Expansion

The increase of blood volume as a cardiac adaptation to endurance training is well established (Carroll *et al.*, 1995; Convertino, 1991; Green *et al.*, 1990), and total blood volume seems to be correlated to endurance training and maximal oxygen uptake (Convertino, 1994). Furthermore, Convertino, (1994) has suggested that there appears to be no significant difference in hemoglobin levels between the trained and untrained. These findings indicate that blood volume expansion mainly consists of an increase in plasma volume. It is well established that trained endurance athletes have greater blood volume compared to fit, healthy but untrained individuals. Blood volume for highly competitive athletes has been shown to be about 104 ml/kg compared to 75-85 ml/kg for nonathletes (Dill *et al.*, 1974). However, the mechanisms underlying the expansion of the blood volume are unclear. There are a number of possible explanations. Firstly, there is a possible increase in the ratio of volume to pressure in the vascular space. Another possibility underlying the increase of blood volume with exercise is an increase in plasma renin and antidiuretic hormone or vasopressin as well as total circulating protein caused by an increase in the oncotic pressure across the capillary membrane (Convertino, 1991). This increase would result in an increased retention of sodium and water by the kidneys. Also chronic training leads to an increase in plasma protein, mainly

albumin, which increases the osmolality of the blood. Thus, increased blood protein allows the blood to hold more fluid.

Convertino *et al.* (1991) has shown that blood volume expansion accompanying exercise training results in increased resting cardiac filling pressure (central venous pressure) and resting stroke volume (100 ml to 117 ml). A few days of endurance training resulted in an increase of plasma volume of about 400 ml, even in relatively fit individuals. This adaptation appears to be an important factor underlying the increase of stroke volume that occurs with endurance training (Convertino, 1991). Increased blood volume facilitates venous return of blood to the heart and enhances the Frank-Starling mechanism, which results in an increase in cardiac output. Therefore, blood volume expansion is an important consideration in cardiovascular performance at rest and during exercise.

2.3 FACTORS UNDERLYING STROKE VOLUME RESPONSE TO EXERCISE

Stroke volume is determined by ventricular filling or preload and myocardial contractility (Feigl, 1974; Vatner & Pagani, 1976). There is a different response in stroke volume among trained and untrained individuals. Typically, athletes have a larger stroke volume (Bevegård *et al.*, 1963; Ekblom & Hermansen, 1968) accompanied by a slow heart rate compared to untrained individuals. Levine (1993) suggested that the larger stroke volume in trained subjects was probably caused by greater carotid sinus afferent nerve activity which leads to greater inhibition of efferent sympathetic nerve traffic. Thus, controlling the change in heart rate is mediated by the baroreflex. Schairer *et al.* (1992) have shown that the increase of stroke volume in athletes during exercise resulted in an increase in ventricular filling and an increase in ventricular emptying, whereas sedentary subjects had only an

increase in ventricular emptying without an increase in ventricular filling. Cross-sectional studies examining cardiac dimensions using radiographic, radionuclide imaging, and echocardiographic techniques comparing sedentary and well-conditioned individuals have shown that chronic physical training does increase ventricular volume (Åstrand & Rodahl, 1977; Blomqvist & Saltin, 1983; Scheuer & Tipton, 1977), that in turn, leads to an increase in stroke volume. These results show that athletes have higher end-diastolic volumes than sedentary individuals at supine rest and during exercise. Also six months of physical training in athletes resulted in an increase in end-diastolic volume from 133 to 167 ml at rest, and from 166 to 204 ml during maximal exercise (Rerych *et al.*, 1980). Anholm *et al.* (1982) have also shown that adaptation to habitual exercise involves left ventricular end-diastolic volume changes.

The previously mentioned study by Boutcher *et al.* (1994) showed that there was a big difference in stroke volume response during cycle exercise in trained and untrained individuals. The trained cyclists in their study increased stroke volume by about 20% during actual exercise, whereas untrained individuals did not have the ability to increase stroke volume. However, how trained athletes can increase stroke volume so early on in exercise is unclear, as are the mechanisms underlying enhanced stroke volume during moderate to hard exercise.

There are several possible mechanisms underlying the enhanced stroke volume of trained individuals observed during actual exercise. Firstly, an increase in stroke volume could be due to either an increase in preload or an increase in myocardial contractility (Vatner & Pagani, 1976). Secondly, Åstrand and Rodahl (1977, p.186) have suggested that the two primary factors affecting stroke volume

are increased venous return to the heart and distensibility of the ventricles. Thirdly, another factor affecting exercise stroke volume is the effectiveness of the muscle pumps to function as a second heart (Rowell, 1986, p.144) by enhancing venous return. Lastly, as mentioned, greater blood volume has been documented as a factor enhancing resting and exercise stroke volume (Convertino, 1991).

2.3.1 Contractility

One of the non-invasive techniques to assess contractility of the heart is the systolic time interval (Ahmed *et al.*, 1972; Lewis *et al.*, 1977; Sheps *et al.*, 1982). Three basic measurements in systolic time interval were suggested by Lewis *et al.* (1977); that is pre-ejection period, which is determined between the onset of depolarization and the beginning of ventricular contraction; left ventricular ejection time, which is determined by at the end point of pre-ejection period when the blood is actually pumped from the left ventricle; and total electromechanical systole (QS_2), which is pre-ejection period plus left ventricular ejection time. Also Marmor *et al.* (1993) suggested that the most frequently used index of left ventricular function is left ventricular ejection fraction.

Contractility of the heart refers to the strength of cardiac contractions under conditions in which ventricular end-diastolic filling, ventricular end-systolic volume, and heart rate are constant (Rowell, 1986, p.265). Most studies indicate that contractility of the heart contributes to the increase of stroke volume during hard exercise. For instance, a study by Sheps *et al.* (1982) showed that during upright exercise a decrease of pre-ejection period/left ventricular ejection time ratio (PEP/LVET ratio) was due to an increase in cardiac output, stroke volume, and contractility. Furthermore, Newlin and Levenson (1979) have suggested that the

interpretations of pre-ejection period is limited by such factors as ventricular preload, afterload; alpha-adrenergic contractility on contractility, non-adrenergic drugs which affect pre-ejection period, and cardiac abnormalities.

It is well known that there is a relationship between pre-ejection period and heart rate (Newlin & Levenson, 1979). Furthermore, Newlin and Levenson (1979) found that pre-ejection period was decreased and heart rate was increased by positive inotropic agents and adrenergic cardio-stimulation, whereas pre-ejection period was unchanged by vagal blockade and atrial pacing. Miyamoto *et al.* (1983) suggested that shortening pre-ejection period during exercise is a consequence of an increase in venous return together with sympathetic stimulation. Furthermore, they concluded that the length of pre-ejection period and left ventricular ejection time during rest and exercise is mainly determined by the amount of blood that is available to the heart. Also a study by Mahler *et al.* (1985) has shown that the increase of stroke volume during submaximal exercise was dependent on Frank-Starling mechanism and increased ventricular contractility.

2.3.2 Venous Return

Venous return is an important factor in controlling cardiac output. This is based on the mechanism called the Frank Starling law of the heart, which allows the heart to pump the blood automatically, whatever amount of blood flows into the right atrium from the veins (Guyton, 1991, p.221). There are several factors that affect enhanced venous return such as: sympathetically induced venous vasoconstriction; skeletal-muscle activity; the effect of venous valves; respiratory activity; and the cardiac suction effect by influencing the pressure gradient between the veins and the heart (Sherwood, 1993, p.329). Venous return of blood can also

be affected by such factors as increased blood volume, increased tone of large blood vessels, and dilatation of small blood vessels (Guyton, 1991, p.225).

2.3.3 Muscle Pumps

The muscle pumps that Rowell (1986, p.144) has called the "second heart" is one possible factor that may enhance venous return during exercise thereby causing an increase in stroke volume. Blomqvist and Stone (1983) have suggested that the systemic circulation can be influenced by rhythmic exercise of the leg either in the sitting or standing position which is caused by contribution of the muscle pumps in the upper and lower leg. Every major muscle group acts as a muscle pump, so when muscles contract the veins are compressed (Gauer & Thron, 1965). Thus, the power of the pump is provided by contractions of the muscles (Ludbrook, 1962). Venous compression results in decreased venous capacity and increased venous pressure that leads to fluid contained in the veins being squeezed back towards the heart (Sherwood, 1993, p.329). The mechanism of the muscle pumps was explained by Van Leeuwen *et al.* (1992) as follows: during contraction of the calf muscles, the blood is squeezed toward the heart, then the venous valves prevent the blood from flowing back. Because of the muscle contractions, the venous pressure is lowered, and the perfusion increases by the same amount. Furthermore, if the next contraction follows before the venous system is refilled, the venous pressure remains decreased for as long as the exercise is continuous.

The effectiveness of the muscle pumps is very important in facilitating venous return (Sheriff *et al.*, 1993). For instance, when a person stands up, the blood will pool in the legs due to the gravitation effect (Sherwood, 1993, p.331). The longer the time spent in the standing position, the more the blood will be pooled

in the leg instead of moving back to the heart. Therefore, when a person stands upright for a long period of time, the blood flow to the brain is reduced which can lead to fainting. In this condition, mild exercise (e.g., leg movement) is important to stimulate the muscle pumps to compress the veins and squeeze the blood back to the heart. Thus, these mechanisms act together in an attempt to increase venous return (Plotnick *et al.*, 1986). The single and strong contraction of the calf muscles during standing resulted in an increase of blood flow by 60 percent (Ludbrook, 1966) also contraction of the calf muscles can produce a driving force of 90 mmHg (Barcroft & Dornhorst, 1949; Stegall, 1966). The muscle pumps also prevent the blood moving backwards because most veins have one way valves that allow the blood to move only in one direction. In addition, Sherwood (1993, p.333) has shown that the respiratory pump also enhances venous return.

There are a number of possible functions of the skeletal muscle pump in the lower leg. Firstly, the action of the muscle pumps will increase the perfusion of the calf muscles resulting in an increased blood back to the heart. Secondly, the veins in the leg act as a reservoir for blood (Ludbrook, 1966; Sherwood, 1993, p.328). Another possible function of the skeletal muscle pumps has been their role in preventing the formation of oedema (Stick *et al.*, 1993). A study by Sheriff *et al.* (1993) showed that the muscle pump contributes to the dramatic increase in muscle blood flow observed at the initiation of exercise. Their study which was performed by a manipulation of muscle contraction rate through treadmill running in dogs showed that the activation of the muscle pump resulted in a decrease in volume of blood from the non-compliant arterial system; this effect resulted in a lowering of arterial pressure. Furthermore, the muscle contraction forced blood into the central

veins that led to increased central venous pressure.

2.3.4 Blood Volume

As briefly mentioned previously, blood volume expansion is one possible factor underlying the increase of stroke volume during exercise. It has been well documented in cross-sectional and longitudinal studies that blood volume expansion occurs as a consequence of physical training. Hopper *et al.* (1988) have shown that during exercise, plasma volume expansion by 400 ml in untrained individuals increased stroke volume by 11%. Also, exercise training for two to four weeks resulted in an increased plasma volume (Convertino, 1991; Convertino *et al.*, 1991). Cross-sectional studies have demonstrated that endurance trained athletes have expanded blood volume about 20-25% greater compared to untrained individuals (Brotherhood, 1975). Longitudinal studies have also showed that up to ten days of endurance training increases blood volume expansion primarily through plasma volume expansion (Convertino *et al.*, 1980; Convertino *et al.*, 1980; Green *et al.*, 1984; Kirby & Convertino, 1986). Allen *et al.* (1992) showed that mild aerobic exercise that consisted of swimming, running, walking, and cycling resulted in plasma volume expansion about 5.9%, whereas Pugh (1969) showed that the blood volume expansion that occurred was associated with a plasma volume expansion of about 7.3%. The average blood volume expansion from longitudinal research is reported to be about 7% (Convertino, 1991).

The major cardiovascular advantages from blood volume expansion (hypervolemia) induced by exercise training appear to be a reduction in heart rate (Convertino, *et al.*, 1980; Convertino *et al.*, 1980; Convertino, 1983; Wyndham *et al.*, 1976) and elevation in stroke volume (Green *et al.*, 1990). Kanstrup and

Ekblom (1982) have shown that acute plasma volume expansion resulted in an increased cardiac output and stroke volume. Furthermore, Convertino (1983) has shown that training induced exercise heart rate and hypervolemia were correlated with a 1% increase in plasma volume being associated with a 1% reduction in exercise heart rate. The mechanism underlying the effect of hypervolemia on stroke volume and heart rate during exercise may be the Frank-Starling law (Convertino, 1991). During rest and exercise, hypervolemia is associated with elevated central venous pressure (Thompson *et al.*, 1990) which is associated with increased stroke volume (Hopper *et al.*, 1988). The increase of stroke volume during exercise may result from greater venous return and increased right atrial filling pressure associated with a greater pressure gradient from the central venous reservoir to the right atrium as a consequence of exercise training (Convertino *et al.*, 1991). Thus, the greater blood volume of endurance trained athletes appears to be a major contributor to their increase in ventricular function (Gledhill *et al.*, 1994). Therefore, hypervolemia may contribute significantly to improved cardiovascular performance by increasing stroke volume (Convertino, 1991).

2.4 PASSIVE EXERCISE PARADIGMS

The majority of studies examining stroke volume response to exercise have shown that it is difficult to examine stroke volume changes during early, light aerobic exercise. Difficulties are caused by factors such as subjects being excited at the start of testing session which increases heart rate and confounds stroke volume response at the start of exercise. One possible way to examine the increase of stroke volume during early, light exercise may be through passive exercise. Passive exercise involves the subjects' limbs being moved by an outside agency while they

remain relaxed and passive.

Few research studies have attempted to investigate the cardiovascular response to passive exercise. However, some passive paradigms have been used to investigate cardiovascular regulation. For instance, Bahnson *et al.* (1949) attempted to examine the effect of passive movement on respiration and oxygen consumption in male subjects using a bicycle connected to an electric motor, at a rate of 60 rpm for 5 minutes. The results showed that during passive movement there was an increase of oxygen consumption about 36.5%, whereas the average respiratory minute volume increase was about 25.3%. The purpose of another study, Morikawa *et al.* (1989) was to examine the respiratory and cardiovascular response by passively moving the legs using a weight that was attached to each leg of paraplegic patients and healthy subjects. They found that the decrease of heart rate occurs in the first five seconds of passive exercise by low intensity passive knee extension. Also a tandem bicycle was used to perform passive exercise in healthy subjects in a study by Nóbrega and Araújo (1993) that focused on heart rate response. Their results showed that high intensity passive exercise, which was performed by a tandem bicycle, resulted in a significant increase in heart rate.

In the present study, passive exercise will be performed using a fix-wheeled bike that will be secured on a treadmill operating at three different intensities (30 rpm, 60 rpm, and 80 rpm) for 3 minutes at each intensity. The focus of the present study will be to examine the cardiovascular response, particularly heart rate and stroke volume of trained and untrained individuals during passive cycle exercise.

2.5 CARDIOVASCULAR RESPONSE TO PASSIVE EXERCISE

Previous research (Morikawa *et al.*, 1989) has shown that passive exercise performed using 1-kg weight attached to the leg resulted in decreased heart rate and a slight increase in stroke volume during the first five seconds in paraplegic patients and healthy subjects. In contrast, Nóbrega and Araújo (1993) have shown that heart rate can increase substantially during passive exercise that emulated sprinting on a tandem bicycle. There is a marked difference in the results of passive exercise of the studies by Morikawa and Nóbrega. Nóbrega *et al.* (1994) have suggested that the type of passive movement which was used could explain the difference between the results of the two studies (Morikawa and Nóbrega). Thus, high intensity passive cycling movements may result in greater muscle receptors activation than low intensity passive rhythmic knee extension.

It is still debatable whether the heart rate response during passive exercise is due to central or peripheral command. Two mechanisms of neural control that underlying the cardiovascular response have been suggested; central command and exercise pressor reflex (Galbo *et al.*, 1987; Mitchell, 1985; Nóbrega *et al.*, 1994). The first mechanism suggests that the cardiovascular response to exercise is due to a direct action of central command from cardiovascular controls areas. The second mechanism suggests that the cardiovascular response to exercise is due to activity from receptors in the skeletal muscle, tendons, and joints. Furthermore, the skeletal muscles that include group III and/or IV afferents are capable of eliciting the increase of heart rate and blood pressure (Coote, 1975; Galbo *et al.*, 1987; Fernandes *et al.*, 1990; McCloskey & Mitchell, 1992; Williamson *et al.*, 1994). Results from the McMahon and McWilliam (1992) study have shown that the

receptors located in contracting muscles are responsible for shortening of the R-R interval, which is mediated by vagal withdrawal. Galbo *et al.* (1987) suggested that cardiovascular response during voluntary dynamic exercise seems to be dominated by activity from the exercising muscles. In contrast, Innes *et al.* (1992) suggested that the response of ventilatory, blood pressure, and heart rate to dynamic exercise are influenced by central neural drive. A study by Mitchell (1985) has shown that the heart rate response is exactly the same whether stimulated electrically (no central command) or voluntarily. Thus, the heart rate response must be due to receptors in the muscle (mechanoreceptors) which provide afferent feedback to cardiovascular controls.

The slight increase of stroke volume during passive knee extension occurred in paraplegic patients who lacked an intact nervous system. These results indicate that the muscle pumps were activated through passive knee extension even in paraplegic subjects. Thus, in the Morikawa study, the activation of the muscle pumps during passive knee extension may have affected the venous return resulting in greater blood move back toward the heart which led to an increase in stroke volume. A further study of passive exercise by Nóbrega *et al.* (1994) has shown that passive cycling for five minutes at 40 rpm and 60 rpm resulted in an increase in cardiac output which was due to a greater increase in stroke volume that was caused by an increase in venous return from passively moved lower limbs. The authors also suggest that cardiac output was influenced by the activation of muscle mechanoreceptors that elicited an increase in myocardial contractility. Also, they found that an increase in blood pressure occurred during passive cycling movements. They suggested that an increase in blood pressure during passive cycling movements

may have been caused by the activation of the muscle mechanoreceptors which are able to evoke a pressor response. Furthermore, they concluded that as a consequence, in humans, reflex cardiovascular response can be produced by activation of the muscle mechanoreceptors.

2.6 SUMMARY

Cardiovascular regulation is significantly influenced by aerobic exercise. The major cardiovascular adaptations to regular aerobic exercise are increased stroke volume and low resting heart rate. The stroke volume response in aerobically trained and untrained individuals is different. Furthermore, the major difference in stroke volume response between trained and untrained individuals is due to the ability of trained individuals to increased stroke volume via enhanced end-diastolic volume accompanied by hypervolemia. Most research has shown that hypervolemia occurs as a consequence of regular aerobic exercise. Nevertheless, the mechanism underlying the increase stroke volume in trained individuals remains unclear. One possible explanation may be the effectiveness of the muscle pumps to facilitate the venous return resulting in an increase in exercise stroke volume.

Passive cycle ergometry may provide a method of examining the role of venous return and exercise stroke volume through passive stimulation of the muscle pumps. Studies that have attempted to investigate the cardiac response during passive exercise have found that there was an increase in stroke volume. The increase of stroke volume may caused by increased venous return as a consequence of activation of the muscle pumps.

CHAPTER THREE: METHODS

3.1 SUBJECTS

Subjects were 10 trained male Cyclists, 10 trained Runners, and 10 fit but untrained Controls males (aged between 18-30 years). Criteria for the Cyclists and Runners were participation in a training regimen of at least four training sessions per week for greater than four years. Controls were active individuals who were not involved in a regular aerobic training program. Subject characteristics are summarized in Table 1. The study was approved by the University of Wollongong Human Experimentation Ethics Committee (see Appendix A) and all subjects provided informed consent (see Appendix B).

3.2 EXPERIMENTAL PROTOCOL

All subjects refrained from eating, smoking, and ingesting caffeine and alcohol at least three hours before testing.

3.2.1 Orientation Session

Subjects were required to read and complete a Physical Activity Readiness-Questionnaire (PAR-Q) (see Appendix F), a Human Subjects consent form (see Appendix B), and a Personal Health and Exercise History questionnaire (see Appendix C). The latter detailing the frequency, intensity, duration, and category of exercise participation, together with any past or present health problems. Finally, subjects were briefly informed of the requirements of the study (see Appendix G).

3.2.2 Testing Session

3.2.2.1 *Baseline Measures*

Cardiac variables such as stroke volume, cardiac output, and heart rate were measured simultaneously with a constant current of impedance cardiography during

rest. Two band electrodes were placed around the neck, the third electrode was placed around the thorax, and the fourth electrode was placed around the abdomen between the xiphoid and umbilicus. Three bipolar electrodes of ECG were placed around the heart in a lead II configuration (Figure 3.1). The Finapres blood pressure was also fitted (Figure 3.2). A mask to monitor breathing was placed. Then, subjects were required to rest in the supine position for 15 minutes, after which data were collected every 25 seconds during the last 5 min (Figure 3.3). Next, subjects moved to the upright position, where data were collected every 25 second for 5 minutes (Figure 3.4). During supine and upright rest subjects were requested to breathe at the required rate (3 s inhale and 3 s exhale) through breathing instructions from the tape recorder.

3.2.2.2 *Passive Cycle Exercise*

Subjects were instructed to sit on the fixed wheel bike and were requested to remain in an upright position with the left hand used to collect blood pressure placed on a tripod positioned at the level of the heart (Figure 3.5). The saddle of the fixed wheel bike was adjusted for every subject based on the following calculations: a measure of the length of the leg, from the head of femur to the feet with shoes (cm) x 0.98, then the length (cm) from these calculations was used to adjust the saddle (McLean, 1992, p.76). The cycling bouts were performed using a fixed-wheel bike that was secured to a treadmill. Subjects cycled continuously for 3 minutes at three different revolutions per minute (30, 60, and 80 rpm). During passive exercise, cardiac output, stroke volume, and heart rate was recorded every 25 seconds, whereas systolic and diastolic blood pressure was recorded every cardiac cycle. The mask to monitor breathing was placed, and subjects were requested to breathe at the

required rate (3 s inhale and 3 s exhale) through breathing instructions from the tape recorder. To ensure no contraction from the leg, three EMG electrodes were placed on the right and the left legs (see Figure 3.5). Two electrodes was placed on the anterior of the thigh and the third electrode was placed on the knee joint. During passive exercise, ECG, EMG, and breathing rate were monitored continuously and subjects were requested to keep their legs as relaxed as possible.

3.2.2.3 *Maximal Exercise Test*

All subjects exercised in the upright position on a stationary electronic-braked cycle ergometer (Excalibur) at a cadence of 70 rpm (Figure 3.6). The initial load was 30 watts (W) for the first 2 minutes and was increased by 1 W every 2 seconds thereafter. During exercise, blood pressure was recorded automatically every minute.

3.3 APPARATUS

3.3.1 Impedance Cardiography

The impedance cardiography (Minnesota Impedance Cardiogram, Model 304 B) was used to record the impedance cardiogram (ICG) using four strip electrodes placed around the neck and the chest (Miles & Gotshall, 1989).

A computer-based system processed and recorded the ECG, basal thoracic impedance between the recording electrodes (Zo: Figure 3.7), and the first derivative of the pulsatile impedance (the maximum rate of change in the impedance waveform on a given beat (dZ/dt_{max}: Figure 3.7). Specialized software using ensemble averaging was used to process the impedance cardiogram (COP, Microtronics Inc., Chapel Hill, NC).

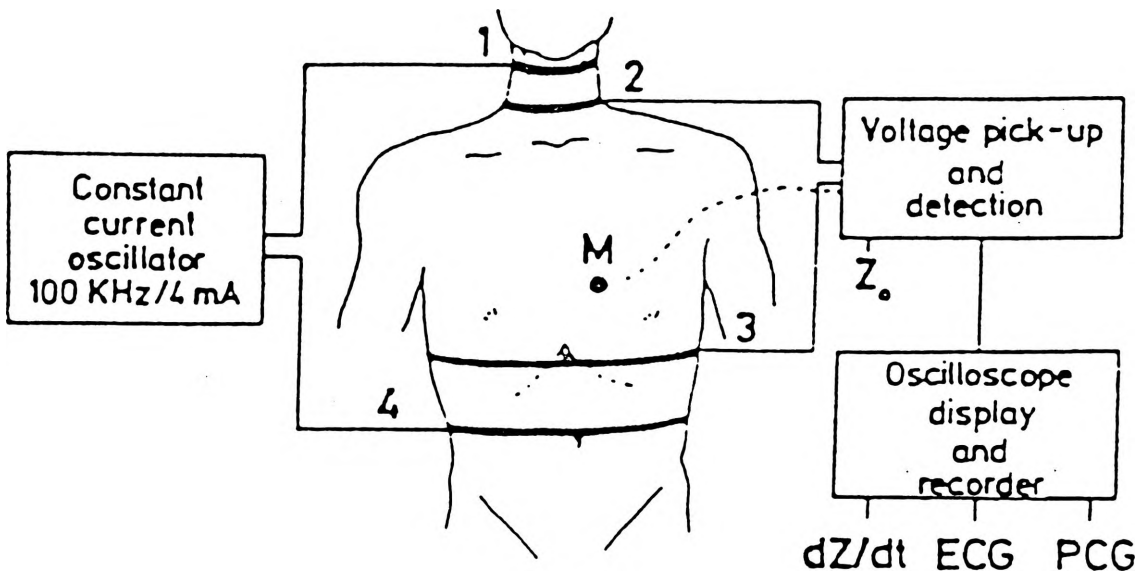

Figure 3.1 Tetrapolar configuration of aluminium electrodes used in impedance cardiography.

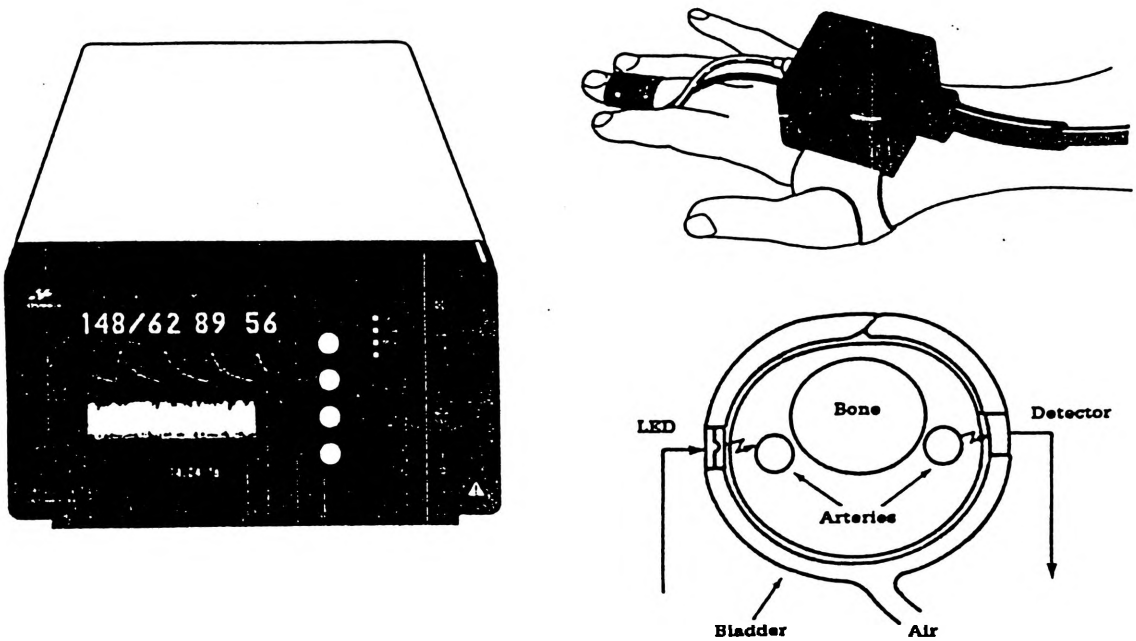

Figure 3.2 The Finapres blood pressure monitor, the finger cuff placement, and a cross section of a finger and the Finapres cuff.

Figure 3.3 A subject in the supine resting position. Note the mask and Amlab physiograph used to monitor breathing rate and tidal volume; the Finapres blood monitor; and the impedance cardiogram.

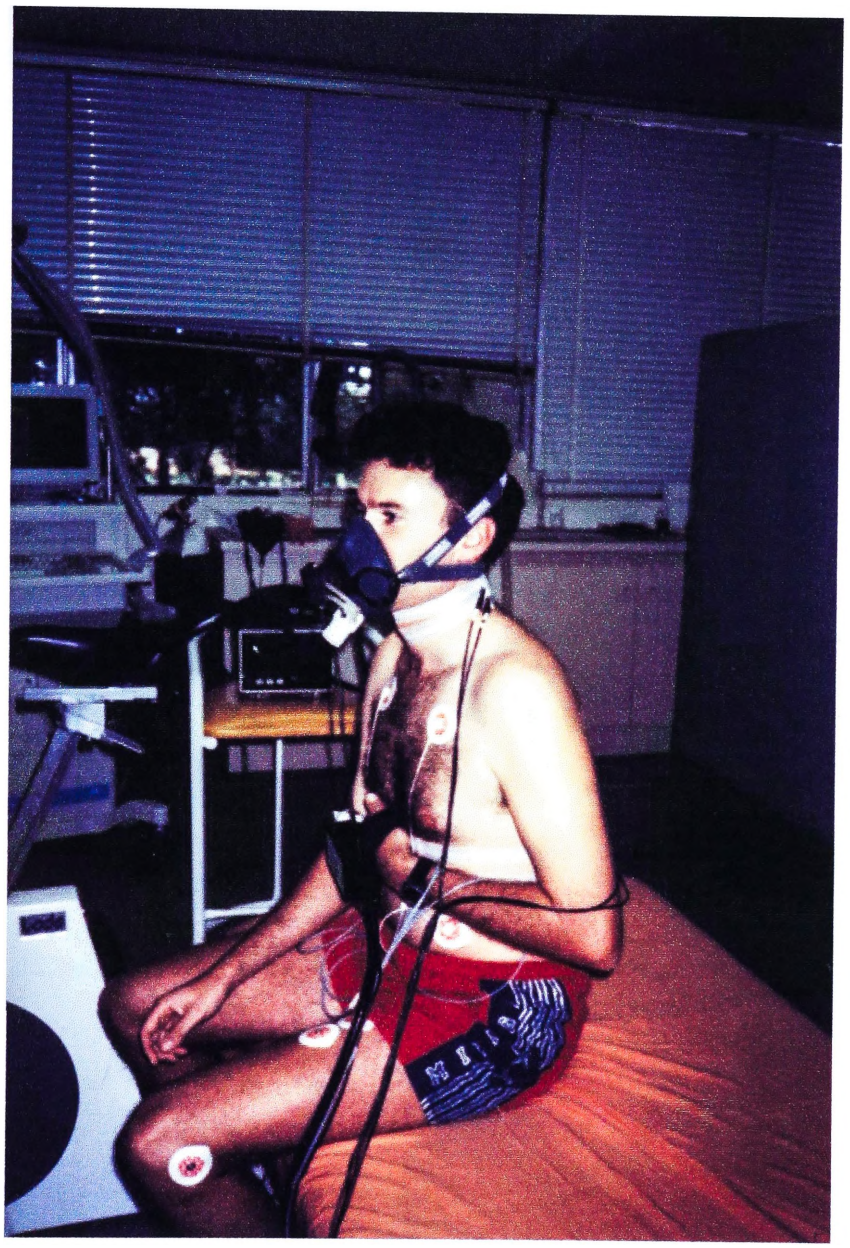

Figure 3.4 A subject in the upright rest position. Note the mask and Amlab physiograph used to monitor breathing rate and tidal volume; the Finapres blood monitor; and the impedance cardiogram.

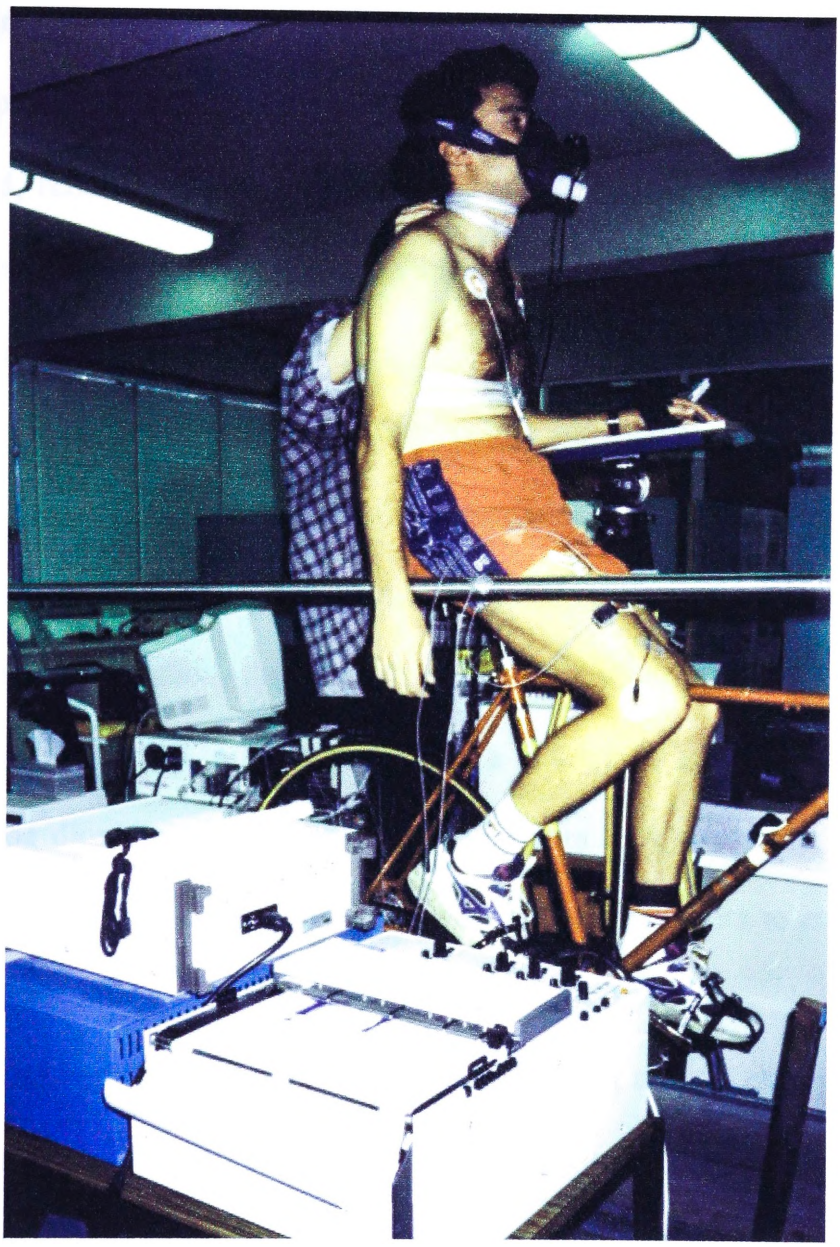

Figure 3.5 The subject set-up during passive cycle exercise. Note the subject's left hand on a tripod positioned at heart level; the face mask used to monitor breathing rate; EMG electrodes to monitor leg muscle contraction; and the ECG and impedance cardiography electrodes.

3.3.1.1 *Validation of Impedance Cardiography*

Impedance cardiography is a noninvasive method for the estimation of stroke volume (Bernstein, 1986; Gastfriend *et al.*, 1986; Miller & Horvarth, 1978) and cardiac output (Ovsyshcher *et al.*, 1993). Impedance cardiography has been shown to be a valid, reliable method for assessing stroke volume during exercise with young subjects in other laboratories (Hatcher & Srb, 1986; Sheps *et al.*, 1982; Tanaka *et al.*, 1986; Teo *et al.*, 1985).

Also, impedance cardiography was compared during exercise to carbon dioxide rebreathing using a SensorMedics metabolic cart (Model 2900) in our laboratory (McLaren, 1995). The measurement of cardiac output in this method is based on the Fick equation, that is cardiac output = $\dot{V}_{co2}/(DCO_2.\bar{v}\text{-a})$, in which $\dot{V}_{co2}$ is carbon dioxide release in ml/min, $DCO_2.\bar{v}$-a is the difference in mixed-venous to arterial carbon dioxide content. This procedure uses Fick's formula, in which oxygen parameters are substituted by carbon-dioxide parameters to estimate the carbon dioxide content of mixed venous blood during exercise.

The test consisted of submaximal exercise using a cycle ergometer. During this test a subject performed submaximal cycle ergometry at a constant pedalling speed (60 revolutions per minute); the intensity progressively increased by 0.8 kilopond every 4 minutes. The incremental increase of load during exercise allowed the subject to achieve a steady-metabolic rate that was required by the rebreathing technique (Defares, 1958). During the test rebreathing and impedance cardiography estimates of stroke volume were acquired; then the stroke volume values were compared. Figures 3.8 shows the heart rate matched values for cardiac output both at rest and during exercise up to heart rates of 150 $b.min^{-1}$ with a young subject.

3.3.1.2 *Reliability of Impedance Cardiography*

The reliability of impedance cardiography as an estimation of stroke volume in humans was tested by Ebert *et al.* (1984). Also, in our laboratory, the assessments of cardiac output and stroke volume during resting conditions in two different occasions in six subjects showed that the correlation between the two sessions was significant, $r = 0.98$; $p<0.01$, (McLaren, 1995).

3.3.2 Finapres Blood Pressure (Ohmeda 2300)

A Finapres blood pressure (Ohmeda 2300) was used to record systolic and diastolic blood pressure on a beat-by-beat basis.

3.3.2.1 *Validity and Reliability of Finapres Blood Pressure*

Blood pressure measurement through the Finapres has been shown to be valid and reliable. The validity and reliability of Finapres blood pressure has been established by comparing Finapres with brachial blood pressure (Imholz *et al.*, 1988; Imholz *et al.*, 1990; and Parati *et al.*, 1989).

3.3.3 Amlab Physiograph (Model 1.7)

An Amlab physiograph (Model 1.7) system, which was linked to a 386 PC computer, was used to record breathing and to asses the time interval between R spikes. Sampling rate was 1000 sec^{-1}.

3.3.4 Face Mask

A face mask that was attached to a flow tube (Morgan, Model AC0980) was used to record breathing pattern, rate, and tidal volume by assessing the pressure of inspired and expired air. A signal conditioning transducer (Farnell, Model 142SC01D) converted the pressure changes occurring in the flow tube to voltage.

3.3.5 Electromyography

An electromyography amplifier (Humtec, Model 100) was used to ensure there was no muscular contraction in the legs during passive cycle exercise. The response of muscle contraction was recorded through a chart recorder (Graphtec Linearcorder, FWR 3701).

3.3.6 Fixed Wheel Bike

A bike (see Figure 3.5) was secured to a Quinton treadmill. The rear wheel was a fixed wheel so that rotation of the wheels via the treadmill would result in passive movement of the subject's legs. Subject's legs were securely fixed to the pedals by toe grips.

3.3.7 Quinton (Q-Plex I)

The metabolic cart used to measure peak oxygen consumption was the Quinton gas analysis system (Model Q-Plex I) that included a Hans Rudolph pneumotachograph (Serial No. 187010), a zirconia oxide oxygen analyzer, and an infra-red carbon dioxide analyzer. Subjects breathed through a Hans Rudolph two-way valve that was connected to the Quinton through low resistance tubing. The valve was held in place by a headset and a nose clamp was fitted (see Figure 3.6).

3.3.7.1 *Validity of Quinton (Q-Plex 1)*

The Quinton Q-Plex I computerized gas analysis system has previously been validated within the present laboratory (Solomon, 1991) for oxygen and carbon dioxide concentrations, and minute ventilation by comparing expired gas volumes and gas concentrations from values obtained independently to those generated by the current system. The partial pressure of oxygen was validated against an Applied Electrochemistry oxygen analyzer (model number S-3A) and the carbondioxide

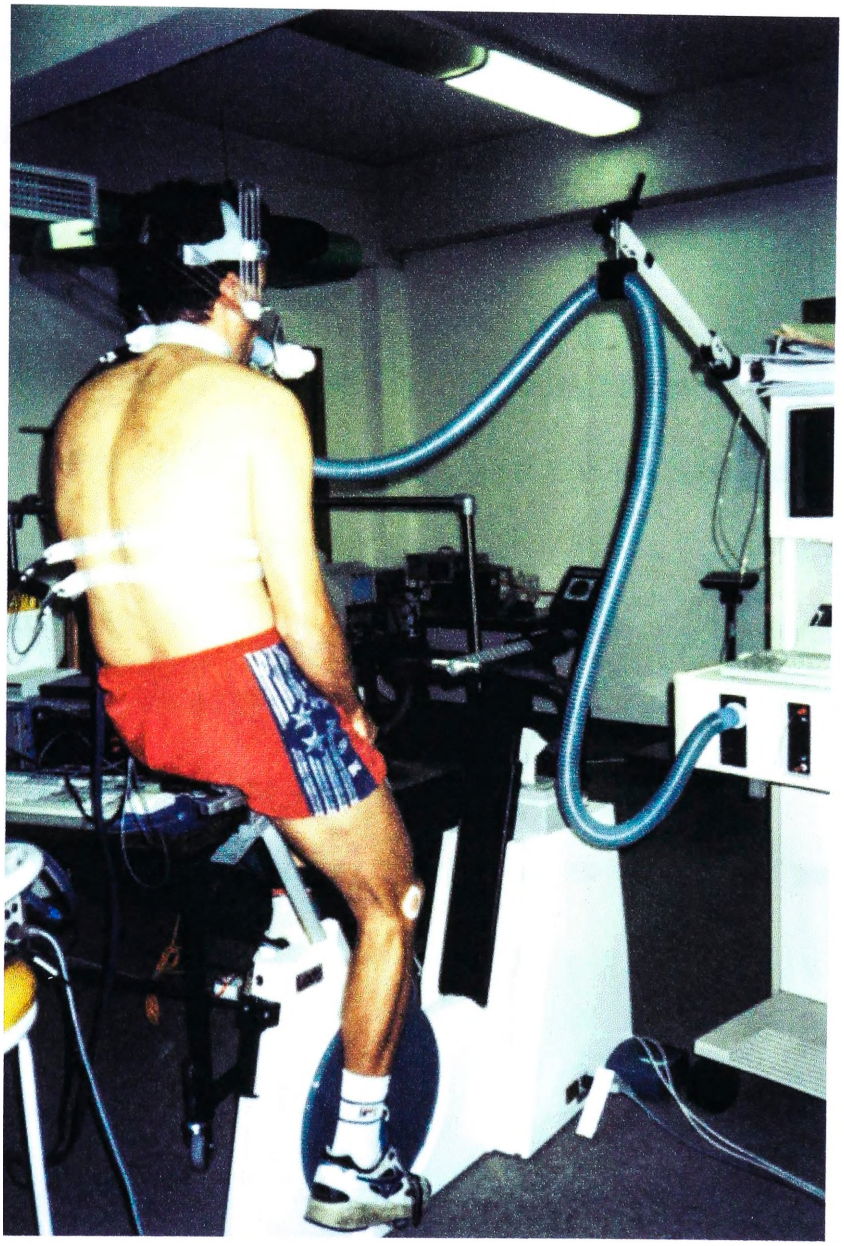

Figure 3.6 The subject set-up during maximal exercise test. The metabolic cart (Quinton system) used to measure maximal oxygen consumption; head set with Hans Rudolph valve; and the ECG and impedance cardiography electrodes.

concentrations were validated against a Beckman carbon dioxide analyzer (model number LB-2). A wedge spirometer was used to validate minute ventilation with volumes from both systems adjusted to BTPS conditions. Quinton Q-Plex 1 generated oxygen uptake and carbon dioxide production has been independently validated against other gas analysis systems (Chypchar *et al.* 1990).

3.3.8 Automated Blood Pressure

Automated blood pressure monitor (Quinton, Model 412) with the cuff was used during the maximal exercise test. The cuff was wrapped around the upper left arm with the microphone placed directly over the brachial artery. During the maximal exercise test systolic and diastolic blood pressure were displayed every minute and recorded manually.

3.3.9 Electronic-braked Cycle Ergometer

A Lode electronically-braked cycle ergometer (Excalibur Sport) was used during actual cycle exercise.

3.4 MEASURES

3.4.1 Anthropometric

Skinfold measures were taken with calipers at eight sites (abdominal, triceps, suprailiac, midaxillary, thigh, calf, biceps, and subscapular) together with height and body mass. Percentage of body fat was assessed from sum skinfold measures at four sites (triceps, biceps, subscapular, and suprailiac) based on the estimation of percent body fat by age (Durnin & Womersley, 1974).

3.4.2 Impedance Cardiogram

Cardiac performance was measured non-invasively using impedance cardiography (Minnesota Impedance Cardiograph (Model 304B). The impedance

cardiogram was collected using a tetrapolar configuration of electrodes (see Figure 3.1) (Miles & Gotshall, 1988).

3.4.3 Heart Rate

Heart rate was calculated based on the mean of inter-beat-interval of R waves from the ECG.

3.4.4 Stroke Volume

Stroke volume was determined by the Kubicek (1966) equation:

$$SV = rho.(L/Zo).\ LVET.\ dZ/dT\ max$$

where; SV = stroke volume,

rho = resistivity of blood (135 $ohm.sec^{-1}$)

L = distance between voltage electrodes (cm),

Zo = basal impedance,

LVET = left ventricular ejection time,

dZ/dT max = maximum rate of change of impedance during cardiac systole ($ohm.sec^{-1}$).

3.4.5 Stroke Index

Stroke Index (SI) was calculated based on the equation,

SI (ml/m^2) = stroke volume divided by body surface area

3.4.6 Cardiac Output

Cardiac output (CO) was calculated based on the equation,

CO (litres) = stroke volume x heart rate

3.4.7 Cardiac Index

Cardiac Index (CI) was calculated based on the equation,

CI ($litres/m^2$) = cardiac output divided by body surface area

3.4.8 Pre-Ejection Period

Pre-ejection period was computed as the interval from the ECG Q wave onset to the dZ/dT B point in milliseconds (Lewis *et al.*, 1977; Newlin & Levenson, 1979; Sherwood *et al.*, 1990; Figure 3.7).

3.4.9 Left-Ventricular Ejection Time

Left ventricular ejection time was computed as the interval from the dZ/dT B point to the dZ/dT X point in milliseconds (Lewis *et al.*, 1977; Newlin & Levenson, 1979; Sherwood *et al.*, 1990; Figure 3.7).

3.4.10 Pre-Ejection Period/Left-Ventricular Ejection Time (PEP/LVET Ratio).

PEP/LVET ratio was computed as the pre-ejection period divided by left-ventricular ejection time.

3.4.11 Systolic/Diastolic Blood Pressure

Systolic and diastolic blood pressure were recorded automatically every cardiac cycle using the Finapres blood pressure monitor.

3.4.12 Mean Arterial Pressure

Mean arterial pressure was calculated using the COP software based on the equation: 1/3 x pulse pressure (systolic pressure - diastolic) + diastolic pressure.

3.4.13 Total Peripheral Resistance

Total peripheral resistance (TPR) represents the resistance of the vasculature to blood flow and was computed according to the equation:

TPR (dyne-seconds.cm^{-5}) = MAP/CO.80.

3.4.14 Rate Pressure Product

Rate pressure product is linear to myocardial oxygen consumption and was computed according to the equation:

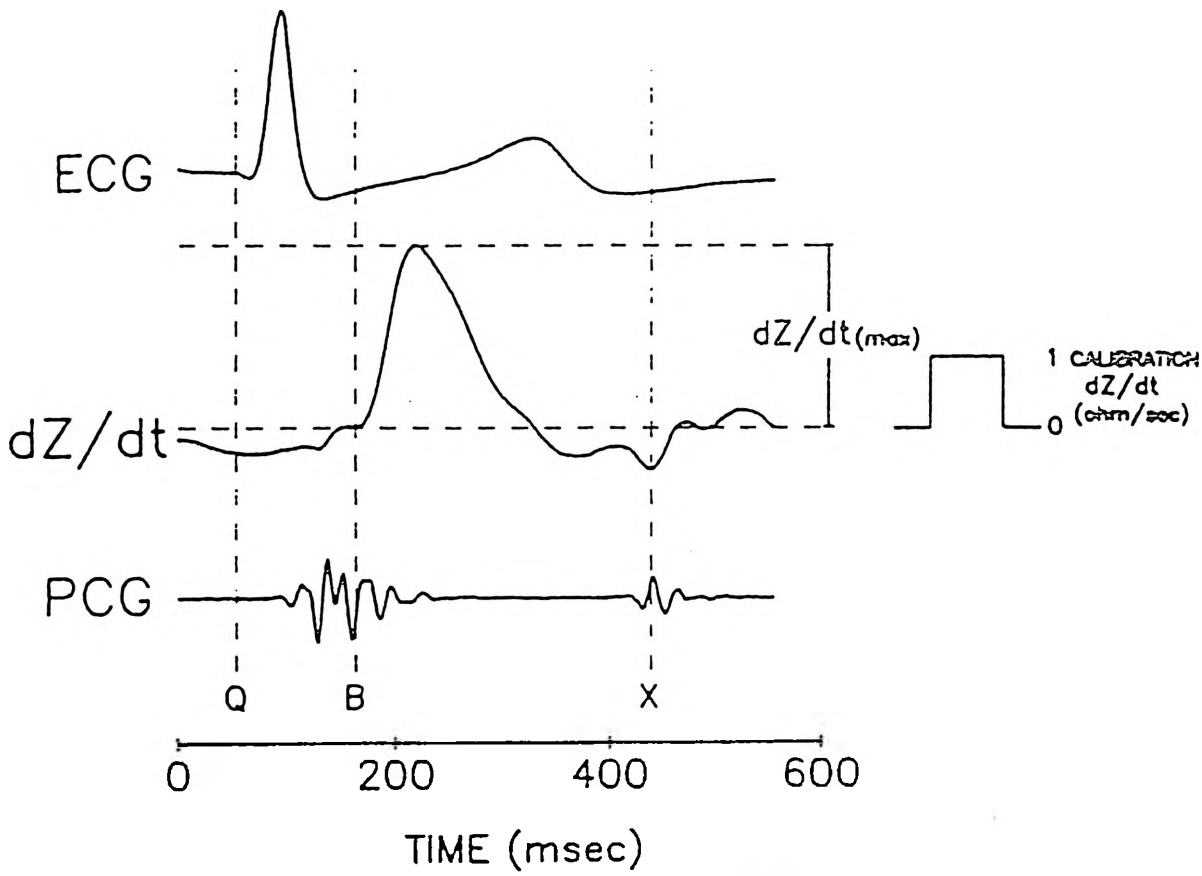

Figure 3.7 Impedance cardiogram waveform components shown are the ECG Q-wave (Q), dz/dt B-point (B), and dz/dt X-point (X). Electrocardiogram (ECG), first derivative of the pulsatile thoracic impedance signal (dz/dt), and phonocardiogram (PCG) recorded during electromechanical systole of a cardiac cycle (from Sherwood *et al.* 1990).

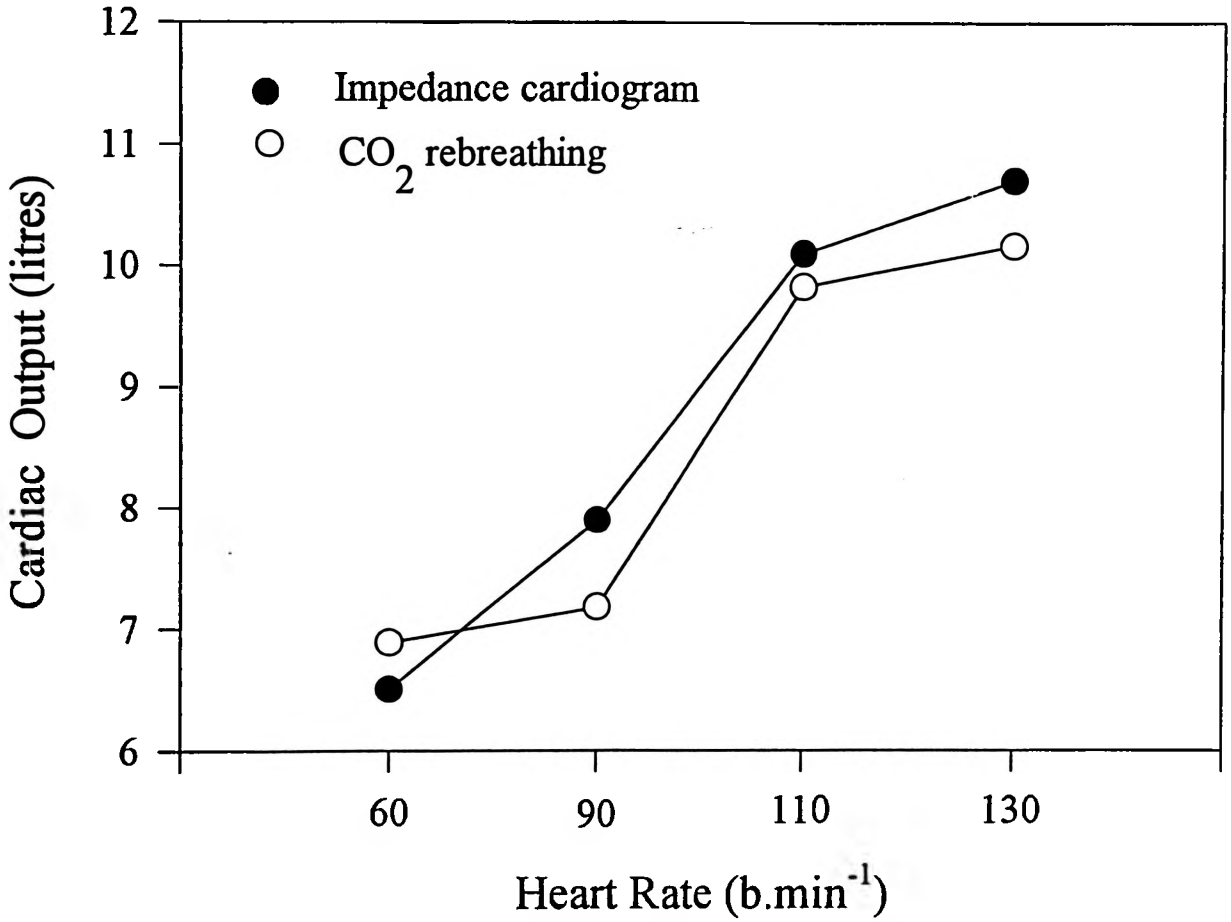

Figure 3.8 Cardiac output-heart rate relationship for one young subject. Cardiac output (litres) determined by carbon dioxide rebreathing and impedance cardiography.

rate pressure product = systolic blood pressure x heart rate / 100

3.4.15 Heart Rate Variability

Heart rate variability (HRV) reflects to a great extent, the parasympathetic effect on the heart. HRV was analyzed using a time series method (HRV_{ts}) through the MXEDIT software package (Delta-Biometrics, Inc, Bethesda, MD). Time-based data was converted from IBIs by sampling successive 200-ms intervals. During supine rest and passive cycle exercise IBIs were plotted and edited to remove and interpolate artifact and outlying values. To remove sources of variance below the two major oscillatory heart rate spectral components a band-pass filter was used.

One of these components, which is termed high frequency, was synchronized with respiration and typically occurs at frequencies at 0.12 Hz and above. The other component, called Mayer waves, is termed low frequency and is centered at around 0.10 Hz and below. The slow frequency characteristics of the Mayer waves correspond to the slow oscillations present in arterial pressure variability (Furlan *et al.*, 1993).

The natural logarithm of the band-passed variance (in msec^2) were then calculated and used as high and low frequency measures of HRV_{ts}. These estimates of HRV_{ts} appear as a linear scale ranging from 0 (minimal HRV_{ts}) to 10 (maximal HRV_{ts}) (Porges, 1985).

3.4.16 Breathing

Subjects' tidal volume were recorded and then monitored to ensure subjects produced a similar tidal volume during exercise at the different intensities. Breathing was recorded using an Amlab physiograph (Model 1.7) that was linked to a 386 PC computer collecting at 1000 sec^{-1}. Breathing pattern, rate, and tidal

volume were recorded by assessing the pressure of inspired and expired air through a flow tube (Morgan, Model AC0980) attached to a face mask. A signal conditioning transducer (Farnell, Model 142SC01D) converted the pressure changes occurring in the flow tube to voltage.

3.4.17 Peak O_2 Consumption

During the maximal oxygen uptake test, $\dot{V}o_{2peak}$ was determined as the oxygen consumption in $L.min^{-1}$ when subjects were unable to continue because of volitional exhaustion.

3.5 DATA PROCESSING

3.5.1 Impedance Cardiogram

Impedance cardiograms were initially processed using ensemble averaging to filter artifact from the impedance cardiogram every 25 seconds. Each impedance wave was edited through the edit mode of the COP software. Data for blood pressure (systolic and diastolic) during passive cycle exercise was averaged every 25 seconds then entered through the blood pressure edit mode to enable mean arterial pressure and total peripheral resistance to be calculated.

3.5.2 Statistical Analysis

The design of the study included both between and repeated measures. The between factor was Group (Cyclists, Runners, Controls) and the repeated measure was Time for each of the measures. Analysis was conducted using the SPSSPC statistical package.

During resting and passive cycle exercise the changes of cardiac variables such as heart rate, stroke volume, cardiac output, stroke index, cardiac index, mean arterial pressure, systolic and diastolic blood pressure, total peripheral resistance,

left-ventricular ejection time, pre-ejection period, PEP/LVET ratio, and rate pressure product were compared using between and repeated measures analysis of variance.

The difference of each variable between groups were compared using One-Way Analysis of Variance. A probability of $p<0.05$ was considered significant. Means and standard error of the mean for all variables at rest and during passive cycle exercise are reported in Appendix D.

One-way analysis of variance with Newman-Keuls post hoc tests were performed to examine differences in subjects' physical characteristics and cardiovascular response during passive cycle exercise when the overall omnibus F was significant. A three (Group: Cyclists, Runners, and Controls) X 4 (Time: supine and upright rest) mixed design was used to examine cardiovascular variables at rest. A three (Group: Cyclists, Runners, and Controls) X 6 (Time: six 25-seconds epochs at each intensity) mixed design was used to examine cardiovascular differences during passive exercise.

Analysis was conducted on both absolute and delta scores (calculated by subtracting the baseline measure from each response during passive cycle exercise at all intensities). For analysis that involved repeated measures the conservative *F*-test correction for degrees of freedom (Geisser & Greenhouse, 1958) was applied when symmetry assumptions were violated. When this occurred degrees of freedom for the *F*-statistic were halved and probability values calculated on these revised values. All data distributions were examined and all assumptions for each statistical analysis were tested.

CHAPTER FOUR: RESULTS

Results of this study are divided into four sections. The first section summarizes subjects' physical characteristics; the second section describes cardiovascular response during rest, whereas the next two sections describe the passive exercise cardiovascular response for all subjects and the passive exercise cardiovascular response between the three groups (Cyclists, Runners, and Controls).

4.1 SUBJECT CHARACTERISTICS

Subject characteristics are shown in Table 1. The trained groups possessed lower resting heart rate compared to Controls although no significant difference existed between groups (Table 1). The three groups showed no significant difference in height. A significant difference between groups existed for age, weight, body surface area, sum of skinfolds, percentage of body fat, and $\dot{V}o_{2peak}$ (Table 1). Runners were significantly older than Cyclists and Controls. The sum of 8 skinfolds, percentage of body fat, body surface area, and weight of Controls was significantly greater than that of Cyclists and Runners. The $\dot{V}o_{2peak}$ for the three groups is illustrated in Table 1. As expected, $\dot{V}o_{2peak}$ expressed in absolute terms, of Cyclists and Runners was significantly higher than that of Controls. Similarly, relative $\dot{V}o_{2peak}$ of Cyclists and Runners was significantly higher than that of Controls. Also, Cyclists had significantly higher absolute and relative $\dot{V}o_{2peak}$ than that of Runners.

Table 1: Physical characteristics of the subjects. Data are means with standard errors of the means in parenthesis.

Variable	Cyclists (n=10)	Runners (n=10)	Controls (n=10)
Age (yr)	20.30 (1.11)	26.20 (1.10)[a]	22.00 (0.75)
Height (cm)	175.72 (2.25)	177.44 (1.41)	179.63 (2.55)
Mass (kg)	65.42 (1.94)	72.74 (1.68)[c]	78.29 (0.92)[b]
Body surface area (m^2)	1.79 (0.04)	1.89 (0.03)[c]	1.96 (0.03)[b]
$\dot{V}O_{2peak}$ ($L.min^{-1}$)	4.95 (0.12)[c]	4.74 (0.19)[c]	3.58 (0.21)
$\dot{V}O_{2peak}$ ($ml.kg^{-1}.min^{-1}$)	76.25 (1.44)[d]	65.50 (2.16)[c]	46.31 (2.36)
Heart rate ($b.min^{-1}$)	58.43 (2.69)	57.21 (3.47)	65.38 (2.93)
Sum of 8 skinfolds (cm)	55.73 (3.31)	69.60 (5.48)[c]	85.88 (4.76)[b]
Body fat (%)	10.09 (0.57)	12.32 (0.88)	15.12 (0.87)[b]

Abbreviations: $\dot{V}O_{2peak}$ = peak oxygen consumption;

[a] = significantly greater than Cyclists and Controls ($p<0.05$);

[b] = significantly greater than Cyclists and Runners;

[c] = significantly greater than Controls;

[d] = significantly greater than Runners and Controls;

[e] = significantly greater than Cyclists.

4.2 BASELINE CARDIOVASCULAR RESPONSE

One-way Anova on supine and upright resting heart rate, stroke volume, stroke index, cardiac output, cardiac index, left-ventricular ejection time, systolic blood pressure, and rate pressure product as well as vagal influence on the heart (HRV_{ts}) revealed no significant differences between the three groups (Table 2; Table 3).

Analysis on supine pre-ejection period, $\underline{F}(2, 27) = 3.69$, $p=0.04$, PEP/LVET ratio, $\underline{F}(2, 27) = 3.20$, $p=0.05$, indicated a significant Group main effect. Post hoc analysis indicated that Cyclists and Runners possessed significantly greater pre-ejection period than Controls (Table 2; see Figure 4.17), also Runners possessed significantly greater PEP/LVET ratio compared to Controls. However, no significant different existed for PEP/LVET ratio during upright rest (Table 3; see Figure 4.18).

Analysis on supine resting diastolic blood pressure and mean arterial pressure revealed no significant difference between groups. However, diastolic blood pressure, $\underline{F}(2, 27) = 3.95$, $p=0.031$, showed a significant Group main effect during upright rest. Post hoc analysis showed that Runners possessed significantly greater diastolic blood pressure (Table 3; see Figure 4.19) and mean arterial pressure (Table 3; see Figure 4.20) during upright rest compared to Controls.

Analysis on upright resting total peripheral resistance revealed a significant Group main effect, $\underline{F}(2, 27) = 3.44$, $p=0.047$. Post hoc analysis indicated that total peripheral resistance of Runners was significantly greater than Controls (Table 3; see Figure 4.21). However, no significant difference was found during supine rest between the three groups (Table 2).

Table 2 : Baseline measures (supine rest). Data are means with standard errors of the means in parenthesis.

Variable	Cyclists	Runners	Controls
Heart Rate ($b.min^{-1}$)	58.43 (2.69)	57.21 (3.47)	65.38 (2.93)
Stroke Volume (ml)	134.29 (6.96)	127.75 (7.26)	121.34 (8.07)
Stroke Index (ml/BSA)*	59.61 (4.69)	49.80 (2.39)	46.11 (2.34)
Cardiac Output (L/min)	7.80 (0.45)	7.24 (0.52)	7.79 (0.38)
Cardiac Index (ml/BSA)*	4.35 (0.24)	3.82 (0.26)	3.97 (0.19)
Pre-Ejection Period (msec)	77.40 (5.75)[a]	80.00 (3.13)[a]	64.23 (3.91)
Left-Ventricular Ejection Time (msec)	297.27 (4.34)	309.33 (5.95)	302.27 (6.06)
PEP/LVET ratio	0.263 (0.02)	0.259 (0.01)[a]	0.213 (0.01)
Systolic Blood Pressure (mmHg)	113.53 (3.11)	119.43 (2.52)	112.45 (2.36)
Diastolic Blood Pressure (mmHg)	66.12 (4.12)	69.53 (3.61)	59.15 (3.73)
Mean Arterial Pressure (mmHg)	81.67 (3.74)	85.88 (3.14)	75.92 (3.48)
Total Peripheral Resistance ($dynes.s/cm^{-5}$)	788.48 (103.71)	994.10 (80.00)	692.83 (96.92)
Rate Pressure Product (HRxSBP/100)	66.43 (3.79)	68.65 (5.05)	73.40 (3.28)
Time Series (msec^2) at low and high frequency	3.43 (0.33) 8.49 (0.37)	3.42 (0.29) 8.51 (0.29)	2.91 (0.34) 8.10 (0.31)

[a] = significantly greater than Controls ($p<0.05$);

* BSA = body surface area.

Table 3 : Baseline measures (upright rest). Data are means with the standard errors means in the parenthesis.

Variable	Cyclists	Runners	Controls
Heart Rate ($b.min^{-1}$)	63.03 (3.00)	63.37 (3.36)	71.23 (2.33)
Stroke Volume (ml)	106.09 (7.16)	94.32 (5.00)	90.77 (5.16)
Stroke Index (ml/BSA)*	61.57 (4.75)	49.43 (3.68)	50.33 (2.70)
Cardiac Output (L/min)	6.57 (0.36)	5.90 (0.34)	6.38 (0.28)
Cardiac Index (ml/BSA)*	3.67 (0.22)	3.11 (0.17)	3.25 (0.13)
Pre-Ejection Period (msec)	90.58 (6.88)[a]	101.25 (4.01)[a]	82.75 (5.32)
Left-Ventricular Ejection Time (msec)	274.20 (7.01)	274.53 (7.66)	268.80 (5.35)
PEP/LVET ratio	0.337 (0.03)	0.368 (0.02)[a]	0.310 (0.02)
Systolic Blood Pressure (mmHg)	129.79 (3.53)	137.10 (4.66)	127.89 (3.38)
Diastolic Blood Pressure (mmHg)	84.46 (4.95)	90.55 (4.26)[a]	74.55 (2.63)
Mean Arterial Pressure (mmHg)	99.23 (4.33)	105.78 (4.31)[a]	92.08 (2.57)
Total Peripheral Resistance ($dynes.s/cm^{-5}$)	1178.00 (139.11)	1501.50 (98.32)[a]	1088.60 (138.77)
Rate Pressure Product (HRxSBP/100)	82.00 (5.02)	87.07 (5.97)	91.44 (4.71)

[a] = significantly greater than Controls ($p<0.05$);

* BSA = body surface area.

4.3 CARDIOVASCULAR RESPONSE DURING PASSIVE CYCLE EXERCISE FOR ALL SUBJECTS COMBINED

4.3.1 Heart Rate

Analysis on heart rate response to passive cycle exercise revealed significant Time main effects at the low, $\underline{F}(6, 174) = 9.65$, $p=0.0001$, medium, $\underline{F}(6, 174) = 17.56$, $p=0.0001$, and high intensities, $\underline{F}(6, 174) = 24.45$, $p=0.0001$. Figure 4.1 shows that heart rate response for all subjects combined was significantly higher than upright rest during all three passive cycle exercise intensities (Table E-1, Appendix E).

4.3.2 Stroke Volume

Analysis on stroke volume response to passive cycle exercise revealed no significant Time main effects at the low, $\underline{F}(6, 174) = 1.23$, $p>0.05$, and medium intensities, $\underline{F}(6, 174) = 1.56$, $p>0.05$. In contrast, stroke volume at the high intensity was significantly higher than baseline, $\underline{F}(6, 174) = 5.49$, $p=0.0001$. As can be seen in Figure 4.2, stroke volume for all subjects combined were significantly higher than upright rest during high intensity passive cycle exercise (Table E-2, Appendix E).

4.3.3 Stroke Index

Analysis on stroke index response to passive cycle exercise revealed no significant Time main effects at the low, $\underline{F}(6, 174) = 1.26$, $p>0.05$, and the medium intensities, $\underline{F}(6, 174) = 1.60$, $p>0.05$. In contrast, stroke index was significantly higher than upright rest at the high intensity, $\underline{F}(6, 174) = 5.45$, $p=0.0001$ (Figure 4.3; Table E-3, Appendix E).

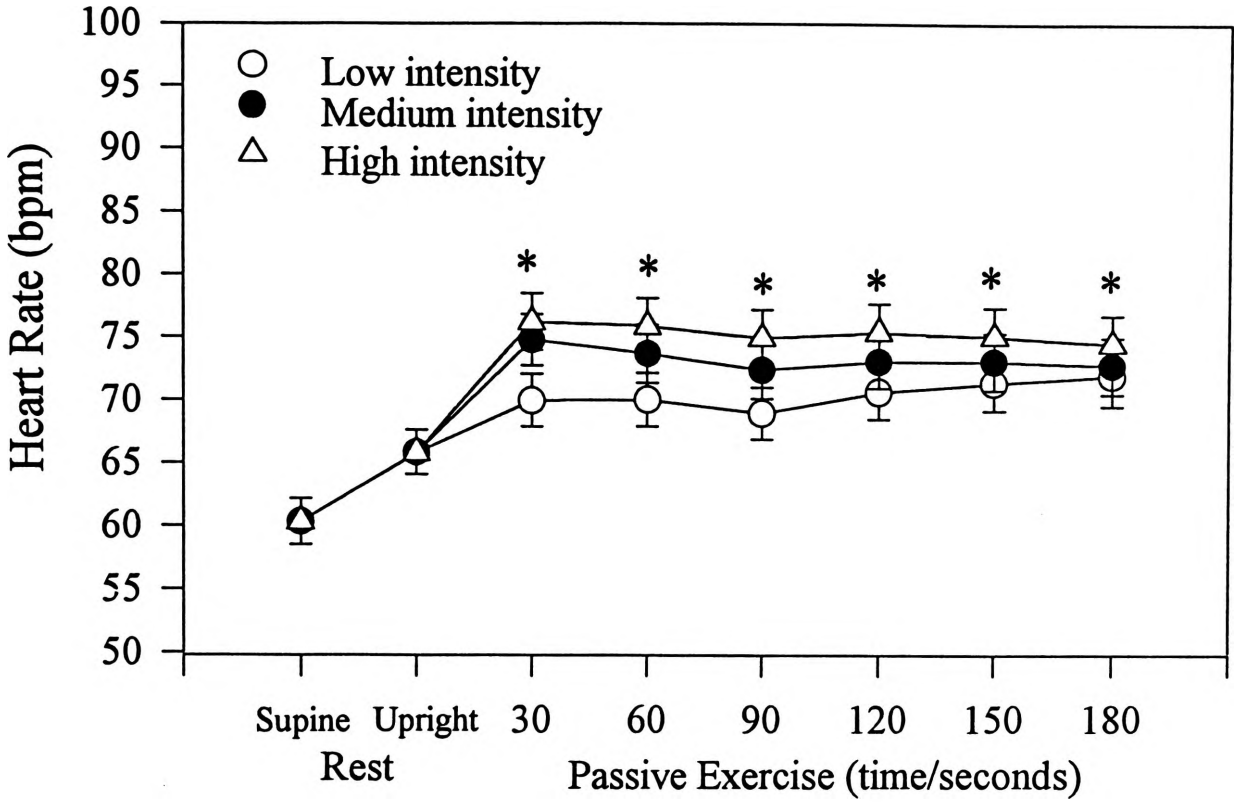

Figure 4.1 Heart rate response at rest and during passive cycle exercise at all intensities for the three groups combined. Data are means with standard errors of the means; (*) indicate that heart rate response during passive cycle exercise at the low, medium, and high intensities was significantly higher than heart rate response during upright rest.

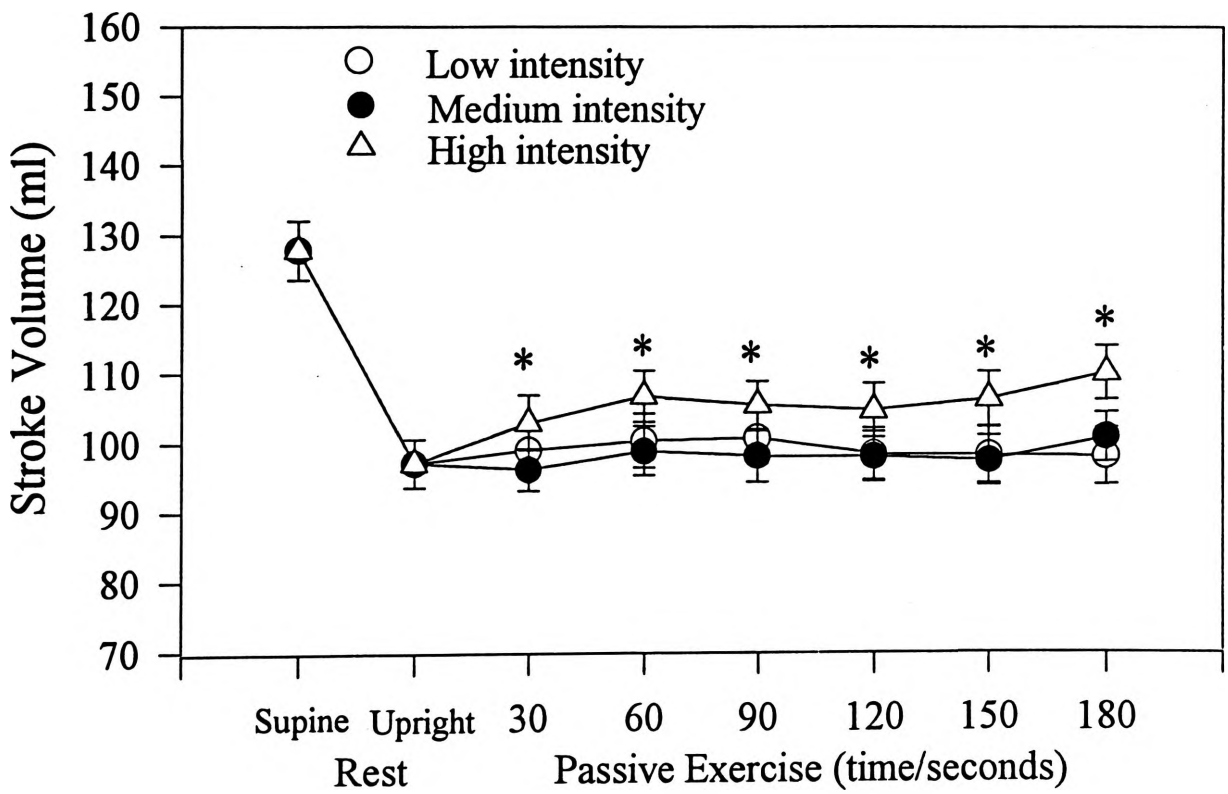

Figure 4.2 Stroke volume response at rest and during passive cycle exercise at all intensities for the three groups combined. Data are means with standard errors of the means; (*) indicate that stroke volume response was significantly higher at the high intensity passive cycle exercise than stroke volume response during upright rest.

4.3.4 Cardiac Output

Analysis on cardiac output response to passive cycle exercise revealed significant Time main effects at the low, $F(6, 174) = 5.68$, $p=0.0001$, medium, $F(6, 174) = 10.81$, $p=0.0001$, and high intensities, $F(6, 174) = 19.06$, $p=0.0001$. Figure 4.4 shows that cardiac output response for all subjects combined was significantly higher than upright rest during all three passive cycle exercise intensities (Table E-4, Appendix E).

4.3.5 Cardiac Index

Analysis on cardiac index response to passive cycle exercise revealed significant Time main effects at the low, $F(6, 174) = 5.95$, $p=0.0001$, medium, $F(6, 174) = 10.92$, $p=0.0001$, and high intensities, $F(6, 174) = 19.36$, $p=0.0001$. Cardiac index for all subjects combined was significantly higher than upright rest during all three passive cycle exercise intensities (Figure 4.5; Table E-5, Appendix E).

4.3.6 Pre-Ejection Period

Analysis on pre-ejection period response to passive cycle exercise revealed significant Time main effects at the low, $F(6, 174) = 2.97$, $p=0.009$, medium, $F(6, 174) = 3.17$, $p=0.006$, and high intensities, $F(6, 174) = 11.19$, $p=0.0001$. Pre-ejection period values for all subjects combined were significantly lower than upright rest during all three passive cycle exercise intensities (Figure 4.6; Table E-6, Appendix E).

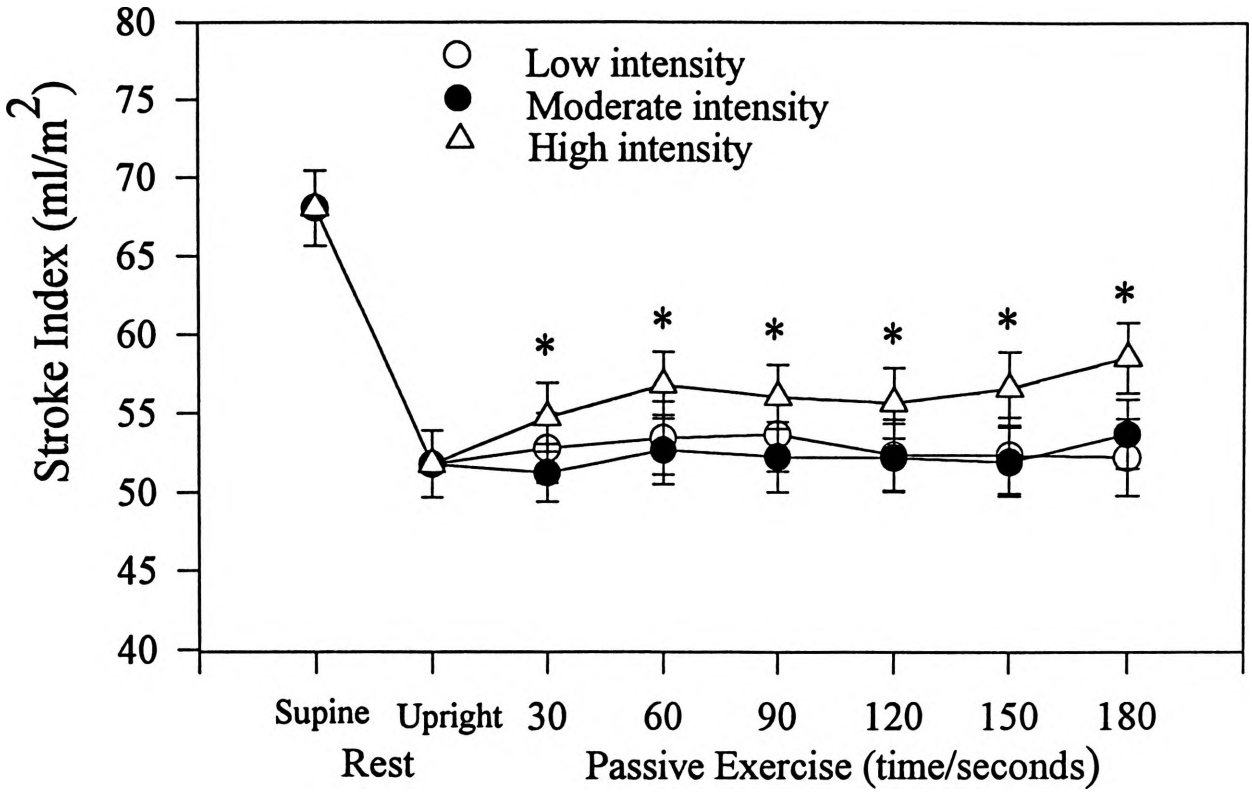

Figure 4.3 Stroke index response at rest and during passive cycle exercise at all intensities for the three groups combined. Data are means with standard errors of the means; (*) indicate that stroke index response was significantly higher at the high intensity passive cycle exercise than stroke index response during upright rest.

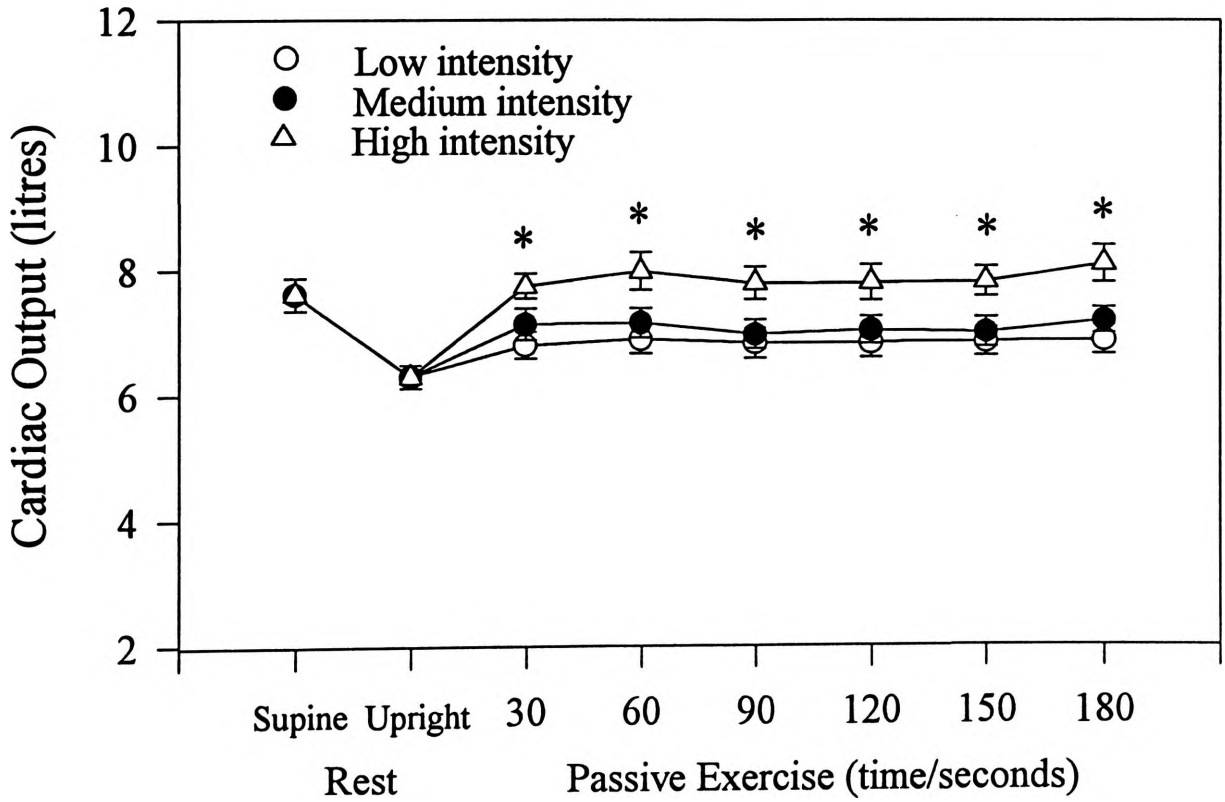

Figure 4.4 Cardiac output response at rest and during passive cycle exercise at all intensities for the three groups combined. Data are means with standard errors of the means; (*) indicate that cardiac output response during passive cycle exercise at the low, medium, and high intensities was significantly higher than cardiac output response during upright rest.

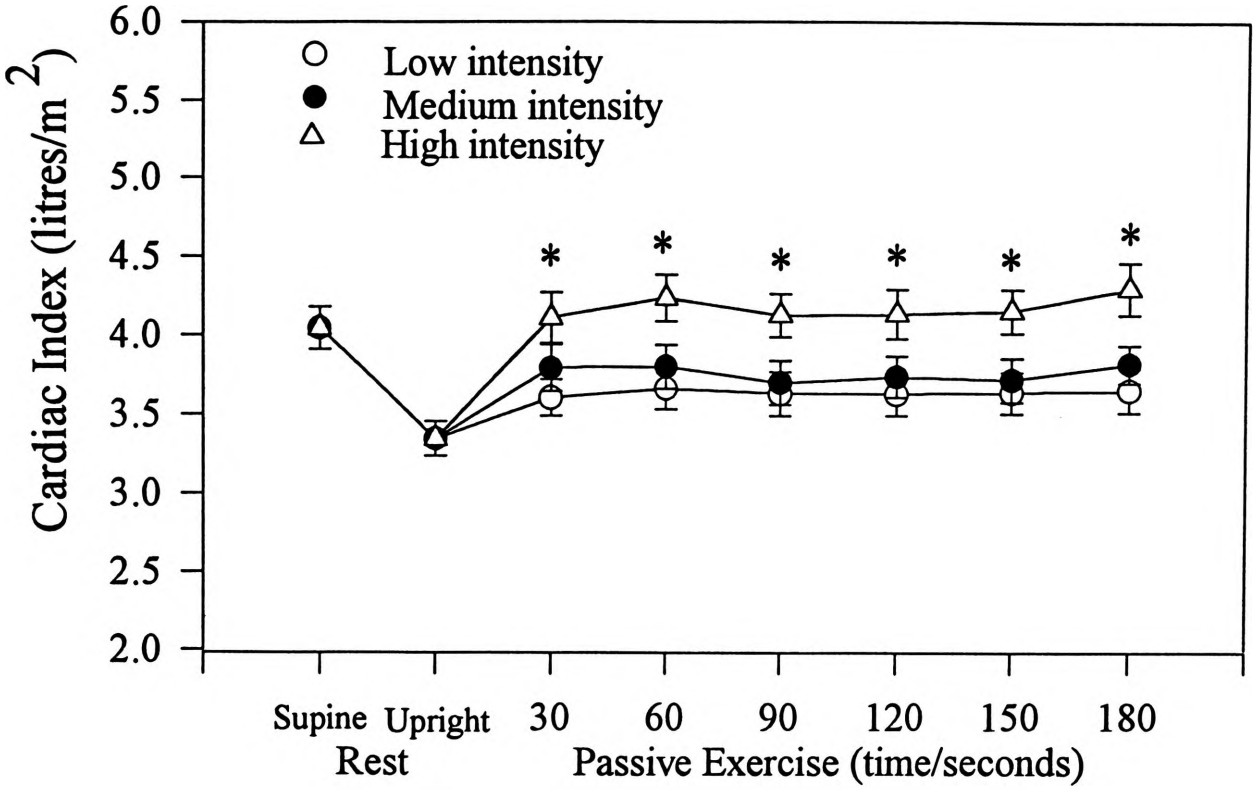

Figure 4.5 Cardiac index response at rest and during passive cycle exercise at all intensities for the three groups combined. Data are means with standard errors of the means; (*) indicate that cardiac index response during passive cycle exercise at the low, medium, and high intensities was significantly higher than cardiac index during upright rest.

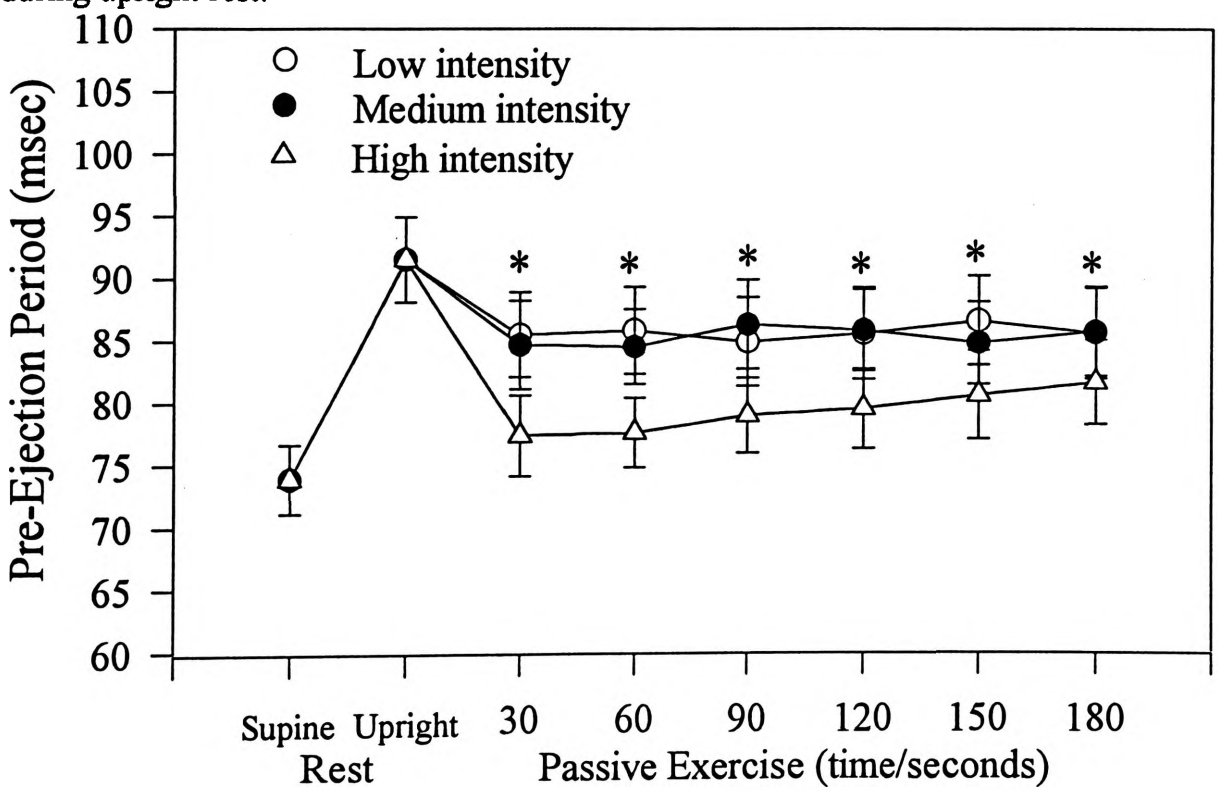

Figure 4.6 Pre-ejection period response at rest and during passive cycle exercise at all intensities for the three groups combined. Data are means with standard errors of the means; (*) indicate that pre-ejection period response during passive cycle exercise at the low, medium, and high intensities was significantly lower than pre-ejection period response during upright rest.

4.3.7 Left-Ventricular Ejection Time

Analysis on left-ventricular ejection time to passive cycle exercise indicated a significant Time main effect at the low, $F(6, 174) = 8.69$, $p=0.0001$, and medium intensities, $F(6, 174) = 5.37$, $p=0.0001$. However, no significant difference existed at the high intensity $F(6, 174) = 1.08$, $p>0.05$. Post hoc analysis revealed that all groups recorded significantly shorter left-ventricular ejection time at the low and medium intensities compared to supine and upright rest (Figure 4.7; Table E-7, Appendix E).

4.3.8 Pre-Ejection Period/Left-Ventricular Ejection Time (PEP/LVET Ratio).

Analysis on PEP/LVET ratio to passive cycle exercise indicated no significant Time main effects at the low, $F(6, 174) = 0.49$, $p>0.05$, and medium intensities, $F(6, 174) = 0.76$, $p>0.05$. However, PEP/LVET ratio for all subjects combined was significantly lower than upright rest only at the high intensity, $F(6, 174) = 7.25$, $p=0.0001$, (Figure 4.8; Table E-8, Appendix E).

4.3.9 Systolic Blood Pressure

Analysis on systolic blood pressure response to passive cycle exercise revealed significant Time main effects at the low, $F(6, 174) = 8.77$, $p=0.0001$, medium, $F(6, 174) = 9.75$, $p=0.0001$, and high intensities, $F(6, 174) = 9.08$, $p=0.0001$. Figure 4.9 shows that systolic blood pressure for all subjects combined was significantly higher than upright rest during all three passive cycle exercise intensities (Table E-9, Appendix E).

4.3.10 Diastolic Blood Pressure

Analysis on diastolic blood pressure response to passive cycle exercise revealed significant Time main effects at the low, $F(6, 174) = 9.49$, $p=0.0001$,

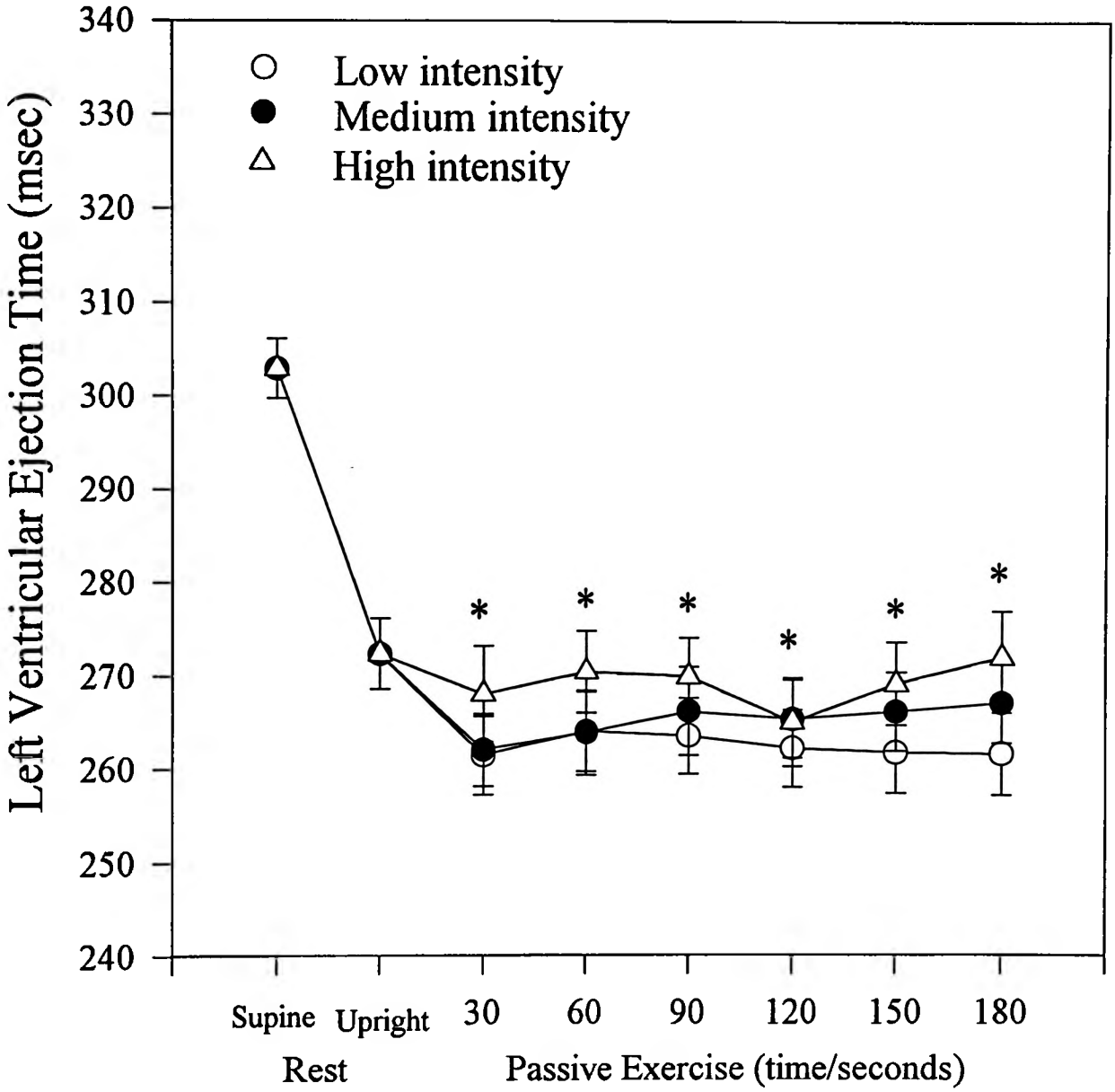

Figure 4.7 Left-ventricular ejection time response at rest and during passive cycle exercise at all intensities for the three groups combined. Data are means with standard errors of the means; (*) indicate that left-ventricular ejection time response at the low and medium intensities of passive cycle exercise was significantly shorter than left-ventricular ejection time response during upright rest.

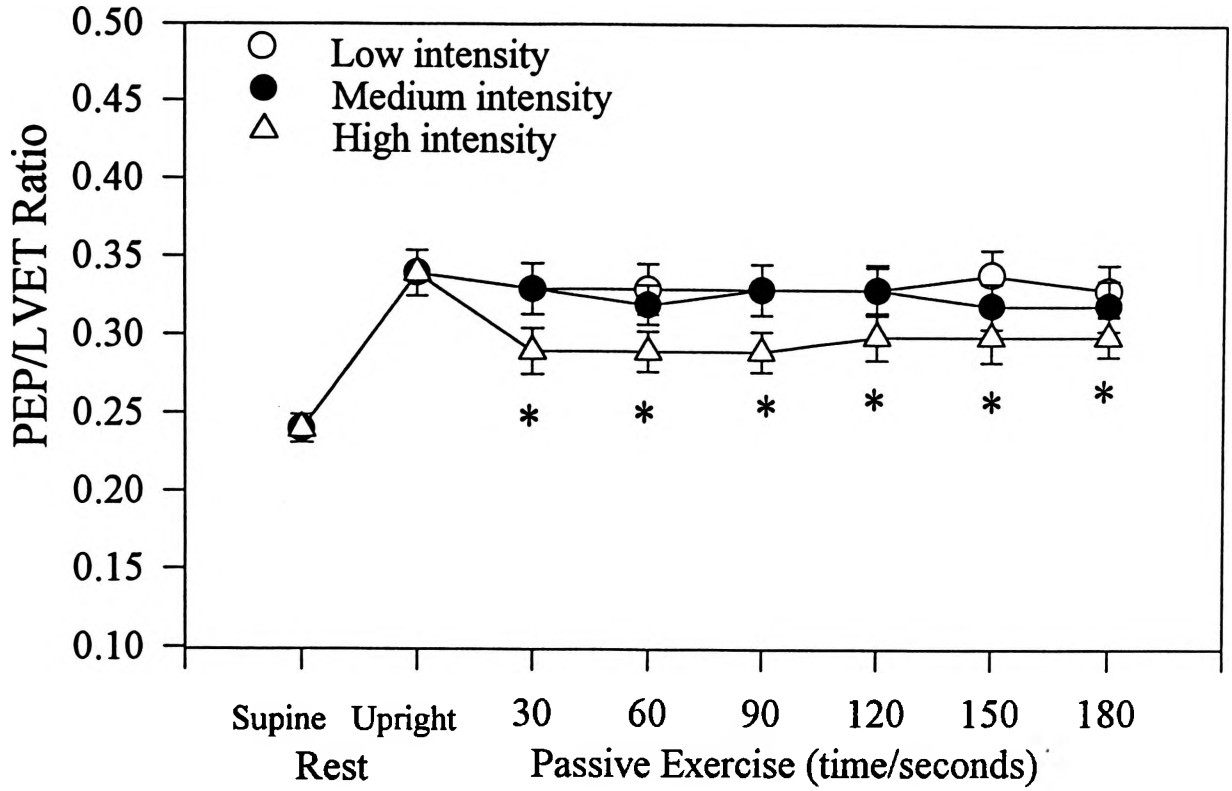

Figure 4.8 PEP/LVET ratio response at rest and during passive cycle exercise at all intensities for the three groups combined. Data are means with standard errors of the means; (*) indicate that PEP/LVET ratio response was significantly lower at the high intensity passive cycle exercise than PEP/LVET ratio response during upright rest.

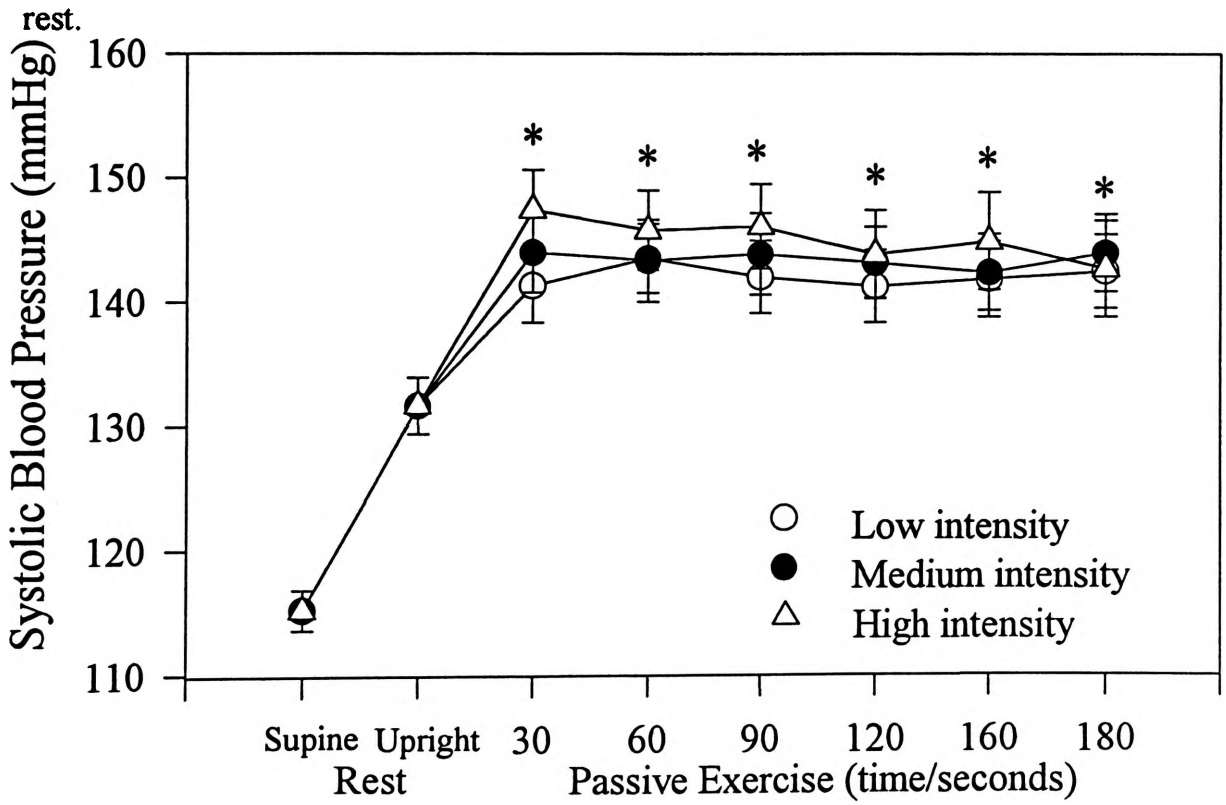

Figure 4.9 Systolic blood pressure response at resst and during passive cycle exercise at all intensities for the three groups combined. Data are means with standard errors of the means; (*) indicate that systolic blood pressure response during passive cycle exercise at the low, medium, and high intensities was significantly higher than systolic blood pressure response during upright rest.

medium, $F(6, 174) = 12.25$, $p=0.0001$, and high intensities, $F(6, 174) = 8.67$, $p=0.0001$. As can be seen in Figure 4.10, diastolic blood pressure for all subjects combined was significantly higher than upright rest during all three passive exercise intensities (Table E-10, Appendix E).

4.3.11 Mean Arterial Pressure

Analysis on mean arterial pressure response to passive cycle exercise revealed significant Time main effects at the low, $F(6, 174) = 10.58$, $p=0.0001$, medium, $F(6, 174) = 12.58$, $p=0.0001$, and high intensities, $F(6, 174) = 9.81$, $p=0.0001$. Mean arterial pressure for all subjects combined was significantly higher than upright rest during all three passive cycle exercise intensities (Figure 4.11; Table E-11, Appendix E).

4.3.12 Total Peripheral Resistance

Analysis on total peripheral resistance response to passive cycle exercise revealed no significant Time main effects at the low, $F(6, 174) = 0.09$, $p>0.05$, and medium intensities, $F(6, 174) = 0.78$, $p>0.05$. However, total peripheral resistance was significantly lower, $F(6, 174) = 5.39$, $p=0.0001$, than upright rest at the high intensity passive cycle exercise (Figure 4.12; Table E-12, Appendix E).

4.3.13 Rate Pressure Product

Analysis on rate pressure product to passive cycle exercise revealed significant Time main effects at the low, $F(6, 174) = 18.88$, $p=0.0001$, medium, $F(6, 174) = 25.94$, $p=0.0001$, and high intensities, $F(6, 174) = 30.15$, $p=0.0001$. As shown in Figure 4.13, rate pressure product for all subjects combined was significantly higher than upright rest during all three passive cycle exercise intensities (Table E-13, Appendix E).

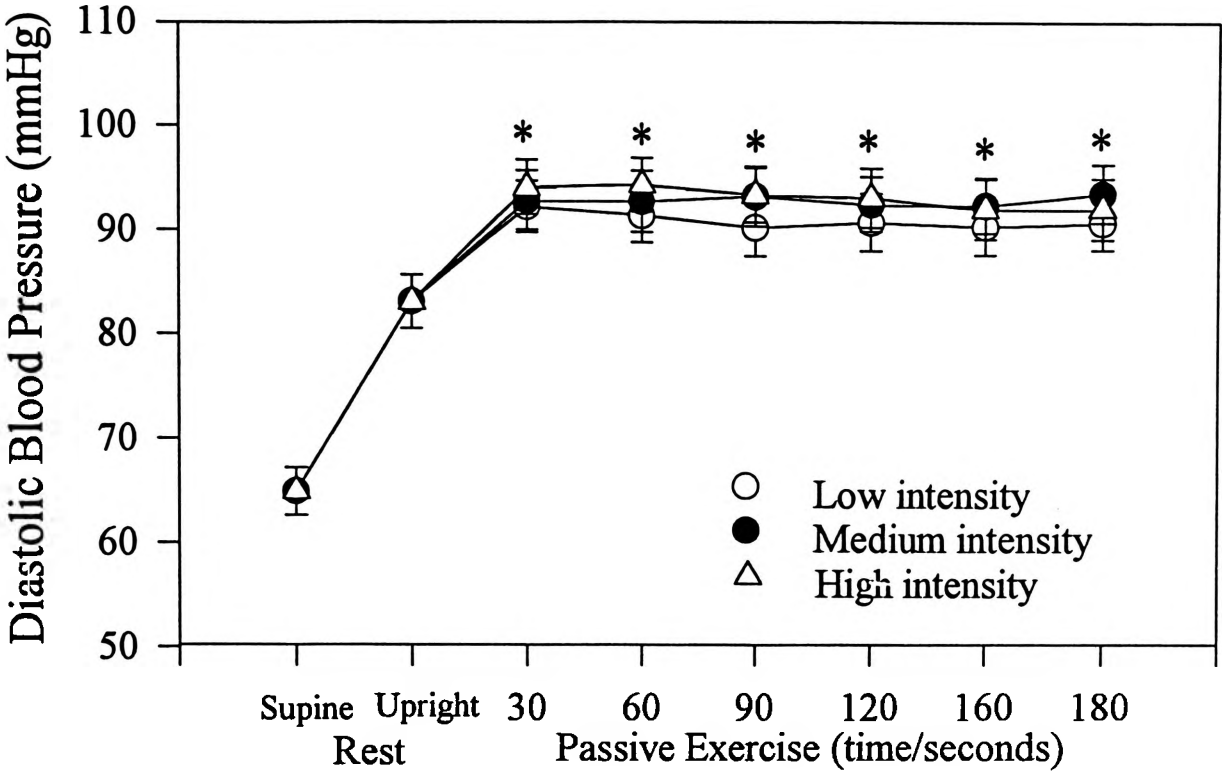

Figure 4.10 Diastolic blood pressure response at rest and during passive cycle exercise at all intensities for the three groups combined. Data are means with standard errors of the means; (*) indicate that diastolic blood pressure response during passive cycle exercise at the low, medium, and high intensities was significantly higher than diastolic blood pressure response during upright rest.

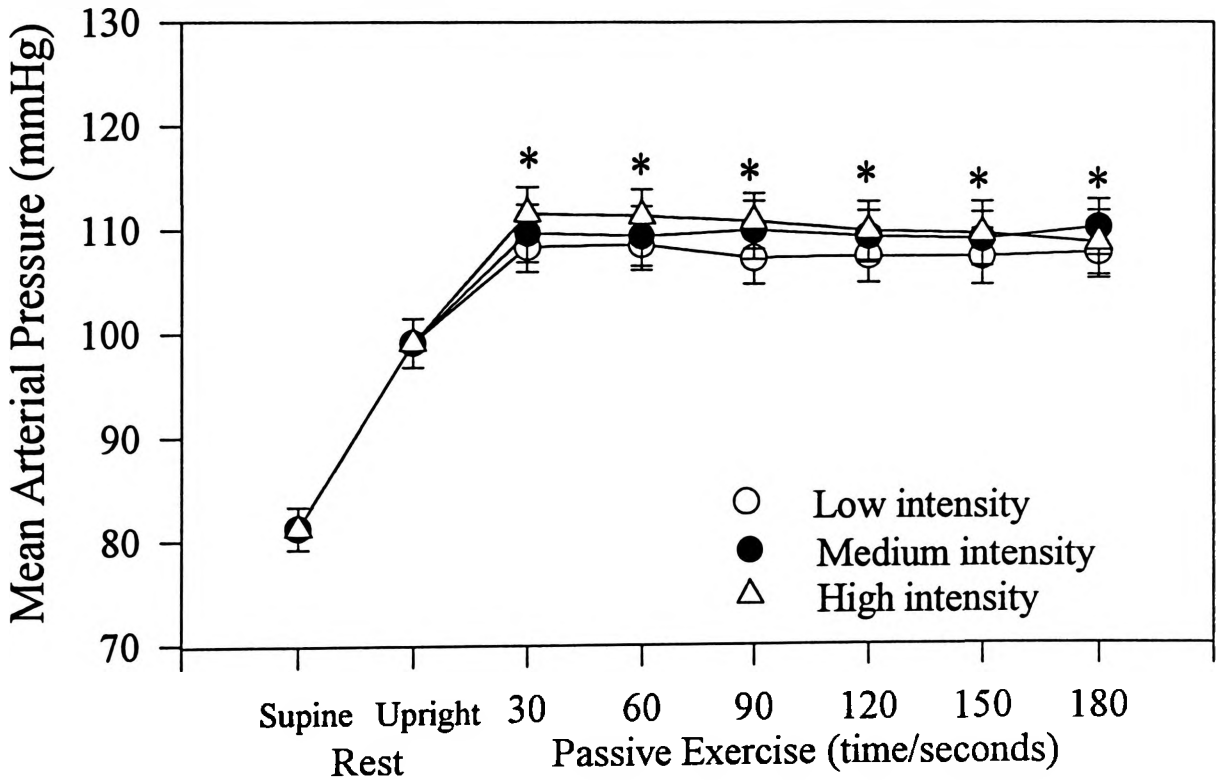

Figure 4.11 Mean arterial pressure response at rest and during passive cycle exercise at all intensities for the three groups combined. Data are means with standard errors of the means; (*) indicate that mean arterial pressure response during passive cycle exercise at the low, medium, and high intensities was significantly higher than mean arterial pressure response during upright rest.

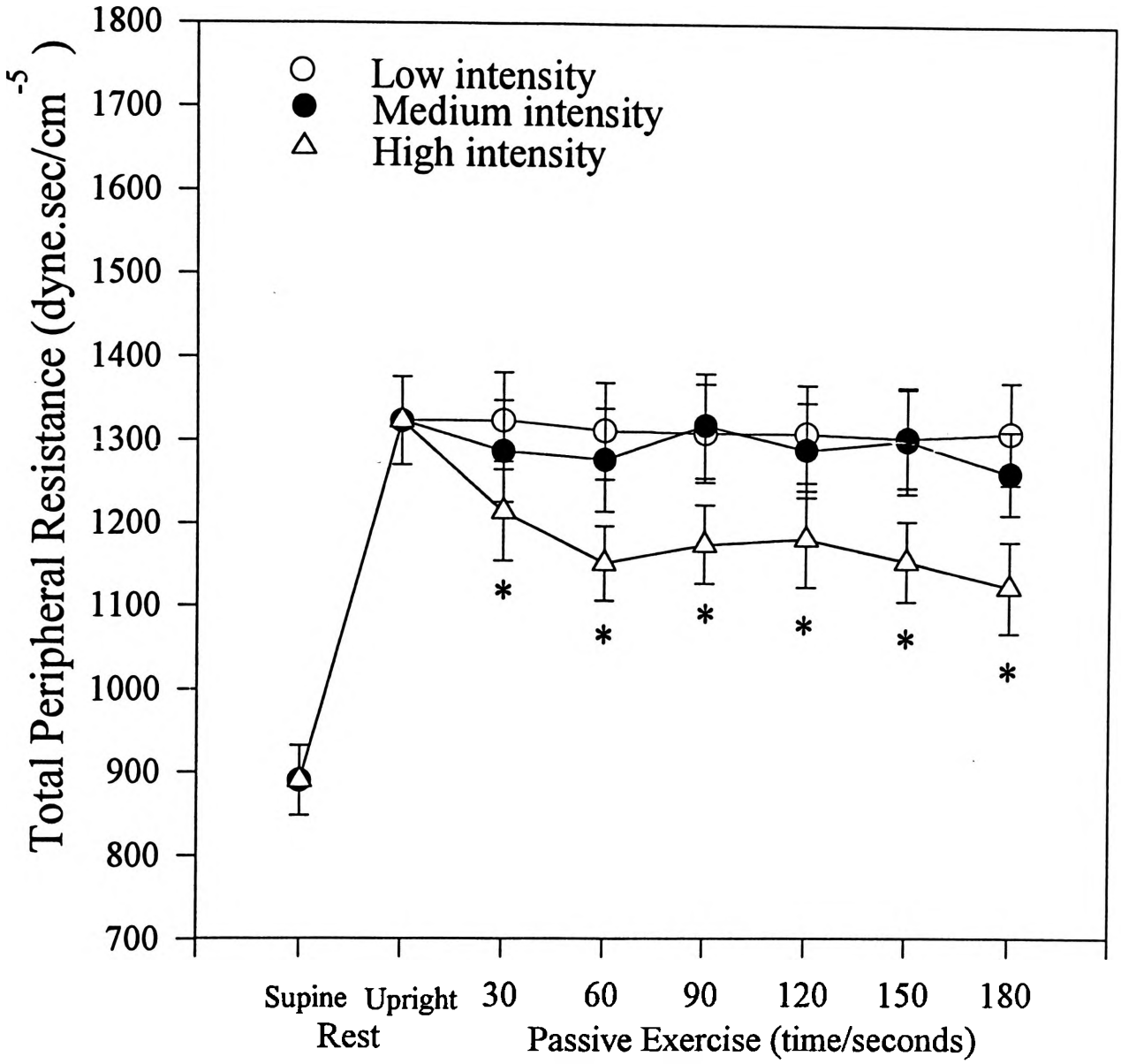

Figure 4.12 Total peripheral resistance response at rest and during passive cycle exercise at all intensities for the three groups combined. Data are means with standard errors of the means; (*) indicate that total peripheral resistance response at the high intensity passive cycle exercise was significantly lower than total peripheral resistance response during upright rest.

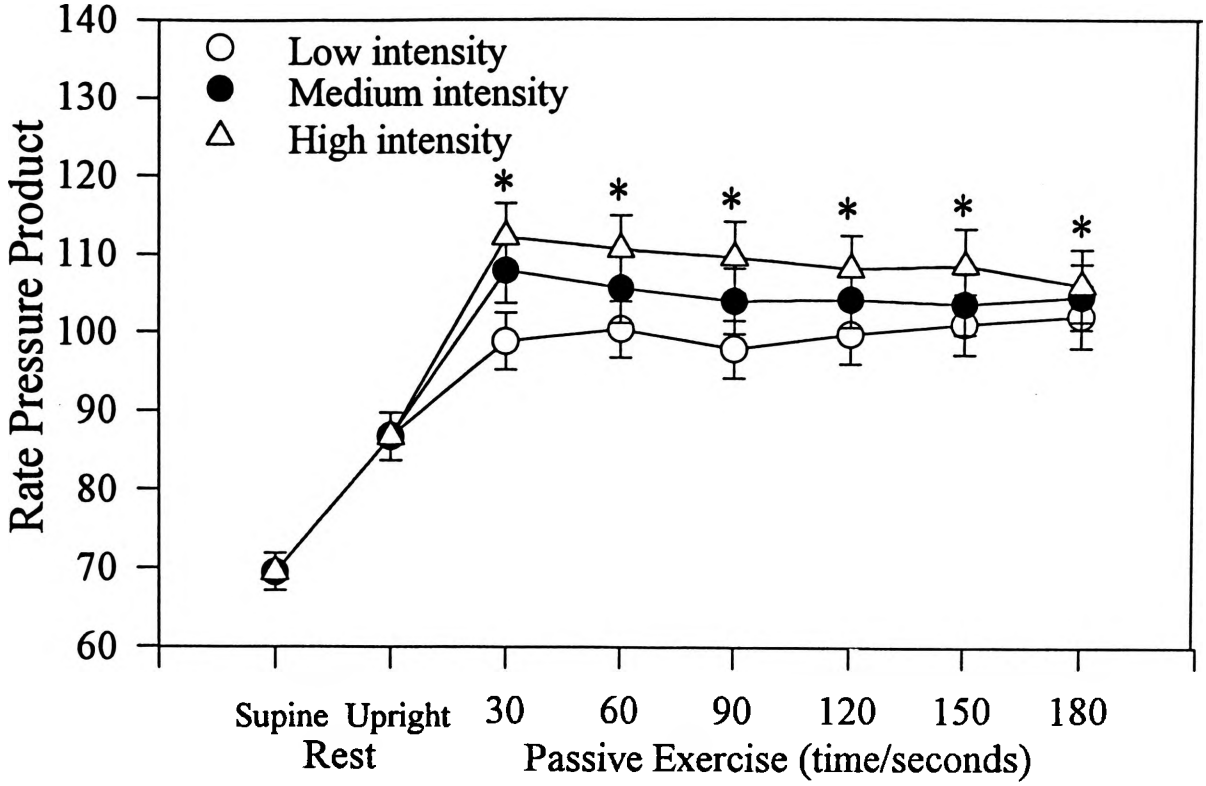

Figure 4.13 Rate pressure product response at rest and during passive cycle exercise at all intensities for the three groups combined. Data are means with standard errors of the means; (*) indicate that rate pressure product response during passive cycle exercise at the low, medium, and high intensities was significantly higher than rate pressure product response during upright rest.

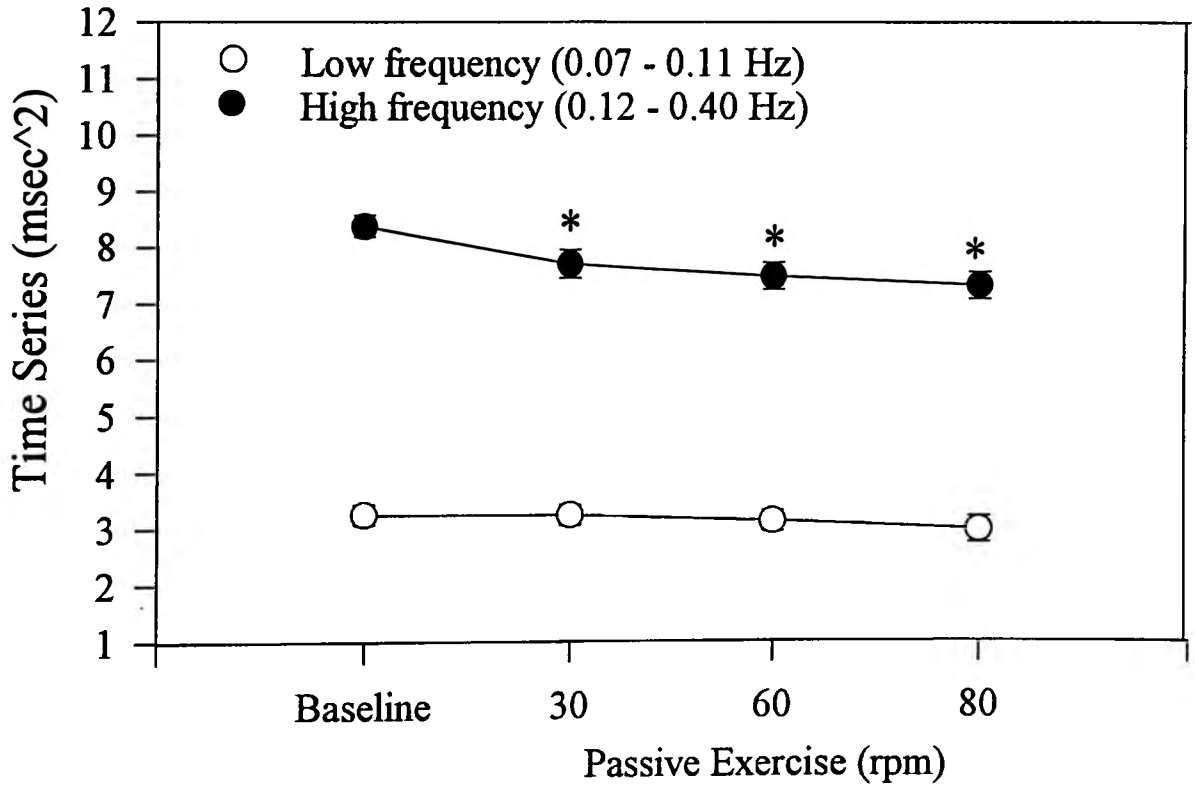

Figure 4.14 Time series (HRV_{ts}) analysis, that assesses vagal influence on the heart, at rest and during passive cycle exercise at all intensities for the three groups combined. Data are means with standard errors of the means; (*) indicate that vagal influence at the high frequnecy (0.12-0.40 Hz) was significantly lower at the low, medium, and high intensities of passive cycle exercise than vagal influence during baseline.

4.3.14 Heart rate variability (HRV_{ts})

Analysis on HRV_{ts} during all three passive cycle exercise intensities revealed a significant Time main effect at the high frequency (0.12 - 0.40 Hz), $F(3, 87) = 28.04$, $p=0.0001$. However, analysis on HRV_{ts} indicated no significance Time main effect existed at the low frequency (0.07 - 0.11 Hz), $F(3, 87) = 1.60$, $p>0.05$. As shown in Figure 4.14, HRV_{ts} for all subjects combined was significantly lower than upright rest during all three passive cycle exercise intensities (Table E-14, Appendix E).

4.3.15 Summary

During all intensities of passive cycle exercise all subjects combined showed a significant increase in heart rate, cardiac output, cardiac index, pre-ejection period, systolic and diastolic blood pressure, mean arterial pressure, and total peripheral resistance as well as rate pressure product. Stroke volume and stroke index were significantly greater only at the high intensity.

PEP/LVET ratio during passive cycle exercise was significantly lower than baseline at the high intensity, although no significant difference existed at the low and medium intensities. No significant difference existed for pre-ejection period at any intensity during passive cycle exercise for the three groups. During passive cycle exercise, left-ventricular ejection time was significantly lower than baseline at low and medium intensities. HRV_{ts} at the high frequency (0.12-0.40 Hz) was significantly lower than upright rest during all intensities of passive cycle exercise. However, HRV_{ts} at the low frequency (0.07-0.11 Hz) did not change throughout passive cycle exercise.

4.4 CARDIOVASCULAR RESPONSE OF CYCLISTS, RUNNERS, AND CONTROLS DURING PASSIVE CYCLE EXERCISE

4.4.1 Heart Rate

Analysis on heart rate response to passive cycle exercise revealed no significant Group main effect or Group by Time interaction at the low intensity, $\underline{F}(2, 27) = 2.24$, $p>0.05$; $\underline{F}(10, 135) = 0.92$, $p>0.05$ (Figure 4.15; Table D-1, Appendix D); the medium intensity, $\underline{F}(2, 27) = 0.61$, $p>0.05$; $\underline{F}(10, 135) = 1.48$, $p>0.05$ (Figure 4.15; Table D-1, Appendix D), or the high intensity, $\underline{F}(2, 27) = 0.31$, $p>0.05$; $\underline{F}(10, 135) = 0.87$, $p>0.05$ (Figure 4.15; Table D-1, Appendix D).

4.4.2 Stroke Volume

Analysis on stroke volume response to passive cycle exercise revealed no significant Group main effect or Group by Time interaction at the low intensity, $\underline{F}(2, 27) = 2.52$, $p>0.05$; $\underline{F}(10, 135) = 1.50$, $p>0.05$, (Figure 4.16; Table D-2, Appendix D); the medium intensity, $\underline{F}(2, 27) = 0.21$, $p>0.05$; $\underline{F}(10, 135) = 1.12$, $p>0.05$ (Figure 4.16; Table D-2, Appendix D), or the high intensity, $\underline{F}(2, 27) = 0.30$, $p>0.05$; $\underline{F}(10, 135) = 1.03$, $p>0.05$ (Figure 4.16; Table D-2, Appendix D).

4.4.3 Stroke Index

Analysis on stroke index response to passive cycle exercise revealed no significant Group main effect or Group by Time interaction at the low intensity, $\underline{F}(2, 27) = 2.25$, $p>0.05$; $\underline{F}(10, 135) = 1.40$, $p>0.05$, the medium intensity, $\underline{F}(10, 135) = 0.20$, $p>0.05$; $\underline{F}(10, 135) = 1.13$, $p>0.05$, or the high intensity, $\underline{F}(2, 27) = 0.34$, $p>0.05$; $\underline{F}(10, 135) = 1.12$, $p>0.05$ (Table D-3, Appendix D).

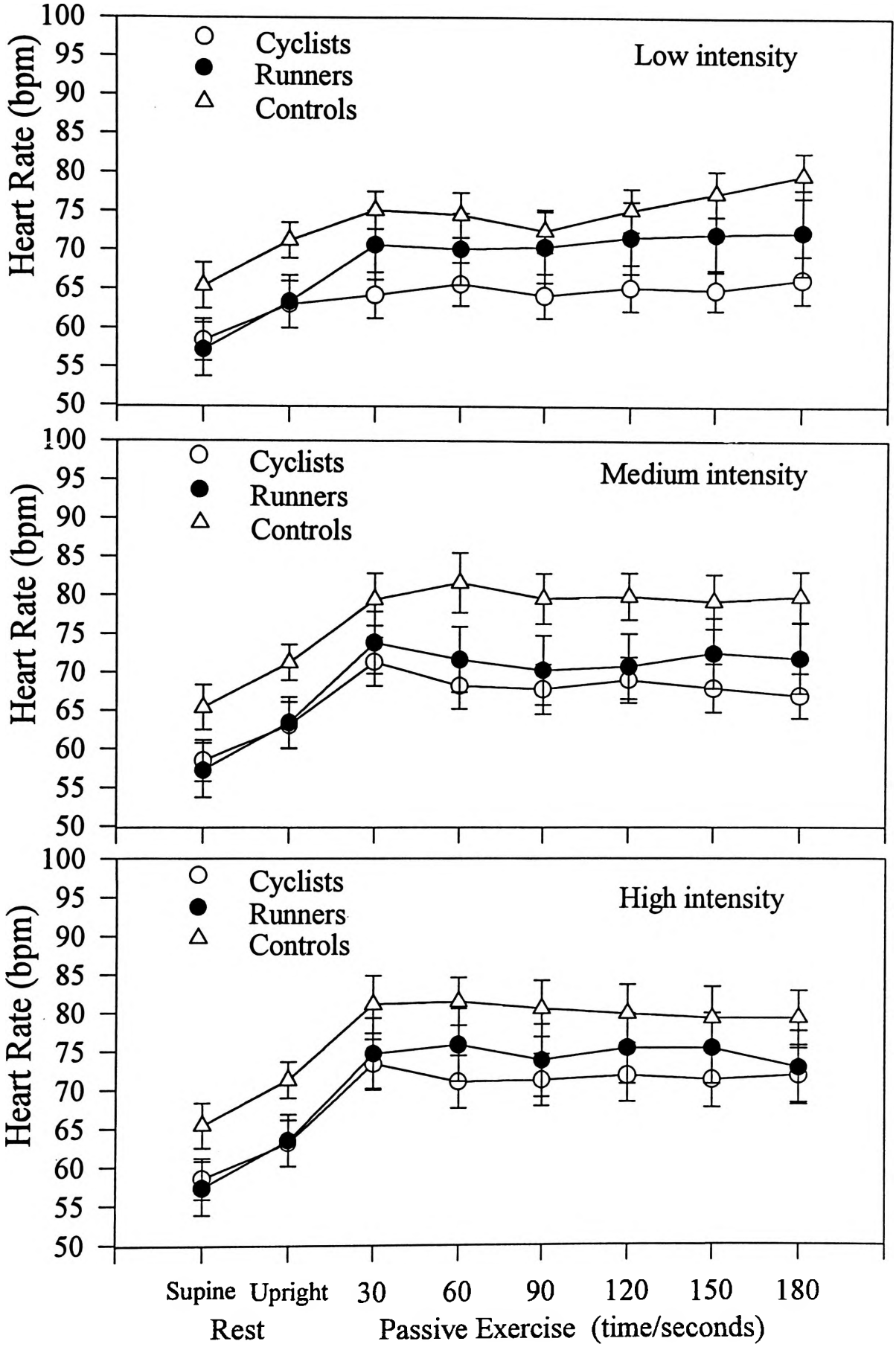

Figure 4.15 Heart rate response of Cyclists, Runners, and Controls at rest and during passive cycle exercise at the low, medium, and high intensities. Data are means with standard errors of the means.

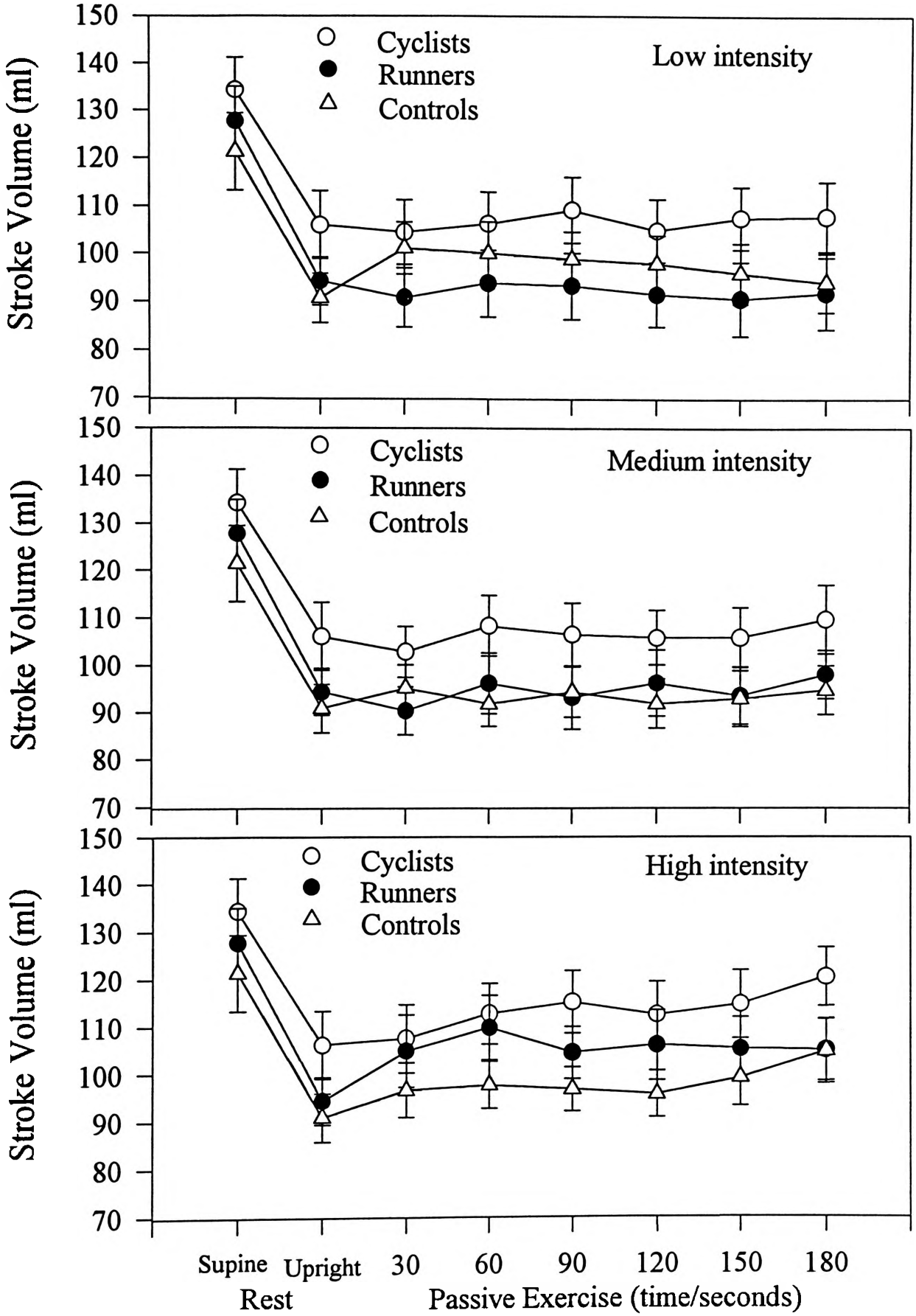

Figure 4.16 Stroke volume response of Cyclists, Runners, and Controls at rest and during passive cycle exercise at the low, medium, and high intensities. Data are means with standard errors of the means.

4.4.4 Cardiac Output

Analysis on cardiac output response to passive cycle exercise revealed no significant Group main effect or Group by Time interaction at the low intensity, $\underline{F}(2, 27) = 1.59, p>0.05$; $\underline{F}(10, 135) = 1.57, p>0.05$, the medium intensity, $\underline{F}(2, 27) = 0.41, p>0.05$; $\underline{F}(10, 135) = 0.72, p>0.05$, or the high intensity $\underline{F}(2, 27) = 0.68, p>0.05$; $\underline{F}(10, 135) = 1.09, p>0.05$ (Table D-4, Appendix D).

4.4.5 Cardiac Index

Analysis on cardiac index response to passive cycle exercise revealed no significant Group main effect or Group by Time interaction at the low intensity, $\underline{F}(2, 27) = 1.17, p>0.05$; $\underline{F}(10, 135) = 1.62, p>0.05$, the medium intensity, $\underline{F}(2, 27) = 0.22, p>0.05$; $\underline{F}(10, 135) = 0.71, p>0.05$, or the high intensity $\underline{F}(2, 27) = 0.81, p>0.05$; $\underline{F}(10, 135) = 1.16, p>0.05$ (Table D-5, Appendix D).

4.4.6 Pre-Ejection Period

Analysis on pre-ejection period response to passive cycle exercise revealed no significant Group main effect or Group by Time interaction at the low intensity, $\underline{F}(2, 27) = 1.06, p>0.05$; $\underline{F}(10, 135) = 0.71, p>0.05$, the medium intensity, $\underline{F}(2, 27) = 0.82, p>0.05$; $\underline{F}(10, 135) = 0.51, p>0.05$, and the high intensity $\underline{F}(2, 27) = 1.60, p>0.05$; $\underline{F}(10, 135) = 1.02, p>0.05$ (Figure 4.17 ; Table D-4, Appendix D).

4.4.7 Left-Ventricular Ejection Time

Analysis on left ventricular ejection time response to passive cycle exercise revealed no significant Group main effect or Group by Time interaction at the low intensity, $\underline{F}(2, 27) = 1.28, p>0.05$; $\underline{F}(10, 135) = 0.52, p>0.05$, the medium intensity, $\underline{F}(2, 27) = 0.21, p>0.05$; $\underline{F}(10, 135) = 0.91, p>0.05$, or the high intensity $\underline{F}(2, 27) = 0.29, p>0.05$; $\underline{F}(10, 135) = 1.04, p>0.05$ (Table D-7,

Appendix D).

4.4.8 Pre-Ejection Period/Left-Ventricular Ejection Time (PEP/LVET Ratio).

Analysis on the PEP/LVET ratio response to passive cycle exercise revealed no significant Group main effect or Group by Time interaction at the low intensity, $\underline{F}(2, 27) = 0.77, p>0.05$; $\underline{F}(10, 135) = 0.77, p>0.05$, the medium intensity, $\underline{F}(2, 27) = 0.23, p>0.05$; $\underline{F}(10, 135) = 0.59, p>0.05$, or the high intensity $\underline{F}(2, 27) = 0.49, p>0.05$; $\underline{F}(10, 135) = 1.56, p>0.05$ (Figure 4.18 ; Table D-8, Appendix D).

4.4.9 Systolic Blood Pressure

Analysis on systolic blood pressure response to passive cycle exercise revealed no significant Group main effect or Group by Time interaction at the low intensity, $\underline{F}(2, 27) = 0.43, p>0.05$; $\underline{F}(10, 135) = 0.55, p>0.05$, the medium intensity, $\underline{F}(2, 27) = 0.41, p>0.05$; $\underline{F}(10, 135) = 1.35, p>0.05$, or the high intensity $\underline{F}(2, 27) = 0.67, p>0.05$; $\underline{F}(10, 135) = 1.04, p>0.05$ (Table D-9, Appendix D).

4.4.10 Diastolic Blood Pressure

Analysis on diastolic blood pressure to passive cycle exercise revealed no significant Group main effect or Group by Time interaction at the low intensity, $\underline{F}(2, 27) = 0.61, p>0.05$; $\underline{F}(10, 135) = 1.18, p>0.05$, the medium intensity, $\underline{F}(2, 27) = 1.66, p>0.05$; $\underline{F}(10, 135) = 1.37, p>0.05$, or the high intensity $\underline{F}(2, 27) = 1.89, p>0.05$; $\underline{F}(10, 135) = 0.61, p>0.05$ (Figure 4.19 ; Table D-10, Appendix D).

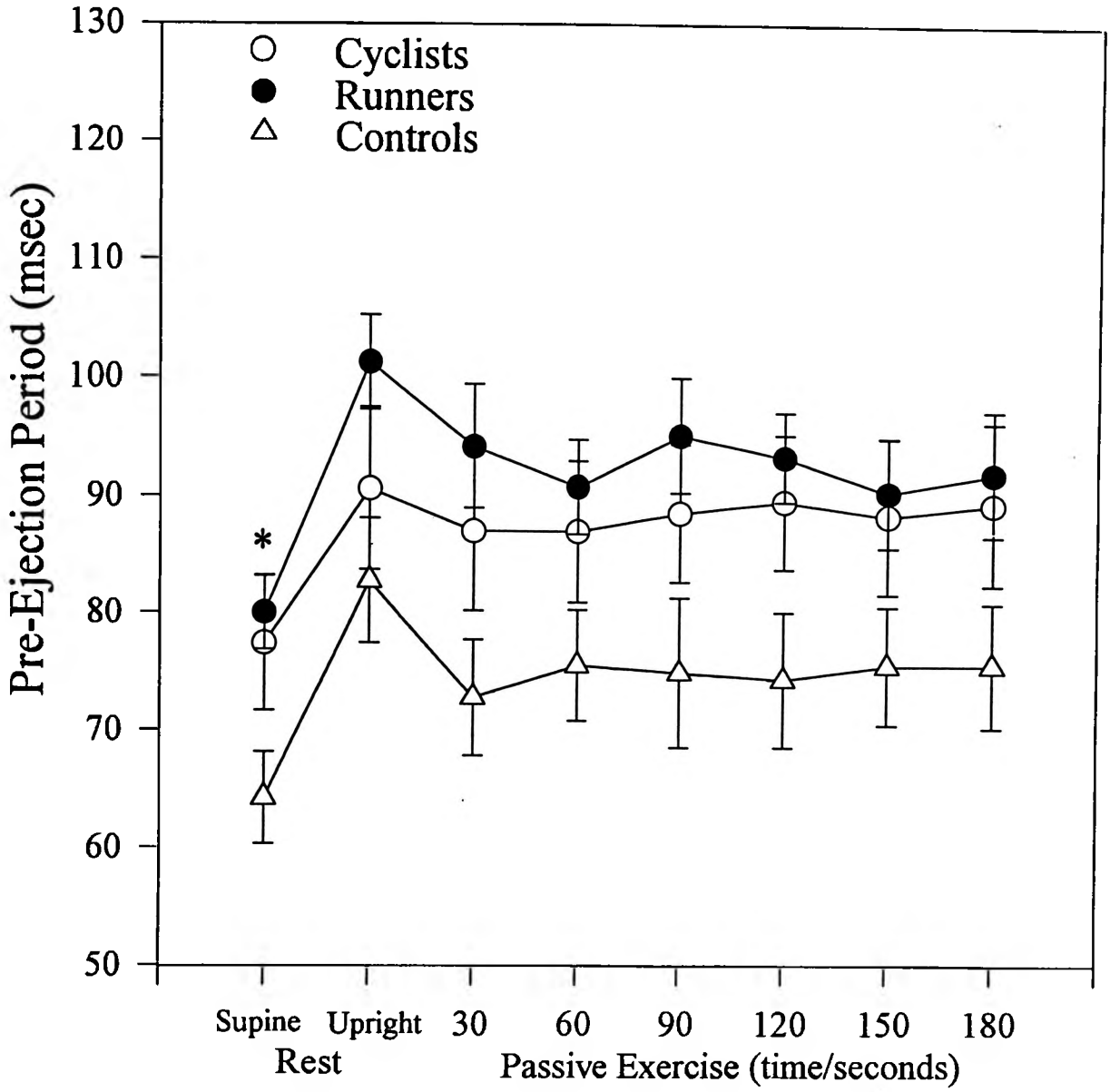

Figure 4.17 Pre-ejection period response of Cyclists, Runners, and Controls at rest and during passive cycle exercise at the medium intensity (60 rpm). Data are means with standard errors of the means; (*) indicates that pre-ejection period reponse of Cyclists and Runners was significantly greater than pre-ejection period response of Controls during supine rest.

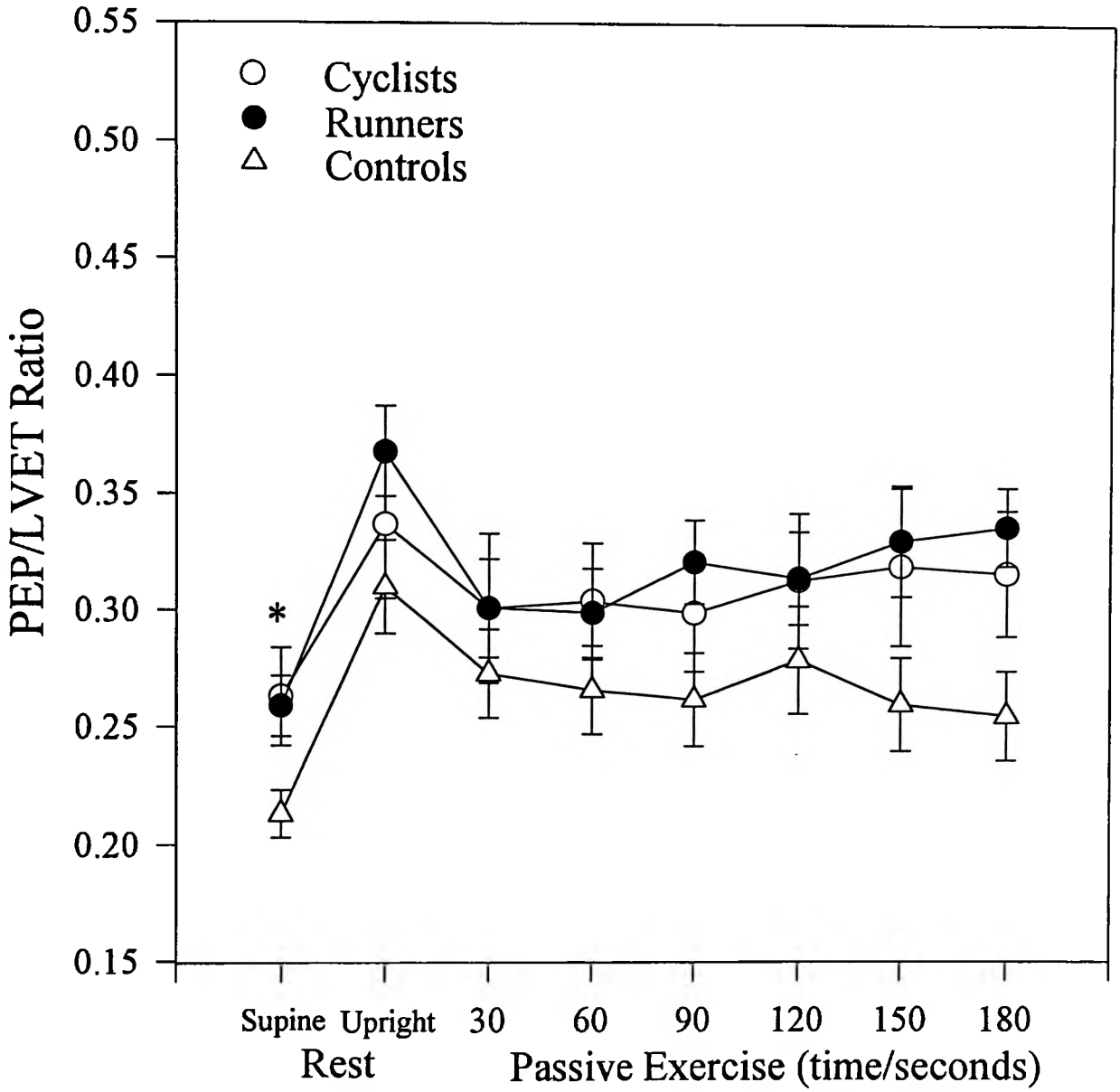

Figure 4.18 PEP/LVET ratio response of Cyclists, Runners, and Controls at rest and during passive cycle exercise at the medium intensity (60 rpm). Data are means with standard errors of the means; (*) indicates that PEP/LVET ratio response of Runners was significantly greater than PEP/LVET ratio response of Controls during supine rest.

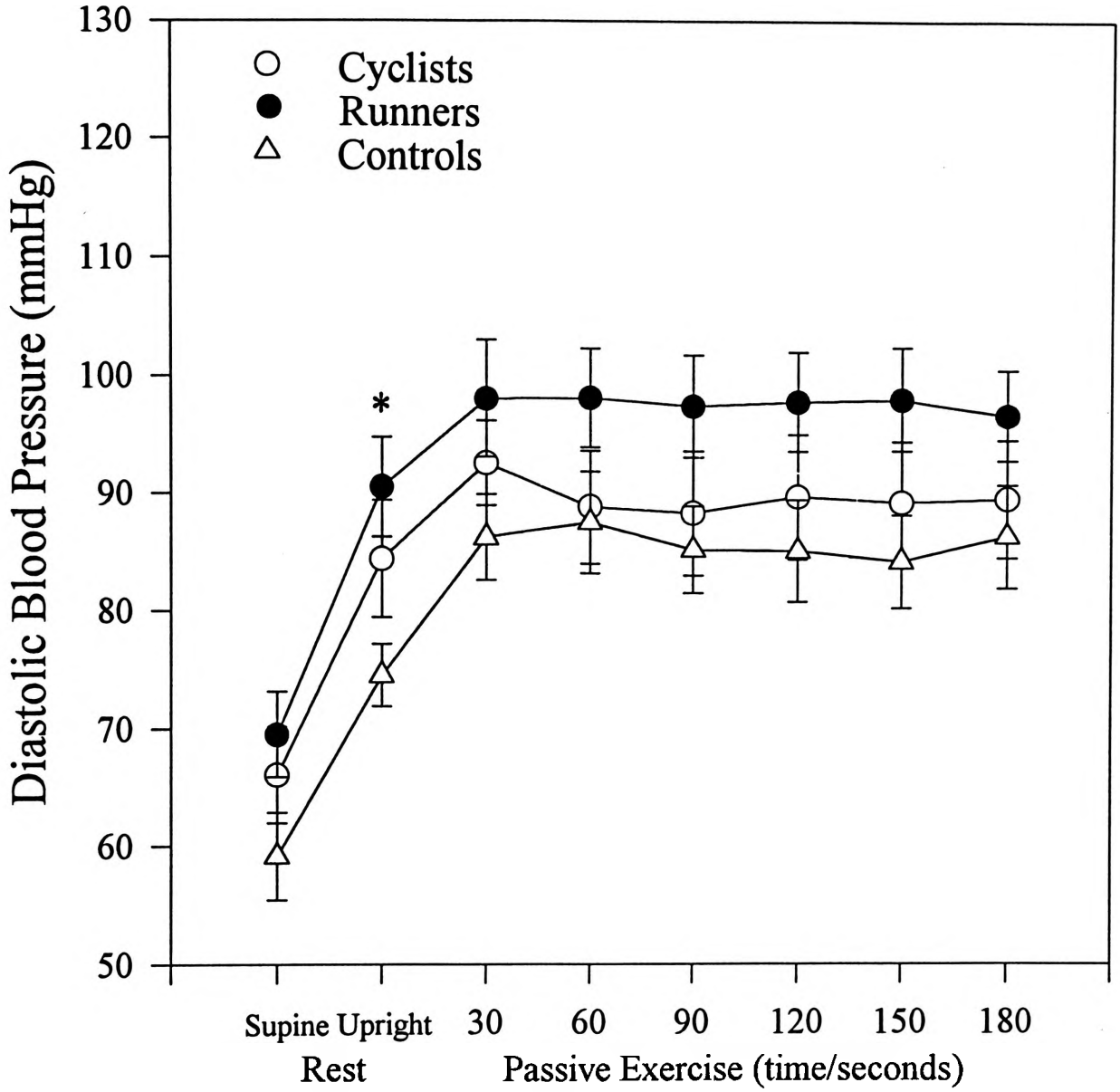

Figure 4.19 Diastolic blood pressure response of Cyclists, Runners, and Controls at rest and during passive cycle exercise at the low intensity (30 rpm). Data are means with standard errors of the means; (*) indicates that diastolic blood pressure response of Runners was significantly greater than diastolic blood pressure response of Controls during upright rest.

4.4.11 Mean Arterial Pressure

Analysis on mean arterial pressure response to passive cycle exercise revealed no significant Group main effect or Group by Time interaction at the low intensity, $F(2, 27) = 0.56, p>0.05$; $F(10, 135) = 0.53, p>0.05$, the medium intensity, $F(2, 27) = 1.21, p>0.05$; $F(10, 135) = 1.34, p>0.05$, or the high intensity $F(2, 27) = 1.43, p>0.05$; $F(10, 135) = 0.70, p>0.05$ (Figure 4.20 ; Table D-11, Appendix D).

4.4.12 Total Peripheral Resistance

Analysis on total peripheral resistance response to passive cycle exercise revealed no significant Group main effect or Group by Time interaction at the low intensity, $F(2, 27) = 0.09, p>0.05$; $F(10, 135) = 1.02, p>0.05$, the medium intensity, $F(2, 27) = 0.15, p>0.05$; $F(10, 135) = 0.80, p>0.05$, or the high intensity $F(2, 27) = 2.26, p>0.05$; $F(10, 135) = 0.93, p>0.05$ (Figure 4.21 ; Table D-12, Appendix D).

4.4.13 Rate Pressure Product

Analysis on rate pressure product response to passive cycle exercise showed no significant Group main effect or Group by Time interaction at the low intensity, $F(2, 27) = 2.52, p>0.05$; $F(10, 135) = 0.73, p>0.05$, the medium intensity, $F(2, 27) = 1.72, p>0.05$; $F(10, 135) = 1.34, p>0.05$, or the high intensity $F(2, 27) = 0.88, p>0.05$; $F(10, 135) = 1.47, p>0.05$ (Table D-13, Appendix D).

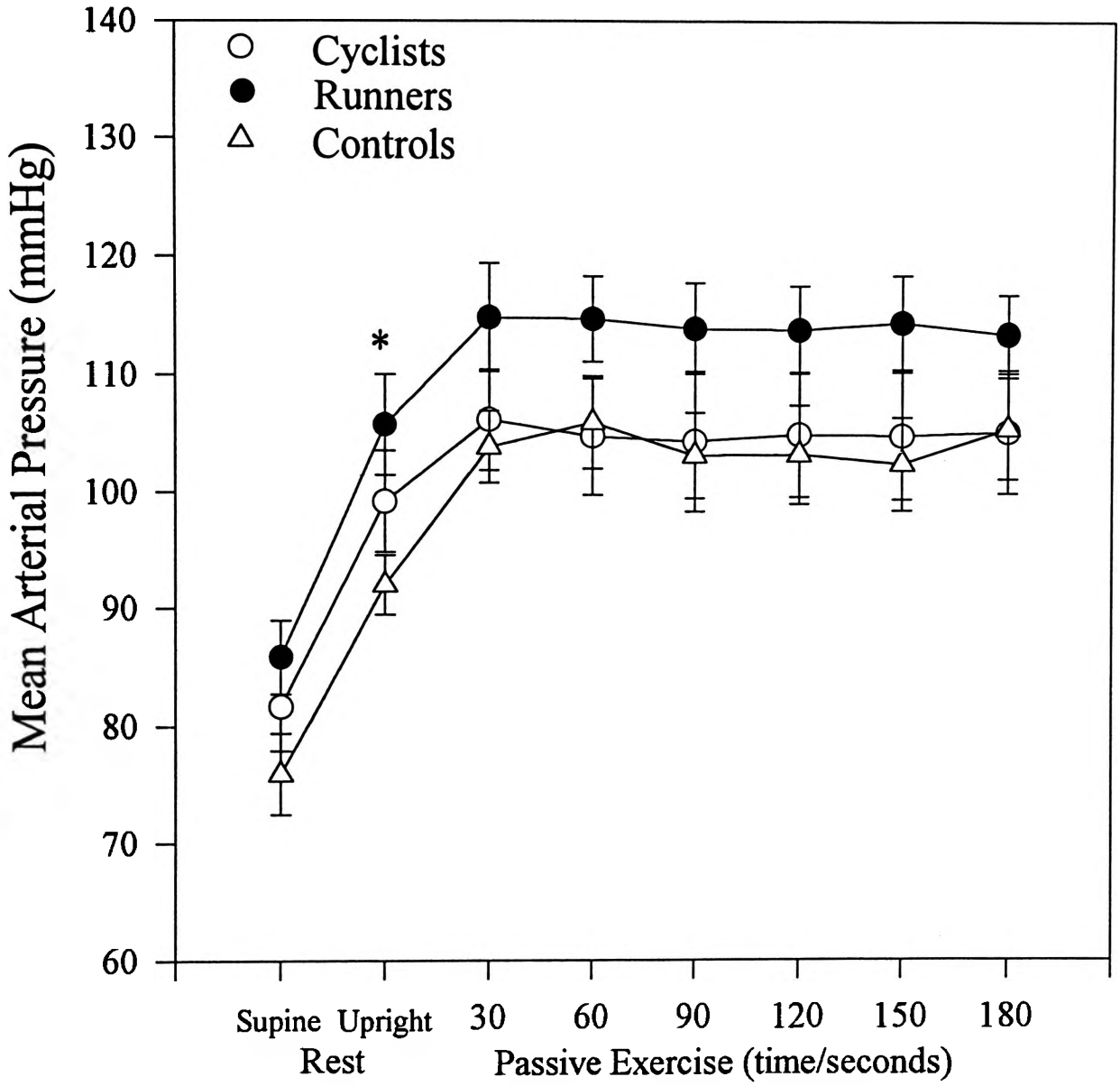

Figure 4.20 Mean arterial pressure response of Cyclists, Runners, and Controls at rest and during passive cycle exercise at the low intensity (30 rpm). Data are means with standard errors of the means; (*) indicates that mean arterial pressure response of Runners was significantly greater than mean arterial pressure response of Controls during upright rest.

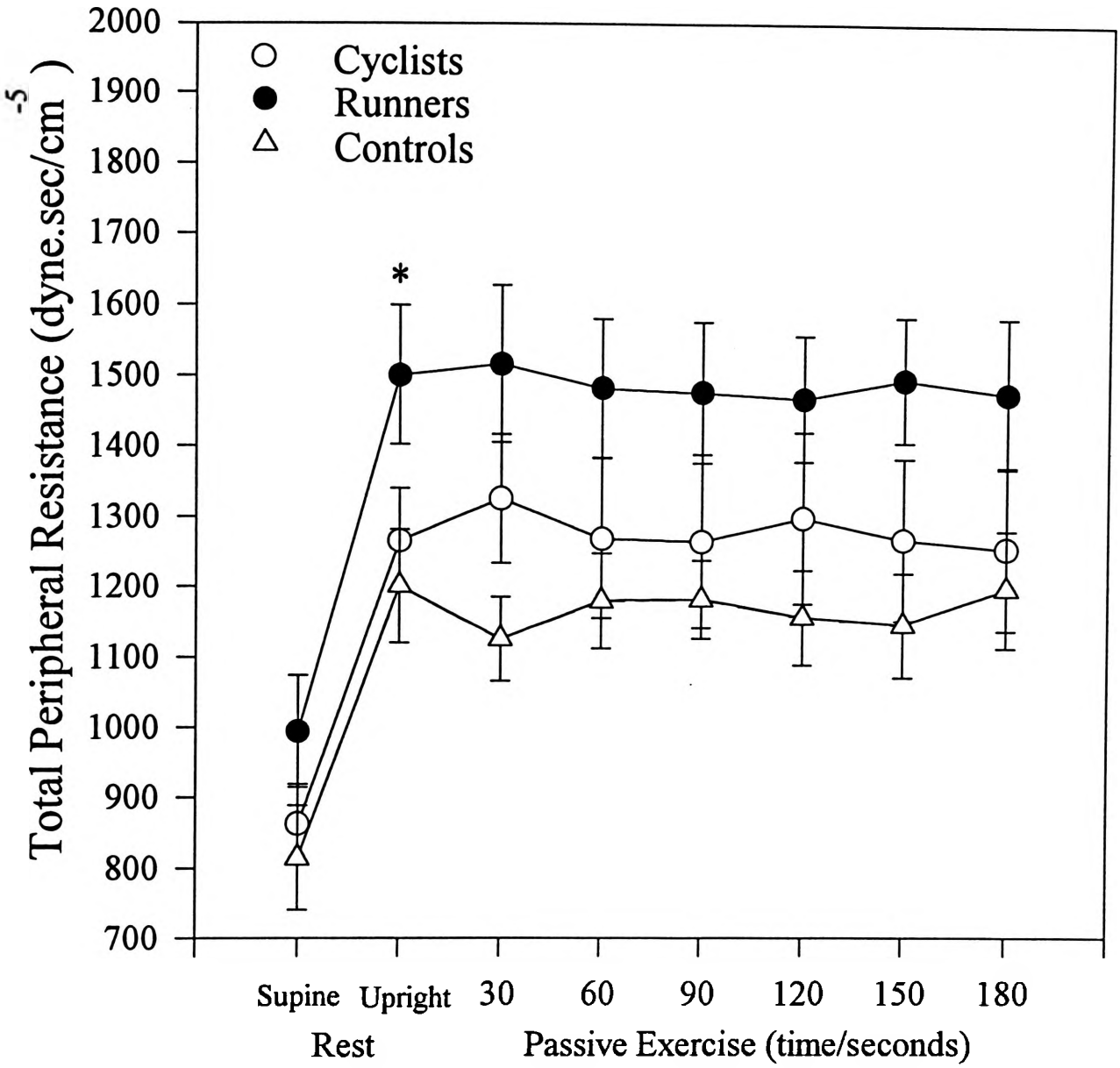

Figure 4.21. Total peripheral resistance response of Cyclists, Runners, and Controls at rest and during passive cycle exercise at the low intensity (30 rpm). Data are means with standard errors of the means; (*) indicates that total peripheral resistance response of Runners was significantly greater than total peripheral resistance response of Controls during upright rest.

4.4.14 Heart Rate Variability (HRV_{ts})

Analysis on HRV_{ts} response revealed no significant Group main effect or Group by Time interaction at the low, $\underline{F}(2, 27) = 0.13$, $p > 0.05$; $\underline{F}(4, 54) = 1.04$, $p > 0.05$ (Figure 4.22 ; Table D-14/Appendix D) and the high frequencies $\underline{F}(2, 27) = 1.79$, $p > 0.05$; $\underline{F}(4, 54) = 1.79$, $p > 0.05$ (Figure 4.22 ; Table D-15, Appendix D).

4.4.15 Summary

During passive cycle exercise at all intensities no significant Group main effects or Group by Time interaction existed for heart rate, stroke volume, stroke index, cardiac output, cardiac index, pre-ejection period, left ventricular ejection time, PEP/LVET ratio, systolic blood pressure, diastolic blood pressure, mean arterial pressure, total peripheral resistance, rate pressure product, or HRV_{ts}.

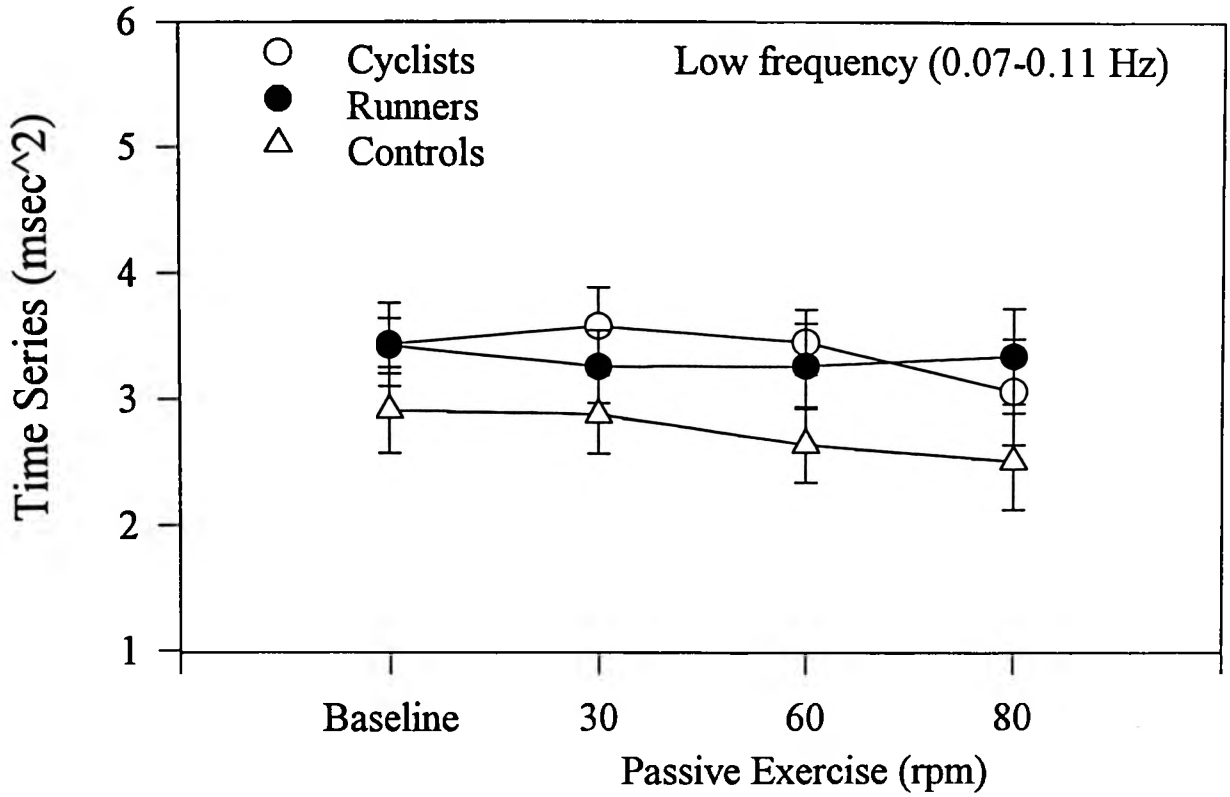

Figure 4.22 Time series (HRV_{ts}) analysis of Cyclists, Runners, and Controls at the low frequnecy at rest and during passive cycle exercise at the low (30 rpm), medium (60 rpm), and high (80 rpm) intensities. Data are means with standard errors of the means.

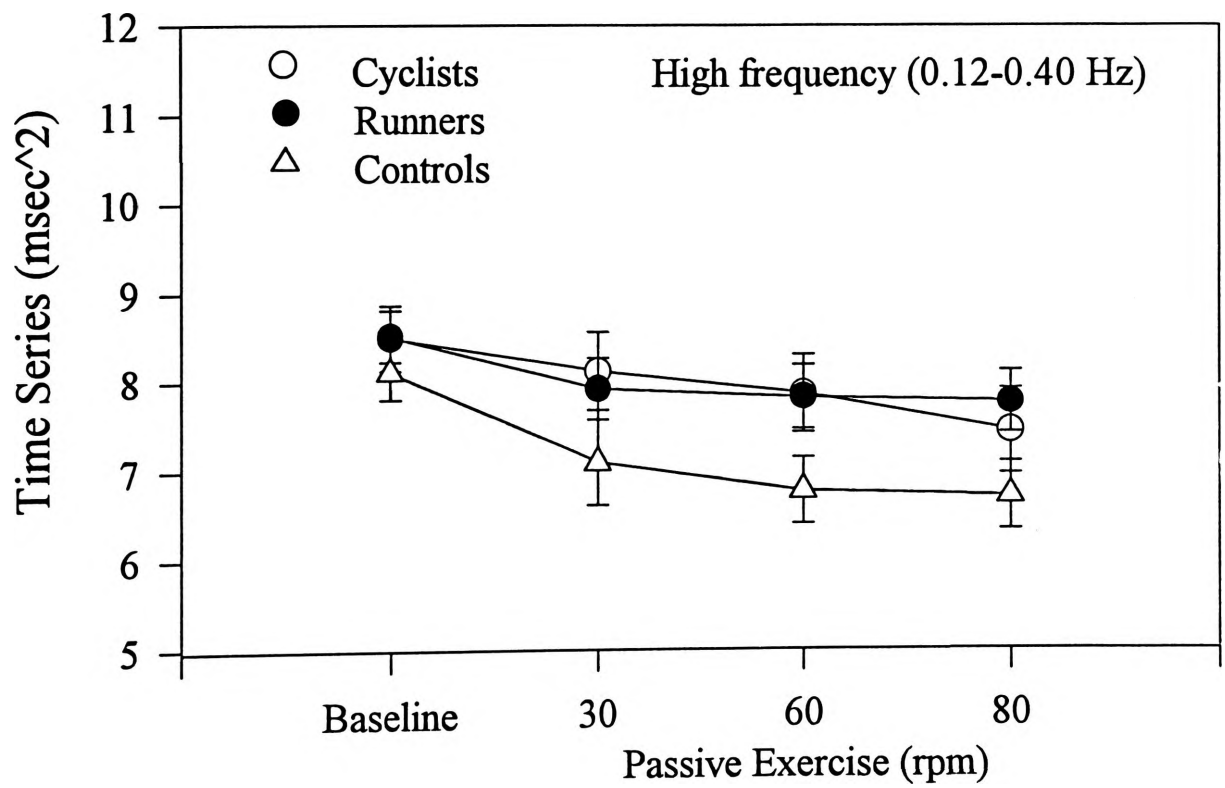

Figure 4.23 Time series (HRV_{ts}) analysis of Cyclists, Runners, and Controls at the high frequency at rest and during passive cycle exercise at the low (30 rpm), medium (60 rpm), and high (80 rpm) intensities. Data are means with standard errors of the means.

CHAPTER FIVE: DISCUSSION

The main purposes of this study were to ascertain the overall cardiovascular response of thirty male subjects to passive cycle exercise and to compare the cardiovascular response to passive cycle exercise of Cyclists, Runners, and Controls. The major findings of this study were firstly, passive cycle exercise resulted in a number of cardiovascular changes. The overall cardiovascular response to passive cycle exercise at all intensities for the three groups combined was increased heart rate, cardiac output, cardiac index, contractility of the heart, systolic and diastolic blood pressure, mean arterial pressure, and rate pressure product. Stroke volume and stroke index response during passive cycle exercise were only increased at the high intensity, whereas PEP/LVET ratio was significantly lower at the high intensity. Left ventricular ejection time was also significantly reduced at the low and medium intensities. Total peripheral resistance and vagal influence of the heart (HRV_{ts}) for all subjects combined were significantly decreased during passive cycle exercise. Secondly, the cardiovascular responses to passive cycle exercise of Cyclists, Runners, and Controls were not significantly different.

The first section will discuss cardiovascular response to passive exercise for all groups combined, whereas the cardiovascular response to passive exercise of Cyclists, Runners, and Controls will be discussed in the second section. Finally, conclusions and limitations will be discussed, and recommendations for future research outlined.

5.1 MAJOR FINDINGS

5.1.1 Cardiovascular Response To Passive Cycle Exercise

For the three groups combined, cardiovascular response was significantly influenced by passive cycle exercise. There was an increase of heart rate, cardiac output, cardiac index, stroke volume and stroke index only at the high intensity, contractility of the heart, systolic and diastolic blood pressure, mean arterial pressure, rate pressure product, and decreased total peripheral resistance and vagal influence on the heart. These findings supports the second hypothesis that passive cycle exercise for all thirty subjects would result in a significant cardiovascular response.

However, the elevation of heart rate at all intensities of passive cycle exercise does not support the third hypothesis that trained subjects carrying out passive cycle exercise would demonstrate a greater increase in stroke volume and greater reduction in heart rate. Furthermore, this finding also does not support previous research (Morikawa *et al.*, 1989) that has shown that decreased heart rate occurred during passive exercise in paraplegic patients and healthy people. Passive exercise in the Morikawa study involved using a 1-kg weight that was attached to each leg; the legs were then passively moved by pulling strings attached to each leg at a rate of 60 times/min for 1-1.5 min. Also stroke volume response to passive exercise was examined for 5 seconds. In the present study we assessed stroke volume response to passive exercise over a 25-second period. Thus, it is possible that the enhanced stroke volume response to both leg movement passive exercise and cycle passive exercise may only occur immediately upon initiation of the passive exercise stimulus.

In contrast, the increase of heart rate found in the present study does support other research (Nóbrega & Araújo, 1993) that has shown an increase of heart rate in healthy people during intense passive exercise. Passive exercise involved subjects sitting on a tandem bicycle that was propelled by an experimenter whilst the subjects sat passively. Passive exercise occurred for 4 seconds with the experimenter on the rear seat of the tandem sprinting as fast as possible. Thus, significant increases in heat rate during passive cycle may only occur at high intensity.

The mechanism underlying the increase of heart rate during passive cycle exercise is likely to be mechanoreceptors that are located in the muscle spindles, joints, and tendons (Coote, 1975; Nóbrega *et al.*, 1994). During passive exercise these receptors may provide feedback to the medulla resulting in an increase of heart rate. The actual increase in heart rate may be brought about by reduced vagal influence on the heart as HRV_{ts} was significantly decreased during passive cycle exercise. HRV_{ts} response was most greatly influenced at the high frequency (0.12-0.40 Hz) as HRV_{ts} did not change at the low frequency (0.07-0.11 Hz).

Another possibility underlying the enhancement of heart rate may be that central command was involved during passive cycle exercise. Benjamin and Peyser (1964) have suggested that passive exercise is never purely passive, and that there is always a certain degree of positive or negative active work involved. However, in the present study the EMG signals showed that little muscular contraction was present. Therefore, the absence of EMG activity indicates that central command did not contribute in a significant way to the heart rate increase during this form of exercise.

During passive cycle exercise stroke volume was significantly larger than

baseline only at the high intensity. Stroke volume showed only small non-significant increases at the low and medium intensities of passive cycle exercise. How was stroke volume increased during high intensity passive cycle exercise? There are a number of possible explanations underlying the enhancement of stroke volume such as an increase in ventricular preload and filling pressure; increased ventricular size or a decreased constraint of the pericardium; an increase in myocardial contractility; or a decrease in ventricular afterload (Feigl, 1974).

However, during passive cycle exercise venous return is likely to be the mechanism responsible for the increase of stroke volume during the high intensity. The faster movement of the legs at the high intensity may have resulted in enhanced venous return to the heart which may have led to an increase in end-diastolic volume or by a muscle mechanoreceptor-evoke increase in myocardial contractility (Nóbrega *et al.*, 1994). Factors that could have affected venous return may have been an increase in the pressure gradient between the veins and the heart (Ludbrook, 1962). For instance, sympathetically induced venous vasoconstriction, skeletal muscle activity, the effect of venous valves, and the cardiac-suction effect could all have enhanced venous return (Sherwood, 1993, p.332) during passive exercise. However, because of the lack of central command during passive exercise and the lack of respiratory change the major factor affecting the enhanced venous return appears to be the pumping action of the skeletal-muscles. Rowell (1986, p.144) thinks that the muscle pumps are so influential on cardiac performance that he has called them the "second heart". As mentioned in Chapter 2, the muscle pumps work by compressing the veins and squeezing the blood towards the heart, resulting in an increase in venous return, which leads to enhanced stroke volume. Surprisingly, the

stroke volume was not significantly increased at the low and medium intensities of passive cycle exercise. However, stroke volume was significantly increased at the high intensity. This finding indicates that passive cycle exercise at the low and medium intensities may not be powerful enough to stimulate the muscle pumps to increase venous return and enhance stroke volume.

Pre-ejection period (isovolumic contraction) was significantly lower at low, medium, and high intensities of passive cycle exercise. Left ventricular ejection time was significantly shorter only at the low and medium intensities, whereas PEP/LVET ratio was significantly lower only at the high intensity. Systolic time intervals are accepted markers of cardiac contractility. However, systolic time intervals only indicate increased beta-adrenergic activity to the myocardium under constant conditions of loading and heart rate. As heart rate and venous return were increased during passive cycle exercise the change in pre-ejection period and left ventricular ejection time are likely to reflect the Frank-Starling mechanism rather than increased beta-adrenergic activity (Newlin & Levenson, 1979).

Systolic, diastolic, and mean arterial blood pressure were significantly increased during passive cycle exercise at the low, medium, and high intensities. The increase of systolic blood pressure from upright to passive exercise was about 16 mmHg, whereas diastolic blood pressure was increased about 10 mmHg. The increase of blood pressure during passive cycle exercise was probably caused by the increase in cardiac output. The increase of cardiac output was likely to be a consequence of the enlarged stroke volume that was brought about by an increase in venous return and/or an increase in muscle mechanoreceptor-evoked myocardial contractility (Nóbrega *et al.*, 1994). Furthermore, increased peripheral resistance

can also cause an increase in blood pressure. However, total peripheral resistance was lower during passive cycle exercise. There are two mechanisms underlying the decrease of total peripheral resistance; decreased vaso-motor tone (decreased sympathetic activity) and increased intrinsic vasodilatation (Guyton, 1991, p.188). It was likely that the increase of blood pressure during passive cycle exercise brought about the decrease in total peripheral resistance.

Passive cycle exercise also resulted in a unique blood pressure response. For example, the increase of blood pressure during passive cycle exercise resembled the blood pressure response pattern found during isometric exercise. However, the decrease of total peripheral resistance during passive cycle exercise showed a similar response as that found with dynamic exercise. Thus, blood pressure and total peripheral resistance response to passive cycle exercise appears as a combination of static and dynamic response, although no voluntary movement from the legs occurred during passive cycle exercise. Furthermore, Stebbins *et al.* (1985) have shown that the activation of group III and IV afferents by passive movement resulted in an increase of intramuscular pressure and tension that brought about an increase in blood pressure. Rate pressure product, which is linear to myocardial oxygen consumption (McArdle *et al.*, 1991, p.307), was also significantly increased during passive cycle exercise at the low, medium, and high intensities. The increase in rate pressure product reflects the increased heart rate and blood pressure brought about by passive cycle exercise.

5.1.2 Group Comparison of Cardiovascular Response During Passive Cycle Exercise

The second purpose of this study was to examine the cardiovascular responses of trained and untrained subjects. Trained subjects were fit Cyclists and

Runners. Both Cyclists (76.25 $\pm$1.44 ml.kg^{-1}.min^{-1}) and Runners (65.50 $\pm$2.16 ml.kg^{-1}.min^{-1}) possessed significantly higher $\dot{V}o_{2peak}$ than Controls (46.31 $\pm$2.36 ml.kg^{-1}.min^{-1}). Also resting heart rate for Cyclists (58.43 $\pm$2.69 b.min^{-1}) and Runners (57.21 $\pm$3.47 b.min^{-1}) were lower than that of Controls (65.38 $\pm$2.93 b.min^{-1}). Despite these differences, results of the group comparison indicated that no differences in cardiovascular response to passive cycle exercise existed. Overall, these findings do not support the hypothesis that trained subjects will have greater stroke volume and greater reduction of heart rate compared to untrained subjects during passive cycle exercise. Interestingly, the most highly trained Cyclist in this study did show a deceleration of heart rate (Figure 5.1) and an increase in stroke volume during passive exercise at the low intensity (Figure 5.2). The increase of heart rate appears to have been caused by an increase of stroke volume. Thus, a decrease in heart rate and an increase in stroke volume during passive cycle exercise may only occur if the subject is extremely well trained.

During passive cycle exercise, no difference in stroke volume was found between groups, although Cyclists and Runners had greater absolute stroke volume relative to body mass at upright rest than Controls. The possible explanation for the lack of stroke volume differences during passive cycle exercise may be that passive cycle exercise did not activate the muscle pumps sufficiently to increase venous return and enhance stroke volume. A previous study (Boutcher *et al.*, 1994) has shown that during actual cycle exercise stroke volume was increased by about 20% in male Cyclists. The difference in stroke volume during actual cycle exercise of trained and untrained appears to be caused by the ability of the trained to increase stroke volume by augmentation of left ventricular end-diastolic volume (Schairer

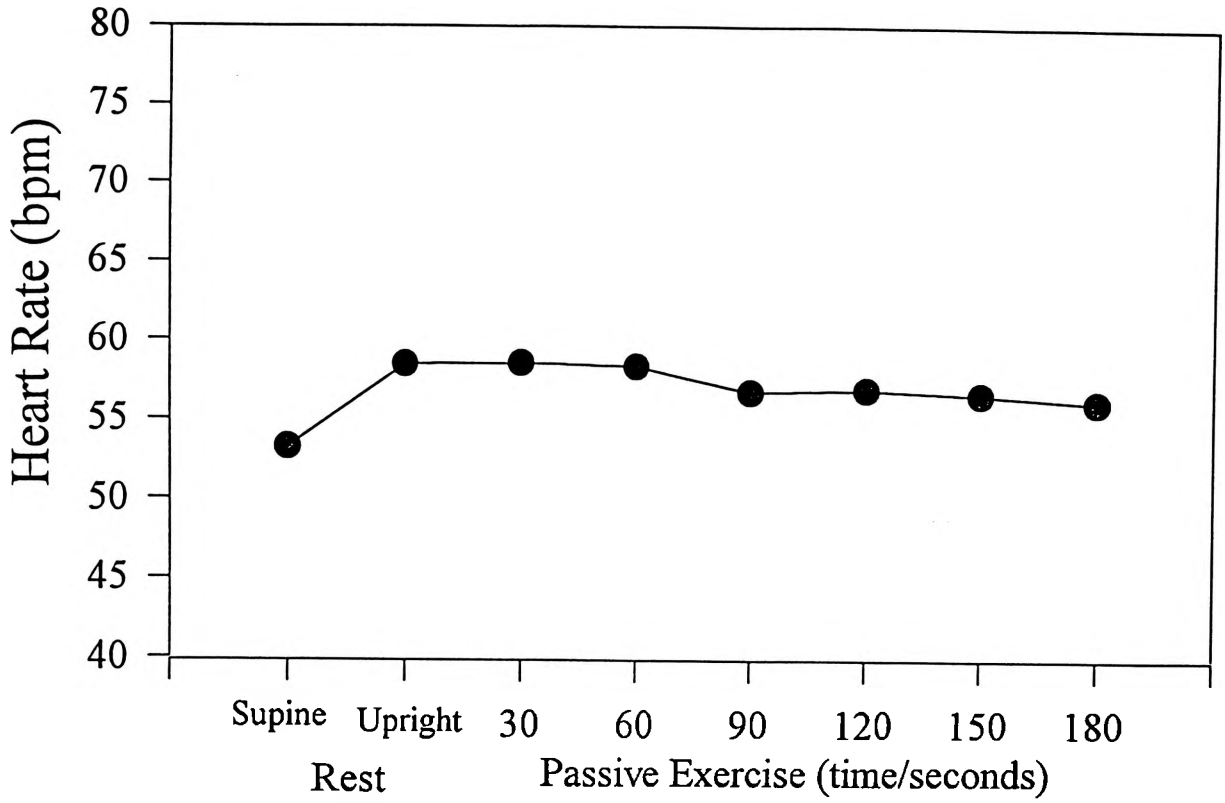

Figure 5.1 The heart rate response of one cyclist during passive cycle exercise at the low intensity (30 rpm).

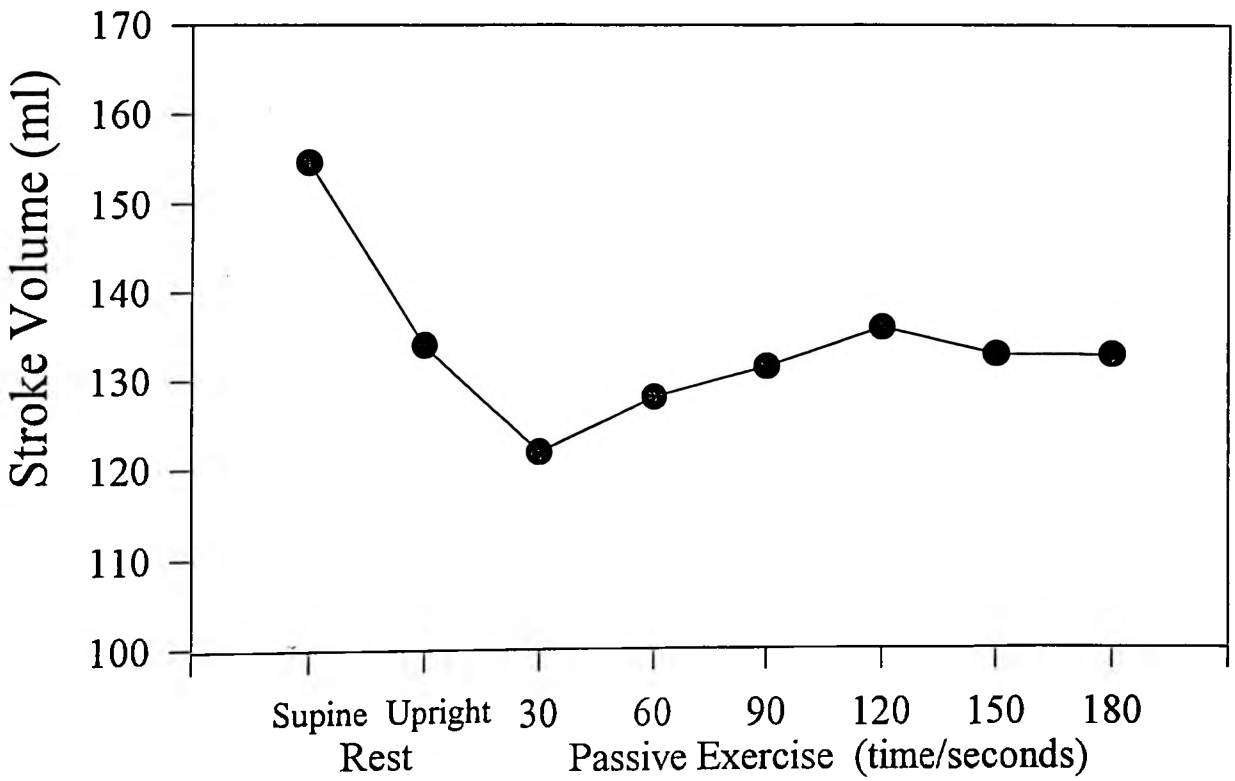

Figure 5.2 The stroke volume response of one cyclist during passive cycle exercise at the low intensity (30 rpm).

et al.,1992). Thus, passive stimulation of the leg muscle pumps may not provide a sufficient increase in left ventricular end-diastolic volume.

During passive cycle exercise, Cyclists and Runners had higher absolute values of heart rate variability (HRV_{ts}) at both low (0.07-0.11 Hz) and high (0.12-0.40 Hz) frequencies compared to Controls (Table D-15; Appendix D). In addition, both Cyclists and Runners had higher absolute values of HRV_{ts} at rest and at low and high frequencies, Cyclists (3.43 $\pm$0.33; 8.49 $\pm$0.37), Runners (3.42 $\pm$0.29; 8.51 $\pm$0.29), and Controls (2.91 $\pm$0.34; 8.10 $\pm$0.31). The mechanism underlying the greater, although not significant, HRV_{ts} of the trained may be the ability of aerobic exercise to enhance parasympathetic tone to the SA node. Also long term aerobic exercise appears to be effective in attenuating the loss of HRV_{ts} which accompanies a sedentary life-style (De Meersman, 1993).

Systolic and diastolic blood pressure, mean arterial pressure, and total peripheral response to passive cycle exercise showed no differences between groups. Resting systolic and diastolic blood pressure of Runners (119.43 $\pm$2.52 mmHg; 69.53 $\pm$3.61 mmHg), Cyclists (113.53 $\pm$3.11 mmHg; 66.12 $\pm$4.12 mmHg), and Controls (112.45 $\pm$2.36 mmHg; 59.15 $\pm$3.73 mmHg) were similar. During passive cycle exercise both Cyclists and Runners had greater systolic and diastolic blood pressure although it was not significantly greater than Controls. Because blood pressure is a function of cardiac output and total peripheral resistance the greater blood pressure of Cyclists and Runners may have resulted because of their greater cardiac output.

5.2 CONCLUSIONS

Overall, for all subjects combined there was a series of cardiovascular responses during passive cycle exercise. Initially, passive cycle exercise resulted in an

increase of heart rate probably caused by the stimulation of mechanoreceptors located in the exercising muscles, joints, and tendons. These receptors may have provided afferent feedback to cardiovascular centers that, in turn, sent impulses to the heart, resulting in an increase of heart rate. The increase in heart rate is likely to have been caused by decreased vagal influence on the heart. The increase of heart rate resulted in an increase in cardiac output which led to an increase in blood pressure. Lastly, the decrease of total peripheral resistance that occurred during passive cycle exercise was likely to have been a response to the increase in blood pressure.

The results of the group comparison showed that no difference in heart rate and stroke volume existed between Cyclists, Runners, and Controls. These findings showed that passive cycle exercise may not have been strong enough to activate the muscle pumps. The lack of stimulation of the muscle pumps would not have resulted in enhanced venous return and, therefore, an increase in stroke volume would not have occurred. The lack of increase in stroke volume of Cyclists, Runners, and Controls indicates that the muscle pump may not be effective during this form of passive cycle exercise.

5.3 LIMITATIONS

One of the assumptions of this study is that trained subjects had greater blood volume than the untrained. The expectation was that because trained subjects would be hypervolemic they would have greater stroke volume and greater reduction of heart rate during passive cycle exercise. Unfortunately, blood volume was not measured. However, the great majority of studies have shown that aerobically trained subjects typically possess high stroke volume and low heart rate. Other

studies have indicated that stroke volume, heart rate, and $\dot{V}o_{2max}$ are all highly correlated with blood volume. Therefore, although blood volume was not measured, the greater stroke volumes, lower heart rates, and higher $\dot{V}o_{2max}$ of the trained subjects may reflect hypervolemia (Convertino, 1991). Also, blood flow in the legs was not directly measured. Therefore, we did not know whether passive cycle exercise was powerful enough to activate the muscle pump to increase venous return and enhance stroke volume.

5.4 RECOMMENDATIONS FOR FUTURE RESEARCH

Future research should focus on the mechanisms underlying the larger stroke volume response of trained athletes by incorporating blood volume and blood flow measurements. The measurement of blood volume and blood flow may help clarify the factors underlying the stroke volume difference between trained and untrained subjects.

Because of the difficulty in avoiding participation of central command in assessing cardiac response to passive exercise, different method to assess cardiac response to passive exercise is needed. For instance, electrical stimulation of the muscle pumps may provide an alternative method.

As mechanical factors may be responsible for the greater venous return and the increase in stroke volume during actual cycle exercise in trained aerobic athletes, future investigations should focus on the role of venous return and the muscle pumps on exercise cardiac performance.

CHAPTER SIX: REFERENCE LIST

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APPENDICES

Appendix A Human Experimentation Ethics Approval

UNIVERSITY OF WOLLONGONG
Office of the Vice-Principal (Administration)

Academic & Student Services Branch

In reply please quote: DC:KM HE95/01
Further Information: Karen McRae (Ext 4457)

22 March 1995

Ms Yati Nurhayati
C/- Dr S. Boutcher
Biomedical Sciences Department
University of Wollongong

Dear Yati Nurhayati

Thank you for your response to the Committee's requirements for your Human Research Ethics application HE95/01.

Your response meets with the requirements of the Committee and your application is now formally approved.

Karen McRae

for

Chairperson
Human Research Ethics Committee

cc. Dr S. Boutcher, Biomedical Sciences

Address: Northfields Avenue Wollongong 2522 Australia
hone (042) 21 3555, Facsimile – Student Services: (042) 21 4322, Research/Secretariat: (042) 21 433

UNIVERSITY OF WOLLONGONG
Office of the Vice-Principal (Administration)

Academic & Student Services Branch

INITIAL CONDITIONAL
In reply please quote: DC:KM HE95/01
Further Information: Karen McRae (Ext 4457)

27 February 1995

Ms Yati Nurhayati
C/- Dr S. Boutcher
Biomedical Sciences Department
University of Wollongong

Dear Ms Nurhayati,

I am pleased to advise that the following Human Research Ethics application has been conditionally approved:

Ethics Number:	HE95/01
Project Title:	Cardiovascular Regulation During Passive Exercise in Trained and Untrained Males
Name of Researchers:	Yati Nurhayati
Approval Date:	21 February 1995
Duration of Clearance:	20 February 1996

This approval is granted subject to satisfactory clarification of the following matters:

(i) that the Consent Form should state the Supervisor's name and contact number.

(ii) that the way in which participants are screened for heart abnormalities is more clearly defined.

Address: Northfields Avenue Wollongong 2522 Australia
ıone (042) 21 3555, Facsimile – Student Services: (042) 21 4322, Research/Secretariat: (042) 21 4338

Please provide written evidence that these conditions have been satisfied to the Secretary of the Committee before the commencement of your research, or approval will be withdrawn.

This certificate relates to the research protocol submitted in your application of 16 January 1995. It will be necessary to inform the Committee of any changes to the research protocol and seek clearance in such an event.

Please note that experiments of long duration must be reviewed annually by the Committee and it will be necessary for you to apply for renewal of this application if experimentation is to continue beyond one year.

Chairperson
Human Research Ethics Committee

cc: Head, Biomedical Sciences
Dr S. Boutcher, Supervisor

Appendix B Informed Consent

INFORMED CONSENT

The researchers conducting this project support the principles governing both the ethical conduct of research, and the protection at all times of the interests, comfort and safety of subjects.

This form and the accompanying Subject Information Package are given to you for your own protection. They contain a detailed outline of the experimental procedures, and possible risks. Your signature below indicates six things:

(1) you have received the Subject Information Package;
(2) you have read its contents;
(3) you have been given the opportunity to discuss the contents with one of the researchers prior to commencing the experiment;
(4) you clearly understand these procedures and possible risks;
(5) you voluntarily agree to participate in the project; and
(6) your participation may be terminated at any point in time without jeopardizing your involvement with the University of Wollongong, or your assessment for this or any other course undertaken through the University.

Any concerns, complaints or further questions may be directed initially to Dr. Stephen Boutcher (Department of Biomedical Science: phone 214-093), or subsequently to the Secretary of the University of Wollongong Human Research Ethics Committee (phone 214-457).

Last name: _____ Given name: _____
Date of Birth: __/__/__
Address: _____
_____

Name and phone number of contact person in case of an emergency:
Name: _____ Phone: _____
Family doctor: _____ Phone: _____
Signature: _____ Date: __/__/__
Witness: Name _____ Signature: _____

Appendix C Personal Health and Exercise History Questionnaire

PERSONAL HEALTH HISTORY

Please complete this form as accurately as possible.
Name:_____ Date:_____ Age:_____
Height:_____ Weight:_____ Sex;_____
Date of birth _____ Ethnicity: _____

1. GENERAL MEDICAL HISTORY

circle one

Any medical complaints? (Please specify) YES NO

_____

_____

Are you on any medication? YES NO
Specify_____
Adrenal disease? YES NO
Fainting spells? YES NO
Hypoglycemia? YES NO
Seizures? YES NO
Diabetes? YES NO
Kidney problems? YES NO
Stomach ulcers? YES NO

2. EXERCISE HISTORY

Do you jog, cycle or swim? YES NO
If yes then specify:
a. how many times a week?_____
b. how long is each session?_____
c. what kind of pace?_____
d. how long have you been regularly exercising?
_____
e. how many years overall have you been exercising?_____
Do you do any other form of exercise?
Specify_____
a. how many times a week?_____
b. how long each session?_____
c. how many years have you participated in this activity?_____

3. CARDIORESPIRATORY HISTORY

Any heart disease now? YES NO
Any heart disease in past? YES NO
Heart murmurs? YES NO
Occasional chest pains? YES NO
Fainting? YES NO
Asthma or allergies? YES NO
Family history of heart disease? YES NO
High blood pressure? YES NO
Shortness of breath after walking flights of stairs? YES NO

4. MUSCULAR HISTORY

Any muscle injuries now?	YES	NO
Muscle injuries in past?	YES	NO
Muscle pains during exercise?	YES	NO
Family history of muscle pains?	YES	NO

5. BONE-JOINT HISTORY

Any bone or joint injuries now?	YES	NO
Any in past?	YES	NO
Ever had swollen joints?	YES	NO

6. PREPAREDNESS FOR TESTING

			SPECIFY
a. Any food during the last 8 hours?	YES	NO	
b. Any liquids during the last 8 hours? (except water).	YES	NO	
c. Any caffeine during the last 8 hours?	YES	NO	
d. Any medication during the last 8 hours? (including insulin, except for basal infusion in pump patients).	YES	NO	
e. Any over the counter drugs during the last 8 hours? (aspirin, antihistamines, nasal sprays, etc).	YES	NO	
f. Any alcohol in last 24 hours?	YES	NO	
g. Any tobacco during the last 8 hours?	YES	NO	
h. Any vigorous exercise in the last 24 hours? (any exercise not part of patient's daily routine ie., routine jogging ok, but marathon running is not. No exercise morning of test).	YES	NO	
i. Any emotional upset in last 24 hours? Depression, crying episodes, anxiety from personal trauma (death, divorce, car accident dentist, etc).	YES	NO	
j. Acute illness in last 48 hours? (cold, flu, measles, etc).	YES	NO	
k. Any hypoglycemic episodes during the last 8 hours?	YES	NO	
l. a) Fasting blood sugar value (mmol)_____ (finger-stick method is ok)			
b) Signs or symptoms of hypoglycemia?	YES	NO	

Appendix D Resting and Passive cycle exercise means and standard errors for all cardiovascular variables of Trained Cyclists, Runners, and Controls.

Table D-1: Heart rate ($b.min^{-1}$) during supine, upright rest, and passive cycle exercise at all intensities (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	58.43 (2.9)	57.21 (3.47)	65.38 (2.93)
Upright rest	1	63.03 (3.00)	63.37 (3.36)	71.23 (2.33)
30 rpm	1	64.21 (2.93)	70.68 (4.49)	75.19 (2.43)
	2	65.64 (2.79)	70.16 (4.64)	74.58 (2.91)
	3	64.18 (2.83)	70.47 (4.61)	72.55 (2.81)
	4	65.24 (2.95)	71.73 (4.65)	75.28 (2.79)
	5	64.93 (2.60)	72.10 (4.83)	77.42 (2.94)
	6	66.27 (3.08)	72.37 (5.57)	77.42 (2.90)
60 rpm	1	71.32 (3.16)	73.82 (4.04)	79.40 (3.35)
	2	68.18 (3.03)	71.63 (4.28)	81.57 (3.87)
	3	67.78 (3.27)	70.25 (4.53)	79.55 (3.25)
	4	68.98 (3.01)	70.77 (4.28)	79.83 (3.00)
	5	67.92 (3.17)	72.48 (4.59)	79.14 (3.50)
	6	66.91 (2.96)	71.81 (4.54)	79.77 (3.25)
80 rpm	1	73.27 (3.17)	74.58 (4.68)	81.01 (3.78)
	2	70.92 (3.40)	75.70 (4.72)	81.34 (3.13)
	3	71.12 (3.37)	73.66 (4.69)	80.40 (3.69)
	4	71.70 (3.42)	75.22 (4.57)	79.71 (3.83)
	5	71.16 (3.60)	75.22 (4.59)	79.13 (4.16)
	6	71.74 (3.51)	72.73 (4.75)	79.20 (3.54)

Table D-2: Stroke volume (ml) during supine, upright rest, and passive cycle exercise at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	134.29 (6.96)	127.75 (7.26)	121.34 (8.07)
Upright rest	1	106.09 (7.16)	94.32 (5.01)	90.77 (5.16)
30 rpm	1	104.62 (6.81)	90.89 (6.13)	101.25 (5.52
	2	106.48 (6.66)	93.95 (6.96)	100.21 (6.67)
	3	109.48 (6.95)	93.45 (6.93)	99.03 (5.81)
	4	105.13 (6.66)	90.69 (6.70)	98.07 (6.07)
	5	107.73 (6.62)	90.75 (7.66)	96.08 (6.36)
	6	108.04 (7.31)	91.91 (7.45)	94.11 (6.14)
60 rpm	1	102.85 (5.37)	90.28 (5.15)	95.12 (4.93)
	2	108.33 (6.35)	96.12 (6.51)	91.77 (4.87)
	3	106.52 (6.63)	93.12 (6.82)	94.28 (5.45)
	4	105.83 (5.75)	96.18 (7.13)	91.79 (5.30)
	5	105.86 (6.34)	93.53 (6.15)	92.82 (5.96)
	6	109.63 (7.20)	98.02 (5.21)	94.66 (5.20)
80 rpm	1	107.38 (7.20)	104.78 (7.65)	96.49 (5.75)
	2	112.61 (6.47)	109.65 (6.81)	97.52 (4.94)
	3	115.01 (6.65)	104.28 (5.49)	96.62 (4.64)
	4	112.31 (7.07)	105.89 (7.39)	95.56 (4.90)
	5	114.56 (7.27)	105.11 (6.63)	98.99 (6.03)
	6	120.38 (6.21)	104.87 (6.51)	104.65 (6.78)

Table D-3: Stroke index (ml/m^2) during supine, upright rest, and passive cycle exercise at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	75.17 (4.29)	67.41 (3.47)	61.63 (3.84)
Upright rest	1	59.61 (4.69)	49.80 (2.39)	46.09 (2.34)
30 rpm	1	58.93 (4.79)	48.02 (3.31)	51.48 (2.62)
	2	59.95 (4.69)	49.72 (3.71)	50.85 (3.01)
	3	61.58 (4.75)	49.43 (3.68)	50.32 (2.70)
	4	59.16 (4.65)	48.50 (3.56)	49.81 (2.77)
	5	60.58 (4.56)	47.99 (4.07)	48.83 (3.02)
	6	60.68 (4.77)	48.58 (3.89)	47.82 (2.95)
60 rpm	1	57.63 (3.56)	47.78 (2.73)	48.41 (2.37)
	2	60.83 (4.31)	50.87 (3.47)	46.61 (2.14)
	3	59.82 (4.42)	49.27 (3.65)	47.94 (2.59)
	4	59.45 (4.05)	50.85 (3.74)	46.63 (2.46)
	5	59.43 (4.21)	49.48 (3.25)	47.26 (3.00)
	6	61.50 (4.58)	51.83 (2.69)	48.13 (2.47)
80 rpm	1	59.88 (3.92)	55.37 (3.93)	49.12 (2.88)
	2	62.94 (3.78)	58.11 (3.79)	49.56 (2.20)
	3	64.25 (3.85)	55.10 (2.74)	49.11 (2.06)
	4	62.76 (4.13)	55.89 (3.68)	48.72 (2.54)
	5	64.15 (4.42)	55.61 (3.54)	50.34 (2.91)
	6	67.38 (3.88)	55.44 (3.33)	53.11 (3.07)

Table D-4: Cardiac output (litres) during supine, upright rest, and passive cycle exercise at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	7.80 (.45)	7.24 (.52)	7.79 (.38)
Upright rest	1	6.58 (.36)	5.90 (.34)	6.38 (.28)
30 rpm	1	6.59 (.32)	6.26 (.38)	7.53 (.33)
	2	6.93 (.44)	6.36 (.35)	7.35 (.39)
	3	6.96 (.46)	6.40 (.44)	7.09 (.37)
	4	6.82 (.47)	6.37 (.38)	7.29 (.39)
	5	6.94 (.43)	6.28 (.36)	7.33 (.38)
	6	7.07 (.47)	6.37 (.38)	7.17 (.36)
60 rpm	1	7.33 (.51)	6.55 (.34)	7.50 (.42)
	2	7.33 (.48)	6.72 (.38)	7.39 (.38)
	3	7.14 (.45)	6.34 (.33)	7.40 (.37)
	4	7.23 (.40)	6.62 (.39)	7.23 (.36)
	5	7.11 (.39)	6.66 (.48)	7.24 (.38)
	6	7.23 (.41)	6.88 (.33)	7.45 (.32)
80 rpm	1	7.79 (.54)	7.70 (.62)	7.72 (.42)
	2	7.92 (.53)	8.16 (.55)	7.83 (.31)
	3	8.11 (.49)	7.61 (.48)	7.67 (.35)
	4	7.92 (.41)	7.86 (.61)	7.58 (.47)
	5	8.05 (.53)	7.71 (.37)	7.66 (.34)
	6	8.59 (.54)	7.50 (.52)	8.17 (.45)

Table D-5: Cardiac index (litres/m^2) during supine, upright rest, and passive cycle exercise at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	4.35 (.24)	3.82 (.26)	3.97 (.19)
Upright rest	1	3.67 (.22)	3.11 (.17)	3.25 (.13)
30 rpm	1	3.69 (.22)	3.32 (.21)	3.83 (.15)
	2	3.89 (.31)	3.37 (.18)	3.73 (.17)
	3	3.91 (.31)	3.39 (.23)	3.61 (.16)
	4	3.83 (.31)	3.37 (.21)	3.71 (.18)
	5	3.89 (.29)	3.32 (.19)	3.73 (.18)
	6	3.96 (.30)	3.37 (.21)	3.65 (.17)
60 rpm	1	4.11 (.32)	3.47 (.19)	3.82 (.21)
	2	4.11 (.32)	3.56 (.21)	3.76 (.18)
	3	4.00 (.31)	3.36 (.19)	3.77 (.18)
	4	4.05 (.27)	3.50 (.21)	3.68 (.16)
	5	3.98 (.26)	3.53 (.26)	3.69 (.19)
	6	4.05 (.26)	3.64 (.18)	3.79 (.15)
80 rpm	1	4.34 (.29)	4.08 (.33)	3.94 (.22)
	2	4.43 (.30)	4.33 (.31)	3.99 (.14)
	3	4.52 (.28)	4.00 (.25)	3.90 (.15)
	4	4.43 (.23)	4.16 (.33)	3.88 (.26)
	5	4.51 (.31)	4.09 (.21)	3.90 (.16)
	6	4.80 (.32)	3.97 (.29)	4.15 (.19)

Table D-6. Pre-ejection period (msec) during supine, upright rest, and passive cycle exercise at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	77.40 (5.75)	80.00 (3.13)	64.23 (3.91)
Upright rest	1	90.58 (6.88)	101.25 (4.01)	82.75 (5.32)
30 rpm	1	91.80 (6.01)	91.20 (4.67)	73.40 (5.55)
	2	88.20 (6.13)	94.60 (5.83)	74.60 (4.93)
	3	89.20 (6.97)	93.60 (4.37)	71.80 (5.12)
	4	90.40 (6.76)	93.00 (5.32)	73.20 (5.69)
	5	89.40 (7.17)	95.00 (4.83)	75.40 (5.35)
	6	88.20 (7.10)	93.00 (5.73)	75.60 (5.47)
60 rpm	1	87.00 (6.78)	94.20 (5.21)	72.80 (4.95)
	2	87.00 (6.03)	90.80 (4.03)	75.60 (4.76)
	3	88.60 (5.85)	95.20 (4.86)	75.00 (6.39)
	4	89.60 (5.72)	93.40 (3.81)	74.40 (5.78)
	5	88.40 (6.70)	90.40 (4.66)	75.60 (5.06)
	6	89.40 (6.91)	92.00 (5.31)	75.60 (5.34)
80 rpm	1	82.00 (7.05)	79.80 (4.25)	70.20 (5.01)
	2	82.20 (5.80)	80.60 (3.38)	69.80 (4.58)
	3	82.00 (5.72)	85.40 (3.63)	69.40 (5.17)
	4	84.80 (6.98)	83.40 (4.15)	70.20 (4.47)
	5	85.40 (7.18)	87.60 (4.89)	68.80 (4.99)
	6	85.20 (6.49)	89.80 (4.39)	69.80 (5.35)

Table D-7. Left-ventricular ejection time (msec) during supine, upright rest, and passive cycle exercise at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	297.27 (4.34)	309.33 (5.95)	302.27 (6.06)
Upright rest	1	274.20 (7.01)	274.53 (7.66)	268.80 (5.35)
30 rpm	1	264.40 (6.96)	257.80 (8.63)	262.60 (7.07)
	2	266.80 (6.06)	261.40 (9.52)	264.40 (7.00)
	3	266.20 (5.91)	259.80 (8.69)	265.20 (6.90)
	4	265.60 (5.66)	258.80 (9.18)	262.80 (7.12)
	5	265.80 (5.35)	257.60 (9.72)	262.40 (7.45)
	6	267.00 (7.06)	258.80 (9.37)	259.60 (7.08)
60 rpm	1	264.80 (6.23)	260.60 (7.77)	261.20 (6.86)
	2	269.20 (5.88)	265.80 (8.09)	257.20 (9.67)
	3	269.80 (6.97)	267.60 (9.38)	261.80 (8.98)
	4	268.80 (6.22)	267.20 (8.18)	260.60 (7.80)
	5	269.80 (6.25)	266.00 (9.23)	263.20 (6.71)
	6	272.60 (6.76)	266.40 (8.54)	262.80 (7.36)
80 rpm	1	276.40 (9.88)	270.40 (8.73)	257.80 (7.82)
	2	273.40 (5.82)	273.00 (8.78)	265.40 (8.34)
	3	275.60 (5.71)	269.00 (8.47)	265.80 (7.20)
	4	271.60 (5.96)	268.00 (8.29)	256.00 (9.97)
	5	273.60 (8.16)	267.40 (7.19)	267.00 (8.26)
	6	272.00 (5.83)	269.80 (7.41)	274.60 (12.0)

Table D-8. Pre-ejection period/Left-ventricular ejection time ratio during supine, upright rest, and passive cycle exercise at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	.263 (.021)	.259 (.013)	.213 (.010)
Upright rest	1	.337 (.032)	.368 (.019)	.310 (.020)
30 rpm	1	.352.(029)	.359 (.025)	.282 (.023)
	2	.335.(029)	.371 (.032)	.286 (.022)
	3	.339.(031)	.366 (.025)	.272 (.021)
	4	.341 (.028)	.366 (.028)	.281 (.024)
	5	.340 (.031)	.376 (.028)	.289 (.023)
	6	.336 (.033)	.367 (.031)	.296 (.024)
60 rpm	1	.332 (.029)	.368 (.029)	.280 (.021)
	2	.326 (.027)	.346 (.023)	.298 (.020)
	3	.332 (.028)	.362 (.027)	.290 (.028)
	4	.337 (.027)	.355 (.022)	.288 (.025)
	5	.332 (.031)	.346 (.024)	.289 (.020)
	6	.333 (.031)	.351 (.028)	.288 (.021)
80 rpm	1	.301 (.032)	.301 (.021)	.273 (.019)
	2	.304 (.025)	.299 (.019)	.266 (.019)
	3	.299 (.025)	.321 (.018)	.262 (.020)
	4	.313 (.029)	.314 (.020)	.279 (.023)
	5	.319 (.034)	.330 (.024)	.260 (.020)
	6	.316 (.027)	.336 (.017)	.255 (.019)

Table D-9: Systolic blood pressure (mmHg) during supine, upright rest, and passive cycle exercise at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	113.53 (3.11)	119.43 (2.52)	112.45 (2.36)
Upright rest	1	129.79 (3.53)	137.10 (4.66)	127.89 (3.38)
30 rpm	1	134.30 (6.46)	149.40 (4.58)	140.20 (3.48)
	2	137.40 (6.08)	148.90 (3.64)	144.10 (4.05)
	3	137.50 (6.11)	148.30 (3.94)	140.10 (4.84)
	4	136.20 (5.82)	146.90 (4.31)	140.50 (4.89)
	5	137.50 (6.47)	148.20 (4.06)	139.70 (5.21)
	6	137.70 (6.41)	147.80 (3.69)	141.70 (5.08)
60 rpm	1	138.70 (6.58)	149.60 (3.80)	143.70 (6.08)
	2	137.90 (6.98)	150.10 (3.39)	141.90 (6.11)
	3	138.70 (7.14)	151.60 (3.08)	141.20 (5.78)
	4	139.70 (6.47)	147.10 (3.72)	142.50 (3.72)
	5	137.70 (6.89)	147.20 (4.46)	142.10 (4.35)
	6	136.60 (7.36)	147.90 (3.54)	147.10 (4.22)
80 rpm	1	140.90 (6.76)	151.80 (4.71)	149.40 (5.08)
	2	137.90 (5.79)	148.70 (4.07)	150.60 (6.12)
	3	139.00 (7.65)	149.90 (4.23)	149.20 (5.27)
	4	141.60 (7.61)	146.10 (5.52)	143.60 (5.51)
	5	137.80 (7.65)	149.60 (7.25)	147.10 (5.45)
	6	139.00 (7.33)	145.10 (7.31)	143.60 (6.07)

Table D-10. Diastolic blood pressure (mmHg) during supine, upright rest, and passive cycle exercise at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	66.12 (4.12)	69.53 (3.61)	59.15 (3.73)
Upright rest	1	84.46 (4.95)	90.55 (4.26)	74.55 (2.63)
30 rpm	1	92.60 (3.62)	98.10 (4.98)	86.30 (3.63)
	2	88.90 (4.80)	98.20 (4.19)	87.60 (4.35)
	3	88.40 (5.28)	97.50 (4.37)	85.30 (3.69)
	4	89.80 (5.33)	97.90 (4.21)	85.20 (4.34)
	5	89.30 (5.24)	98.10 (4.35)	84.30 (3.96)
	6	89.60 (5.04)	96.70 (3.83)	86.40 (4.42)
60 rpm	1	90.60 (5.19)	98.30 (4.66)	90.00 (4.98)
	2	89.50 (5.54)	98.80 (4.89)	90.40 (4.79)
	3	90.40 (5.64)	100.20 (4.38)	89.60 (4.61)
	4	91.20 (5.52)	96.70 (3.94)	90.20 (4.26)
	5	90.20 (5.67)	97.20 (3.88)	90.40 (4.25)
	6	89.70 (5.63)	98.80 (4.88)	92.70 (3.96)
80 rpm	1	93.40 (5.33)	96.80 (4.16)	92.40 (4.28)
	2	91.70 (4.88)	96.80 (4.32)	94.90 (4.54)
	3	91.00 (5.04)	95.60 (4.05)	94.10 (4.76)
	4	93.20 (5.28)	95.20 (5.58)	91.60 (4.25)
	5	90.40 (5.46)	95.30 (5.41)	91.00 (4.49)
	6	90.20 (5.44)	95.30 (5.89)	91.30 (4.45)

Table D-11. Mean arterial pressure (mmHg) during supine, upright rest, and passive cycle exercise at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	81.67 (3.74)	85.88 (3.14)	75.92 (3.48)
Upright rest	1	99.23 (4.33)	105.78 (4.31)	92.08 (2.57)
30 rpm	1	106.20 (4.28)	114.90 (4.57)	103.90 (3.07)
	2	104.80 (4.96)	114.80 (3.59)	106.00 (3.95)
	3	104.40 (5.97)	114.00 (3.86)	103.20 (3.64)
	4	105.00 (5.35)	113.90 (3.68)	103.30 (4.23)
	5	104.90 (5.44)	114.50 (3.94)	102.50 (4.00)
	6	105.20 (5.28)	113.50 (3.25)	105.50 (4.35)
60 rpm	1	106.30 (5.35)	115.10 (4.04)	107.50 (4.88)
	2	105.30 (5.79)	115.50 (3.99)	107.20 (4.79)
	3	106.20 (5.92)	116.90 (3.44)	106.50 (4.62)
	4	107.10 (5.68)	113.30 (3.19)	107.30 (3.89)
	5	105.70 (5.92)	113.70 (3.36)	107.60 (3.78)
	6	104.90 (6.06)	114.90 (3.92)	110.50 (3.53)
80 rpm	1	108.90 (5.55)	114.70 (3.82)	111.00 (3.83)
	2	106.80 (4.80)	113.90 (3.83)	113.10 (4.32)
	3	106.70 (5.58)	113.40 (3.89)	112.10 (4.40)
	4	108.80 (5.71)	111.90 (5.18)	108.60 (4.19)
	5	106.00 (5.91)	113.20 (5.78)	109.30 (4.41)
	6	106.10 (5.79)	111.50 (6.16)	108.30 (4.44)

Table D-12. Total peripheral resistance (dyne.sec/cm^{-5}) during supine, upright rest, and passive cycle exercise at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	862.31 (57.34)	994.48 (80.00)	814.68 (74.33)
Upright rest	1	1267.03 (73.92)	1501.85 (98.33)	1202.45 (80.96)
30 rpm	1	1327.30 (91.51)	1518.40 (110.68)	1127.40 (59.84)
	2	1271.40 (113.86)	1484.90 (98.17)	1182.30 (67.87)
	3	1267.90 (123.82)	1479.60 (99.91)	1185.10 (55.75)
	4	1301.40 (122.62)	1471.50 (89.41)	1159.20 (67.51)
	5	1270.10 (116.19)	1498.10 (88.95)	1148.50 (74.54)
	6	1256.80 (117.50)	1478.20 (106.79)	1199.30 (83.76)
60 rpm	1	1230.30 (119.84)	1447.00 (94.66)	1187.40 (90.31)
	2	1220.50 (125.36)	1424.90 (106.46)	1189.30 (78.66)
	3	1252.90 (118.89)	1521.60 (98.88)	1185.40 (84.37)
	4	1234.70 (108.08)	1422.20 (104.84)	1216.80 (75.87)
	5	1241.90 (111.83)	1447.10 (134.05)	1219.80 (76.11)
	6	1215.20 (113.88)	1363.80 (74.34)	1210.80 (65.30)
80 rpm	1	1183.60 (115.47)	1265.70 (110.58)	1197.20 (91.99)
	2	1126.20 (92.52)	1160.40 (84.65)	1174.10 (63.69)
	3	1091.70 (88.11)	1250.40 (96.36)	1188.70 (59.32)
	4	1135.60 (92.47)	1216.10 (118.87)	1199.60 (102.1)
	5	1097.00 (97.19)	1213.80 (99.39)	1158.40 (56.14)
	6	1032.50 (96.53)	1257.50 (118.16)	1085.40 (57.27)

Table D-13. Rate pressure product (HR x SBP/100) during supine, upright rest, and passive cycle exercise at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	66.43 (3.79)	68.65 (5.05)	73.40 (3.28)
Upright rest	1	82.00 (5.02)	87.07 (5.97)	91.44 (4.71)
30 rpm	1	86.04 (5.33)	105.20 (6.91)	105.66 (4.88)
	2	89.65 (4.61)	104.06 (6.74)	107.95 (6.07)
	3	87.84 (5.14)	104.06 (6.76)	102.29 (6.60)
	4	89.18 (6.36)	104.90 (6.84)	106.06 (5.97)
	5	88.90 (4.99)	106.67 (7.48)	108.56 (6.54)
	6	90.66 (5.24)	106.41 (7.92)	110.15 (6.51)
60 rpm	1	98.63 (6.26)	110.33 (6.52)	115.06 (8.27)
	2	93.31 (5.37)	107.23 (6.25)	116.89 (9.30)
	3	93.40 (5.99)	105.92 (6.25)	113.38 (8.31)
	4	95.80 (5.16)	103.36 (5.64)	114.30 (6.66)
	5	93.11 (6.04)	105.72 (6.03)	113.09 (7.10)
	6	91.25 (6.33)	105.64 (6.38)	118.03 (7.25)
80 rpm	1	102.75 (6.18)	112.53 (7.00)	121.69 (8.29)
	2	97.46 (5.75)	111.70 (6.24)	123.24 (8.11)
	3	98.40 (6.68)	110.02 (7.26)	121.05 (8.58)
	4	101.06 (6.89)	109.06 (6.80)	115.16 (8.18)
	5	97.95 (7.57)	111.47 (7.48)	117.35 (8.79)
	6	99.65 (7.40)	104.76 (8.11)	114.89 (8.71)

Table D-14. Time series (msec^2) at high frequency (0.12-0.40 Hz) during baseline and passive cycle exercise at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Cyclists	Runners	Controls
Baseline	8.49 (.37)	8.51 (.29)	8.10 (.31)
30 rpm	8.11 (.44)	7.91 (.35)	7.08 (.48)
60 rpm	7.85 (.44)	7.81 (.36)	6.76 (.37)
80 rpm	7.43 (.48)	7.76 (.35)	6.70 (.38)

Table D-15. Time series (msec^2) at low frequency (0.07-0.11 Hz) during baseline and passive cycle exercise at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Cyclists	Runners	Controls
Baseline	3.43 (.33)	3.42 (.22)	2.91 (.34)
30 rpm	3.58 (.31)	3.26 (.29)	2.88 (.31)
60 rpm	3.46 (.26)	3.27 (.34)	2.65 (.30)
80 rpm	3.07 (.42)	3.35 (.38)	2.52 (.38)

Appendix E **Resting and Passive cycle exercise means and standard errors for all cardiovascular variables of Cyclists, Runners, and Controls combined.**

Table E-1: Heart rate ($b.min^{-1}$) during supine, upright rest, and passive cycle exercise for the three groups combined at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Values
Supine rest	1	60.34 (1.82)
Upright rest	1	65.88 (1.78)
30 rpm	1	70.03 (2.07)
	2	70.13 (2.09)
	3	69.07 (2.07)
	4	70.75 (2.13)
	5	71.48 (2.21)
	6	72.02 (2.40)
60 rpm	1	74.85 (2.06)
	2	73.79 (2.34)
	3	72.53 (2.28)
	4	73.19 (2.13)
	5	73.18 (2.28)
	6	72.83 (2.26)
80 rpm	1	76.29 (2.27)
	2	75.99 (2.27)
	3	75.06 (2.32)
	4	75.54 (2.29)
	5	75.17 (2.38)
	6	74.56 (2.31)

Table E-2: Stroke volume (ml) during supine, upright rest, and passive cycle exercise for the three groups combined at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Values
Supine rest	1	127.79 (4.26)
Upright rest	1	97.06 (3.48)
30 rpm	1	98.92 (3.61)
	2	100.21 (3.88)
	3	100.65 (3.87)
	4	98.30 (3.75)
	5	98.19 (4.06)
	6	98.02 (4.11)
60 rpm	1	96.08 (3.03)
	2	98.74 (3.56)
	3	97.97 (3.71)
	4	97.93 (3.57)
	5	97.40 (3.61)
	6	100.77 (3.52)
80 rpm	1	102.88 (3.95)
	2	106.59 (3.62)
	3	105.30 (3.45)
	4	104.59 (3.87)
	5	106.22 (3.89)
	6	109.97 (3.87)

Table E-3: Stroke index (ml/m^2) during supine, upright rest, and passive cycle exercise for the three groups combined at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Values
Supine rest	1	68.07 (2.39)
Upright rest	1	51.83 (2.13)
30 rpm	1	52.84 (2.21)
	2	53.50 (2.32)
	3	53.78 (2.35)
	4	52.49 (2.26)
	5	52.47 (2.44)
	6	52.36 (2.45)
60 rpm	1	51.27 (1.83)
	2	52.77 (2.20)
	3	52.34 (2.25)
	4	52.31 (2.18)
	5	52.06 (2.19)
	6	53.82 (2.16)
80 rpm	1	54.79 (2.17)
	2	56.87 (2.13)
	3	56.15 (2.02)
	4	55.79 (2.23)
	5	56.70 (2.30)
	6	58.64 (2.24)

Table E-4: Cardiac output (litres) during supine, upright rest, and passive cycle exercise for the three groups combined at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Values
Supine rest	1	7.61 (.26)
Upright rest	1	6.28 (.19)
30 rpm	1	6.79 (.22)
	2	6.88 (.23)
	3	6.82 (.24)
	4	6.83 (.24)
	5	6.85 (.23)
	6	6.87 (.24)
60 rpm	1	7.13 (.25)
	2	7.15 (.24)
	3	6.96 (.23)
	4	7.03 (.22)
	5	7.00 (.24)
	6	7.19 (.24)
80 rpm	1	7.74 (.20)
	2	7.97 (.31)
	3	7.78 (.26)
	4	7.79 (.28)
	5	7.81 (.24)
	6	8.09 (.29)

Table E-5: Cardiac index (L/m^2) during supine, upright rest, and passive cycle exercise for the three groups combined at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Values
Supine rest	1	4.05 (.14)
Upright rest	1	3.35 (.11)
30 rpm	1	3.61 (.12)
	2	3.67 (.13)
	3	3.64 (.14)
	4	3.64 (.14)
	5	3.65 (.13)
	6	3.66 (.14)
60 rpm	1	3.80 (.15)
	2	3.81 (.14)
	3	3.71 (.14)
	4	3.75 (.13)
	5	3.73 (.14)
	6	3.83 (.12)
80 rpm	1	4.12 (.16)
	2	4.25 (.15)
	3	4.14 (.14)
	4	4.15 (.16)
	5	4.17 (.14)
	6	4.31 (.17)

Table E-6: Pre-ejection period (msec) during supine, upright rest, and passive cycle exercise for the three groups combined at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Values
Supine rest	1	73.88 (2.77)
Upright rest	1	91.53 (3.39)
30 rpm	1	85.47 (3.42)
	2	85.80 (3.51)
	3	84.87 (3.57)
	4	85.53 (3.71)
	5	86.60 (3.61)
	6	85.60 (3.68)
60 rpm	1	84.67 (3.58)
	2	84.47 (3.03)
	3	86.27 (3.55)
	4	85.80 (3.26)
	5	84.80 (3.32)
	6	85.67 (3.55)
80 rpm	1	77.33 (3.24)
	2	77.53 (2.80)
	3	78.93 (3.02)
	4	79.47 (3.22)
	5	80.60 (3.58)
	6	81.60 (3.44)

Table E-7: Left-ventricular ejection time (msec) during supine, upright rest, and passive cycle exercise for the three groups combined at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Values
Supine rest	1	302.96 (3.20)
Upright rest	1	272.51 (3.78)
30 rpm	1	261.60 (4.26)
	2	264.20 (4.29)
	3	263.73 (4.08)
	4	262.40 (4.19)
	5	261.93 (4.34)
	6	261.80 (4.46)
60 rpm	1	262.20 (3.90)
	2	264.07 (4.57)
	3	266.40 (4.78)
	4	265.53 (4.21)
	5	266.33 (4.21)
	6	267.27 (4.29)
80 rpm	1	268.20 (5.13)
	2	270.60 (4.37)
	3	270.13 (4.09)
	4	265.20 (4.75)
	5	269.33 (4.43)
	6	272.13 (4.92)

Table E-8: Pre-ejection period/left-ventricular ejection time (PEP/LVET ratio) during supine, upright rest, and passive cycle exercise for the three groups combined at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Values
Supine rest	1	.24 (.01)
Upright rest	1	.34 (.01)
30 rpm	1	.33 (.02)
	2	.33 (.02)
	3	.33 (.02)
	4	.33 (.02)
	5	.34 (.02)
	6	.33 (.02)
60 rpm	1	.33 (.02)
	2	.32 (.01)
	3	.33 (.02)
	4	.33 (.01)
	5	.32 (.01)
	6	.32 (.02)
80 rpm	1	.29 (.01)
	2	.29 (.01)
	3	.29 (.01)
	4	.30 (.01)
	5	.30 (.02)
	6	.30 (.01)

Table E-9: Systolic blood pressure (mmHg) during supine, upright rest, and passive cycle exercise for the three groups combined at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Values
Supine rest	1	115.14 (1.61)
Upright rest	1	131.59 (2.29)
30 rpm	1	141.30 (3.01)
	2	143.47 (2.76)
	3	141.97 (2.93)
	4	141.20 (2.92)
	5	141.80 (3.09)
	6	142.40 (2.99)
60 rpm	1	144.00 (3.24)
	2	143.30 (3.31)
	3	143.83 (3.28)
	4	143.10 (2.88)
	5	142.33 (3.07)
	6	143.87 (3.10)
80 rpm	1	147.37 (3.23)
	2	145.73 (3.18)
	3	146.03 (3.41)
	4	143.77 (3.51)
	5	144.83 (3.93)
	6	142.57 (3.89)

Table E-10: Diastolic blood pressure (mmHg) during supine, upright rest, and passive cycle exercise for the three groups combined at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Values
Supine rest	1	64.93 (2.27)
Upright rest	1	83.19 (2.57)
30 rpm	1	92.33 (2.46)
	2	91.57 (2.63)
	3	90.40 (2.68)
	4	90.97 (2.77)
	5	90.57 (2.74)
	6	90.90 (2.61)
60 rpm	1	92.97 (2.84)
	2	92.90 (2.94)
	3	93.40 (2.87)
	4	92.70 (2.63)
	5	92.60 (2.67)
	6	93.73 (2.80)
80 rpm	1	94.20 (2.61)
	2	94.47 (2.58)
	3	93.57 (2.61)
	4	93.33 (2.84)
	5	92.23 (2.89)
	6	92.27 (2.98)

Table E-11: Mean arterial pressure (mmHg) during supine, upright rest, and passive cycle exercise for the three groups combined at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Values
Supine rest	1	81.16 (2.07)
Upright rest	1	99.03 (2.37)
30 rpm	1	108.33 (2.41)
	2	108.53 (2.48)
	3	107.20 (2.58)
	4	107.40 (2.64)
	5	107.30 (2.69)
	6	107.73 (2.55)
60 rpm	1	109.63 (2.76)
	2	109.33 (2.86)
	3	109.87 (2.81)
	4	109.23 (2.51)
	5	109.00 (2.58)
	6	110.10 (2.69
80 rpm	1	111.53 (2.53)
	2	111.27 (2.48)
	3	110.73 (2.66)
	4	109.77 (2.83)
	5	109.50 (3.06)
	6	108.63 (3.11)

Table E-12: Total peripheral resistance (dyne.sec/cm^{-5}) during supine, upright rest, and passive cycle exercise for the three groups combined at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Values
Supine rest	1	890.49 (42.09)
Upright rest	1	1323.78 (53.02)
30 rpm	1	1324.37 (58.13)
	2	1312.87 (58.02)
	3	1310.87 (58.88)
	4	1310.70 (58.42)
	5	1305.57 (59.25)
	6	1311.43 (61.87)
60 rpm	1	1288.23 (60.81)
	2	1278.23 (61.72)
	3	1319.97 (62.71)
	4	1219.23 (56.87)
	5	1302.93 (64.09)
	6	1263.27 (50.26)
80 rpm	1	1215.50 (59.66)
	2	1153.57 (45.36)
	3	1176.93 (47.66)
	4	1183.77 (58.83)
	5	1156.40 (48.99)
	6	1125.13 (55.34)

Table E-13: Rate pressure product (HR x SBP/100) during supine, upright rest, and passive cycle exercise for the three groups combined at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	Data	Values
Supine rest	1	69.49 (2.35)
Upright rest	1	86.84 (3.01)
30 rpm	1	98.97 (3.63)
	2	100.55 (3.58)
	3	98.06 (3.71)
	4	100.05 (3.84)
	5	101.38 (3.93)
	6	102.41 (4.02)
60 rpm	1	108.01 (4.14)
	2	105.81 (4.38)
	3	104.23 (4.15)
	4	104.49 (3.55)
	5	103.97 (3.88)
	6	104.97 (4.23)
80 rpm	1	112.32 (4.26)
	2	110.80 (4.25)
	3	109.82 (4.54)
	4	108.43 (4.21)
	5	108.92 (4.69)
	6	106.43 (4.65)

Table E-14. Time series (msec^2) at low (0.07-0.11 Hz) and high (0.12-0.40 Hz) frequencies during baseline and passive cycle exercise for the three groups combined at each intensity (30 rpm, 60 rpm, and 80 rpm). Data are means with standard errors of the means in parenthesis.

Activity	low (0.07-0.11 Hz)	high (0.12-0.40 Hz)
Baseline	3.25 (.18)	8.36 (.19)
30 rpm	3.24 (.18)	7.69 (.25)
60 rpm	3.13 (.18)	7.47 (.24)
80 rpm	2.98 (.23)	7.30 (.24)

Appendix F Physical Activity Readiness Questionnaire (PAR-Q)

PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (PAR-Q)
A Self-administered Questionnaire for Adults

PARTICIPANT IDENTIFICATION

PAR-Q & YOU

PAR-Q is designed to help you help yourself. Many health benefits are associated with regular exercise, and the completion of PAR-Q is a sensible first step to take if you are planning to increase the amount of physical activity in your life.

For most people physical activity should not pose any problem or hazard. PAR-Q has been designed to identify the small number of adults for whom physical activity might be inappropriate or those who should have medical advice concerning the type of activity most suitable for them.

Common sense is your best guide in answering these few questions. Please read them carefully and check the ❑ YES or NO opposite the question if it applies to you.

YES	NO		
❑	❑	1	Has your doctor ever said you have heart trouble?
❑	❑	2	Do you frequently have pains in your heart and chest?
❑	❑	3	Do you often feel faint or have spells of severe dizziness?
❑	❑	4	Has a doctor ever said your blood pressure was too high?
❑	❑	5.	Has your doctor ever told you that you have a bone or joint problem such as arthritis that has been aggravated by exercise, or might be made worse with exercise?
❑	❑	6.	Is there a good physical reason not mentioned here why you should not follow an activity program even if you wanted to?
❑	❑	7.	Are you over age 65 and not accustomed to vigorous exercise?

IF YOU ANSWERED

YES to one or more questions

If you have not recently done so, consult with your personal physician by telephone or in person BEFORE increasing your physical activity and/or taking a fitness test. Tell him what questions you answered YES on PAR-Q, or show him your copy.

programs

After medical evaluation, seek advice from your physician as to your suitability for:

- unrestricted physical activity, probably on a gradually increasing basis.
- restricted or supervised activity to meet your specific needs, at least on an initial basis. Check in your community for special programs or services.

NO to all questions

If you answered PAR-Q accurately, you have reasonable assurance of your present suitability for:

- A GRADUATED EXERCISE PROGRAM- A gradual increase in proper exercise promotes good fitness development while minimizing or eliminating discomfort.
- AN EXERCISE TEST- Simple tests of fitness (such as the Canadian Home Fitness Test) or more complex types may be undertaken if you so desire.

postpone

If you have a temporary minor illness, such as a common cold.

Figure 3.1 The Physical Activity Readiness Questionnaire (PAR-Q) is useful in health fair or mass testing situations for screening out individuals at risk for cardiovascular or metabolic disease.[3]

Appendix G Information For Subjects

INFORMATION FOR SUBJECTS

ITEM 1: PROJECT OBJECTIVES

The aim of the project is to investigate the cardiovascular regulation during passive cycle exercise.

ITEM 2: RATIONALE

Passive cycle exercise causes blood to return to the heart which causes an increase in stroke volume. As trained cyclists and runners possess greater blood volume, we expect that they will record greater increases in stroke volume than the untrained.

ITEM 3: TEST PROCEDURES

Testing will involve application of surface electrodes and a blood pressure cuff (on the third finger). Measures of cardiovascular function, such as heart rate and blood pressure, will be collected during the experimental session. The study consists of measurement of cardiovascular response during resting position and during passive cycle exercise. Passive cycle exercise involves sitting on a fixed wheel bike that was secured on a treadmill; the treadmill moves the pedals of the bike and consequently the lower limbs of the person sitting on the bike. Subjects make no effort to cycle and simply sit on the bike and relax. The passive cycle exercise is performed against no resistance at three different intensities continuously, for three minutes, at each intensity.

ITEM 4: RISKS AND DISCOMFORTS

Before the session begins, your cardiac function will be screened for abnormalities; this will include examination of the three major heart leads and the impedance cardiogram (e.g., abnormal axis, conduction problems, assessment of the impedance wave for valvular problems). During the experimental session it is anticipated that your heart rate and blood pressure will rise, although <u>not</u> to levels higher than you would commonly experience.

ITEM 5: INQUIRIES

Questions concerning the procedures and/or rationale used in this study are welcome at any time. Please ask for clarification of any point which you feel is not explained to your satisfaction. Your initial contact person is the investigator conducting this project (Dr. Steve Boutcher, Department of Biomedical Science: phone 214-093). Subsequent inquiries may be directed to Karen McRae (Secretary of the Human Ethics Committee, phone 214-457).

ITEM 6: FREEDOM OF CONSENT

You are free to deny consent before or during the experiment. In the latter case such withdrawal of consent should be made at the time you specify, and not at the end of a particular trial. Your participation and/or withdrawal of consent will <u>not</u> influence your present and/or future involvement with the University of Wollongong. You have the right to withdraw from any experiment, and this right shall be preserved over and above the goals of the experiment.

ITEM 7: CONFIDENTIALITY

All questions, answers, and results of this study will be treated with absolute confidentiality. Subjects will be identified in the resultant manuscripts, reports or publications by the use of subject codes only.