# CARDIAC STRUCTURE AND FUNCTION IN YOUNG ATHLETES

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## **ABSTRACT**

The aim of the study was to examine the effects of a long-term endurance-training on resting and exercising cardiac responses on male adolescents. The sample consisted of 13 endurance-trained and 7 non-athletic male adolescents (x  $\pm$  SE, age = 15.33  $\pm$ 0.33 and  $15.15 \pm 0.23$  years, respectively). The volunteers underwent echocardiography at rest to determine left ventricular end diastole dimension (LVDd) and end systole dimension (LVDs), left ventricular posterior wall (PW), stroke volume (SV) and cardiac output (CO). On separate days, tests were conducted on a cycle ergometer to measure VO<sub>2</sub>max and anaerobic power, and skeletal muscle strength and endurance was determined on an isokinetic dynamometer. The relative peak oxygen uptake ( $VO_2$ max ml/kg/min) was significantly higher (p < 0.05) in the endurance group (54.4  $\pm$  1.8) than the control group (45.8  $\pm$  1.6). The mean trial time was significantly longer in the endurance group (12.9  $\pm$  0.7 minutes) than the control  $(10.4 \pm 0.8 \text{ minutes}; p < 0.05)$  and there was a significant relationship (r= 0.51; p < 0.05) between the length of test and peak oxygen uptake. No significant differences were noted between the two group in rest heart rate, peak heart rate, LVDd, LVDd/BSA, LVDs, LVDs/BSA, PW, PW/BSA, stroke volume (SV), SV indexed,

cardiac output (CO) and CO indexed. The data indicate that endurance-trained male adolescents had higher value of maximal oxygen uptake than untrained male adolescents. The improvements in maximal oxygen uptake were associated with longer exercise time. Endurance-trained adolescents did not exhibit greater left ventricular internal dimension, left ventricular wall thickness, SV and CO at rest compared to the untrained. This observation suggests that endurance training stimulus need to be of greater intensity, duration and frequency in adolescent. In addition, the control group may have been engaged in sufficient exercise as to blur any differences that otherwise may have occurred. Furthermore, genetic factors may be more important in determining cardiovascular development and performance in adolescents, than was previously thought.

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#### **CHAPTER 1**

#### 1.0 INTRODUCTION

Cardiovascular capacity is an important factor in sport and physical activity.

During exercise, the cardiovascular system serves as the body's mechanism for transportation of gases, nutrients and metabolic products. Although the effects of long term exercise training on the structure and function of the heart have been described for adults, they are not as well understood for children and adolescents.

In adult athletes, the structure of the cardiovascular system adapts to intensive long - term endurance training. Ikaheimo (1979) studied the effects of chronic exercise training on 10 sprinters and 12 endurance runners using ECG, echocardiography and chest x-ray film. For both groups of athletes the left ventricular end-diastolic volume, and the left ventricular wall thickness and mass were greater than normal, but the wall thickness and mass in the endurance runners were higher than in the sprinters. There is evidence that adaptations observed in the cardiac structure and function of athletes include left ventricular enlargement, chamber wall thickening, an increase in cardiac contractility, an

increase in cardiac output and electrocardiograph (ECG) changes. Stork et al. (1992) used 2-dimensional Doppler echocardiography to evaluate left ventricular filling in 25 endurance adult athletes (aged 31±9 years) of different training levels at rest and during cycling exercise. The results showed that the athletes with a higher training loads had a more pronounced increases of left ventricular dimension (LVD), left ventricular mass and stoke volume index. This increase indicates a beneficial effect of additional training on LV systolic structure and function. At the same time, the endurance-trained athletes showed normal LV filling at rest. Smith et al. (1994) estimated myocardial mass of seven college wrestlers (above 18 years of age) before and after a competitive season. The results suggested that significant increases in myocardial mass occurred over the course of a competitive wrestling season associated with increased left ventricular wall thickness.

Due to methodological and ethical constraints, many researches have reported that specific cardiac adaptations occur with specific exercise training in adults. Studies investigating the cardiac adaptive changes to exercise training in children (especially in adolescent athletes) are rare. We have found reports about how the changing of cardiac structure and function exactly in adolescent athletes (aged 14)

to 16 years old). A major methodological hurdle is that numerous physiological functions in children and adolescents that enhance exercise performance are affected not only by training but also by growth and maturation. Also, it relatively uncommon for children to engage in just one mode of intensive exercise training over a period of time sufficient to elicit specific changes to cardiac structure and function (compare adult athletes). There are many questions that arise as how exercise training may affect the cardiac structure and function of children and adolescents. Are cardiac structure and functional adaptive changes to exercise training in children and adolescents similar to changes observed in adult population? Can the specificity of training be detected in the heart of young athletes who have specialized in specific sports? Do children with certain cardiac advantages, such as increased stroke volume at rest, choose to participate in endurance activity, or is the training primarily responsible for the cardiac adaptations. These are interesting questions and a study about cardiac structure and function in young athletes during rest and exercise may provide answers to these.

Adolescent athletes are growing and heart function is an important factor during growth and exercise, so it is of significance to find out how prolonged specific

exercise training affects adolescent athletes' heart function. This study will be the first to compare the effects of different types of athletic training on the cardiac structure and function of adolescents. According to the results of research, we may make suggestions about what cardiac characteristics can be expected from young athletes and the type of training with which these changed (if any occur) can be associated.

Most previous studies comparing cardiac structure and function in athletes and non-athletes were cross-sectional in nature, and so it is not possible to elucidate whether the exercise training is responsible for all, or some, of the traits. The alternative concept is that athletes who already have cardiac advantages engage in self-selection in to these endurance-type activities. It would require extensive longitudinal studies to shed more lightly on these alternative hypotheses. Or perhaps cardiac structure and function in athletes is a combination of both exercise training and pre-existing talent. In the current research, cross-sectional observations were made for young, adolescent athletes and age-matched sedentary controls. If the differences between the groups were lower than adult or even absent, then this lends some indirect support for the hypothesis that it was the

exercise training itself which exerted strong influences on cardiac structure and function.

On the basis of the results of research, we may be able to describe specific characteristics of heart function in young athletes and it may be possible to identify children who will have particular aptitudes to certain activities.

#### 1.1 RATIONALE

Some studies within the literature have described cardiovascular adaptations following intensive and long-tern training in adults. The rationale behind this study is that adolescent athlete who have been involved for over 12 months of endurance exercise, might exhibit cardiac structure and functional adaptive changes that differ from a relatively sedentary control group. For example, during endurance training an increased left ventricular volume may cause eccentric cardiac enlargement (ie increased outside diameter), and also be associated with an increased left ventricular internal diameter. Conversely, strength-trained athletes may present a concentric cardiac enlargement, with an increased left ventricular wall thickness with small or absent changes to outside cardiac

dimensions, as a result of high arterial blood pressures elicited during high-intensity muscular conditioning. Other physiological adaptations may also prevail in aerobic power, strength and body composition.

Adolescent athletes are growing and heart function is an important factor during growth and exercise. If a better physiological understanding of the adolescent athletes' heart function can be achieved, (heart rate, stroke volume, cardiac output, left ventricular end-diastolic volume, left ventricular end-systolic dimension, left ventricular diastolic posterior wall thickness, VO<sub>2</sub> peak, high intensity sprints-anaerobic power), suggestions may be made about what cardiac characteristics can be expected from young athletes and the type of training with which these changes (if any occur) can be associated.

#### 1.2 THE SIGNIFICANCE AND PURPOSE OF THE STUDY

The purpose of the study will be to compare the effects of a long-term endurance training on resting and exercising cardiac responses in two groups of adolescents (endurance and control groups). Additional laboratory testing will determine if

any differences exist among the groups for maximal aerobic power ( $VO_2$  peak), muscle strength, muscle power, body composition.

#### 1.3 LIMITATIONS

In conducting the study, the following limitations are recognized:

## 1.3.1 The nature of training

It was not possible for the investigator to control the intensity, duration, frequency or even specific mode of exercise training for the active participants. It was also not possible to control any other form of exercise training, such as compulsory sport or physical education, or informal activities such as walking, playing and other leisure activities, that the participants were involved with. At the time of enrolment in the study, the investigator needed to make decisions regarding the suitability, or otherwise, of each participant for allocation in to either the Exercise Group or the Control Group.

#### 1.3.2 The nutritional status

All participants were requested to consume a moderate balanced meal including some carbohydrates two hours prior to the exercise testing.

Other factors that may influence cardiac structure and function

Factors such as maturational age, and genetic factors that may be important in the development of the heart in parallel to the effects of exercise training were neither assessed, nor controlled for.

#### 1.4 DELIMITATIONS

1.4.1 The study was delimited to boys whose age ranged from fourteen to sixteen years.

1.4.2 The number of participants was restricted to twenty, with thirteen participants in endurance groups and seven in control group.

The original objective was to compare three group of boys: (i)

endurance-trained; (ii) strength-trained; and (iii) a sedentary control group. It proved to very difficult to recruit boys who had engaged predominantly in strength training for the previous 12 months, and so the project needed to be modified to eliminate the second group. For the present study the two testing groups were endurance training group and control groups. The anaerobic test is for compare the peak power between groups. Criteria for inclusion into the endurance-trained group was that the participants had participated in endurance training at least three times per week for the past year. Participants of control group should not have been involved intensive sports training for more than once per week during the past year. This meant that compulsory attendance of a weekly Physical Education class was not a reason for exclusion from the study.

1.4.3 Selection criteria for all two groups included the need for all participants to be free of injuries at the time of testing and for three months prior to testing. In addition, participants (and their parents) needed also to be able to report an absence of long term use of medications that may affect exercise performance, eg beta blockers.

Recruits for this project were asked to volunteer following permission from their parents, and their respective institutions and coaches.

The nature of testing was selected to complement the focus of the research.

Testing was therefore delimited to tests for endurance ( $VO_2max$ ), strength (Cybex testing), cardiac function (ECG) and structure (echocardiography), and body composition.

#### 1.5 DEFINITION OF TERMS

During the study the following definition of terms will be used:

#### 1.5.1 Anthropometric Measures

These terms refer to body height, body weight and body composition:

Body height (cm). The linear size measure of the body.

Body weight (kg). The weight of the total body composition of a person.

Skinfold Measurement (mm). An indication of subcutaneous fat development at a specific site on the body.

#### 1.5.2 Cardiovascular Structure and Function Measures

This generic term refers to the several measures that may reflect cardiovascular structure and function of the participants in the study such as heart rate and maximal oxygen uptake, electrocardiogram (ECG) and echocardiogram.

Maximal Oxygen Uptake ( $VO_2$  max). The maximal rate of oxygen consumed by a participant during incremental exercise on a cycle ergometer to volitional exhaustion.  $VO_2$ max is expressed in absolute terms ( $1.min^{-1}$ ) and in relative terms, as a function of body weight ( $ml.kg^{-1}min^{-1}$ ).

Peak Heart Rate. It is the maximal rate at which the heart of a participant is observed to be beating during the maximal oxygen uptake test.

Electrocardiogram (ECG). The ECG is an electronic device that is used to detect the electrical changes associated with contraction of the heart muscle by electrodes attached at specific sites on the surface of the body.

Echocardiogram. Echocardiograms employ the harmless emission of ultrasound waves to assess both the function and structure of the heart. An echocardiogram can be used to measure stroke volume, cardiac output, left ventricular end-diastolic dimension (LVEDd), left ventricular end-systolic dimension (LVEDs) and left ventricular diastolic posterior wall thickness (LVPW) of heart.

## 1.5.3 Anaerobic Test

Anaerobic tests measure the power produced during a brief exercise test under the demands of an intensive exercise load. The anaerobic component of this testing comprised of 5 bouts of 6 seconds of maximal sprint cycling with 24 seconds for recovery between each. This test was performed on a modified air-braked Repco cycle ergometer.

#### 1.5.4 Isokinetic Cybex Dynamometer

This is a measure of muscular strength and power, using 'peak torque' and 'peak power,' as measures of concentric contraction. For the purpose of the present study it was used to measure both of leg strength while the participant sat on a seat

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and flexed and extended the lower part of his leg against a relatively heavy resistance (ie a constant angular velocity of 60 degrees per second through a range of approximately 90 degrees). At the same time, both of leg power was measured by asking the participant to kick his lower leg 5 times against a lighter resistance(180 degrees per second).

## **Glossary of Terms**

Term	Description
Body height	The linear size measure of the body
Body weight	The weight of the total body composition
	of a person
Skinfold measurement	An indication of subcutaneous fat
	development at a specific site on the body
Heart rate	Frequency of the cardiac cycle per minute
Peak heart rate	The maximal heart rate at which the heart
	of a participant is observed to be beating
	during the maximal oxygen uptake test
Stroke volume (SV)	The amount of blood ejected from the
	ventricles during systole

Cardiac output (CO)	The amount of blood pumped by the heart
	per minute
Afterload	Afterload is impedance or resistance to
	ventricular emptying
Cardiac reserve	The maximum percentage that the cardiac
	output can increase above normal
Left ventricular end-diastolic dimension	Main chamber size while heart is relax
(LVDd)	
Left ventricular end-systolic dimension	Main chamber size while heart contract
(LVDs)	
Left ventricular diastolic posterior wall	Rear wall thickness of main chamber while
thickness (LVPW)	heart is relax
Maximal oxygen uptake (VO <sub>2</sub> max)	The maximal rate of oxygen consumed by
	a participant during incremental exercise
	on a cycle ergometer to volitional
	exhaustion. VO <sub>2</sub> max is expressed in
	absolute terms (l.min <sup>-1</sup> ) and in relative
	terms, as a function of body
	weight(ml.kg <sup>-1</sup> min <sup>-1</sup> )

High intensity sprints anaerobic power	The power produced during a brief exercise
	test under the demands of an intensive
	exercise load
Echocardiogram (ECG)	An electronic device that is used to detect
	the electrical changes associated with
	contraction of the heart muscle by
	electrodes attached at specific sites on the
	surface of the body

#### **CHAPTER 2**

#### REVIEW OF LITERATURE

The following review of literature is divided into five major sections. These sections are: (1) Overview of the Anatomy and Function of the Heart, (2) Methods of Evaluating Cardiac Structure and Function, (3) Changes in Cardiac Structure and Function with Age, (4) Changes in Cardiac Structure and Function with Training in Adults, (5) Changes in Cardiac Structure Function with Training in Younger Population.

#### 2.1 Overview of the Anatomy and Function of the Heart

The cardiovascular system consists of the heart together with the vessels of the general circulation (systemic circulation) and with the vessels of the lungs (pulmonary circulation). The heart is the major organ of the cardiovascular system. It pumps the blood and forcing it into the blood vessels, which then deliver the blood to all parts of the body to exchange building and fuel material,

metabolic products, heat and messenger substances among the various organs and tissues.

#### 2.1.1 The four chambers of heart

The heart is a hollow, muscular organ that lies in the mediastinim between the lungs. It has four chambers – the right and left atria, the right and left ventricles, which are lined with endocardium, the heart valves, a muscular wall (myocardium) and an outer sac surrounding the heart (pericardium). The atria receive blood returning to the heart. The ventricles pump blood out into the great arteries. The left ventricle is thicker than the right ventricle because it has to pump blood against the much greater resistance presented by the vessels that deliver blood to all the body systems.

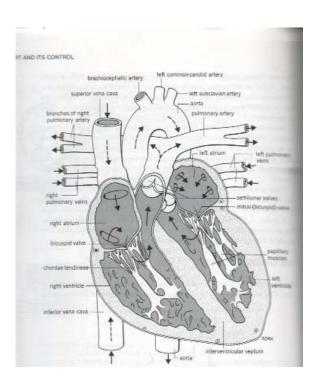
The route of the blood circulation is following:

low-oxygenated blood returns to the right atrium of the heart through the inferior and superior vena cavae. Then the blood flows from the right atrium to the right ventricle. The right ventricle pumps it into the pulmonary circulation through the pulmonary arteries. During pulmonary circulation, gases in blood are exchanged,

then deoxygenated blood change to oxygenated blood. After that, the left atrium receives the oxygenated blood from the pulmonary veins and sent it to the left ventricle. The left ventricle pump blood into the great arteries and the systemic circulation (where the blood offers oxygen and fuels to various tissues, so oxygenate blood change to deoxygenated blood). At last deoxygenated blood return to the inferior and superior vena cavae again.

Diagram 2.1

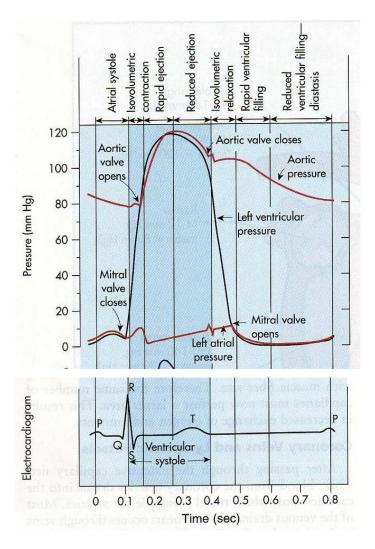
The chambers of the heart.



(SOURCE: From J.E. Crouch and J.R. McClintic, 1971.)

## 2.1.2 The cardiac cycle

Diagram 2.2 The cardiac cycle



(SOURCE: From M.O.Catherine and S.p.Alan. 1995)

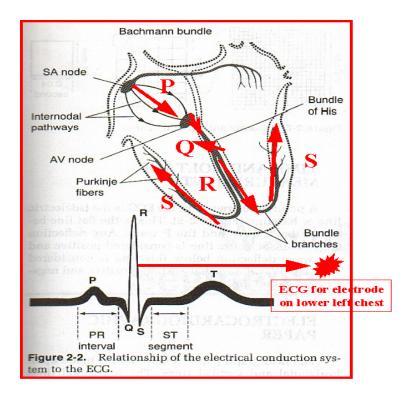
The cardiac cycle is a sequence of events that occurs during one complete heartbeat. Each cardiac cycle involves atria contraction and ventricular contraction. As the atria contract, additional blood is forced into the ventricles.

Then ventricular contracting that follows, forces blood through the semilunnar valves into the systemic and pulmonary circulation. At the same time the atria have returned to a relaxation status (diastole) and are again filling with blood.

The volume of blood pumped by the ventricle during one beat is called the stroke volume (SV). Cardiac output (CO) is the amount of blood pumped by one ventricle in one minute. Cardiac output is the product of stroke volume and heart rate. There are many factors that affect cardiac output. The main factors are venous return, afterload, heart rate and the force of contraction of the cardiac muscle fibers. The greater the amount of blood delivered to the heart by the veins or contractility of the heart, the more blood the heart pumps. Increased SV and heart rate results in a increased CO. On the other hand, Increased after load has a negative influence on cardiac performance because it creates an increased work load for the heart (George et al. 1985).

#### 2.1.3 The electrical activity of the heart

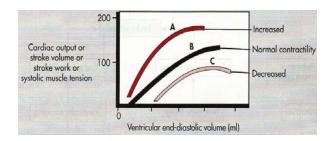
Diagram 2.3 Relationship of the electrical conduction system to the ECG.



The heart has its own conduction system and can beat independently of its nerve supply. However, elaborate mechanisms exist to regulate the heartbeat so that its rate and strength of contraction adjusts to the changing needs of the body.

#### 2.1.4 Venous return

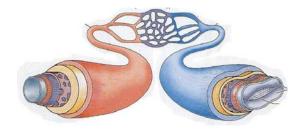
Diagram 2.4 Relationship of the ventricular end-diastolic volume to the CO or SV.



The volume of blood returning to the heart is called as "preload". According to Starlings law of the heart, the more blood returned to the heart by the veins, the greater the volume of blood that will be pumped during the next contraction (systole). In physiological terms, preload refers to myocardial fiber length. If the amount of blood delivered to the heart increases, then the myocardial fibers lengthen, and a greater amount of blood is pumped out of the heart. Endurance activities such as distance running significantly contribute to end-diastolic volume increases, which increases the preload component of cardiac stress. Bello et al. (1995) studied in a group of 10 professional elite bicyclists (mean age: 25.1 years) during competitive activity, echocardiographically and Doppler derived. During the aerobic period a significant increase in end diastolic volume was observed due to the greater venous return from exercising muscles.

#### 2.1.5 Afterload

Figure 2.1



"Afterload" refers to the resistance against which the ventricles must pump when ejecting blood. If the afterload increase, it may be more difficult for the heart to pump blood forward into the circulation. For example, in hypertension, high peripheral resistance to blood flow results in an increased afterload stress that can cause the heart muscle to hypertrophy and sometimes fail. Higher blood pressure is, greater afterload. During strength training, high muscle tension can cause an increased afterload stress, and then results in a greater left ventricular mass and wall thickness. Fleck (1989) studied the cardiac structure of 14 junior elite Olympic weight lifters (mean age 18.4 years) and 14 controls (mean age 17.8 years). Systolic and diastolic left posterior wall thickness, left ventricular short axis, left ventricular transverse long axis, septal wall thickness, right ventricular wall thickness, and right ventricular short axis were determined by using cardiac magnetic resonance imaging. The result indicated left ventricular diastolic internal dimension, right ventricular wall thickness are not affected by weight lifting in young athletes. However, left ventricular wall thickness and intraventricular septal wall thickness are both greater in absolute and relative to body surface area, lean body mass.

#### 2.1.6 The cardiac contraction

When the force of contraction of the cardiac muscle fibers increases, the stroke volume increases, and this in turn increases cardiac output.

#### 2.1.7 Heart rate

Heart rate is the one of the two major determinants of cardiac output. Heart rate is particularly important during moderate to heavy exercise as the stroke volume plateaus at approximately 40% of maximal effort (VO<sub>2</sub>max). A number of other factors can influence the heart rate, including hormones, ion concentration, and changes in body temperature, exercise, sex and age.

## 2.2 Methods of Evaluating Cardiac Function

During clinical examination and research testing there are many methods of evaluating the cardiac function of patients or participants.

#### 2.2.1 Heart rate

Information about heart rate with exercise and rest is the most reliable of the cardiovascular variables, since values can be accurately measured at all intensities of physical activity by electrocardiography or other electronic methods. It is useful, then, to analysis cardiovascular responses to exercise with an examination of changes in maximal and sub maximal heart rate in young populations.

#### 2.2.2 Blood pressure

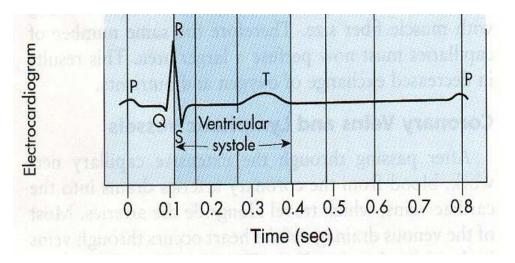
Blood pressure is the pressure exerted by the blood on the blood vessels of the body. If blood pressure is too high, the heart has to work hard to overcome the pressure and, as such, the health of the heart is compromised.

#### 2.2.3 Electrocardiography (ECG)

An ECG measures the electrical activity of the heart and is often recorded it on graph paper. The graphical display from an ECG allows the evaluation of the sequence and magnitude of the electrical impulses generated by the heart. Several electrode positions are used for recording the surface electrocardiogram: standard leads I, II, III (Einthoven leads), aVR, aVL and aVF (Goldberger leads), and the

unipolar chest leads  $V_1$ - $V_6$  (Wilson's leads). The 12-lead recording makes up the standard electrocardiogram recording, althugh other lead positions can be used for research purposes. The normal ECG consists of the P wave, P-R interval, QRS complex, T wave, Q-T interval, and ST segment.

Diagram 2.5



Information supplied by an ECG includes heart rate and rhythm, abnormalities of conduction, muscular damage, hypertrophy, effects of electrolyte imbalance.

Diagram 2.6 The graphical display from an ECG



(SOURCE: From John R. Hampton, THE ECG MADE EASY.1998, p. 5.)

#### 2.2.3.1 The normal ECG includes:

a) limits of normal durations. PR interval: 2	a)	∠UUIIIS
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QRS complex duration: 120ms

QT interval: 400ms

b) Rhythm. Sinus arrhythmia

Supraventricular extrasystoles are always normal.

- c) Cardiac axis. ORS complex predominantly upward in leads I, Il, Ill.
- d) QRS complex. Small Q waves normal in leads l, VL,  $V_6$

R wave smaller than S wave in  $V_1$ 

R wave in  $V_6$  less than 25 mm

R wave in  $V_6$  plus S wave in  $V_1$  less than 35 mm

- e) ST segment should be isoelectric.
- f) T wave may be inverted in leads III, VR, and  $V_1$ .

#### 2.2.3.2 ECG disorder included conduction problems and Rhythm

abnormalities.

a) Conduction problems: First degree block

Second degree block

Third degree block

Right bundle branch block

Left bundle branch block

b) Rhythm abnormalities: Extrasystoles

Excape beats

**Tachycardias** 

Bradycardias

Special uses of the ECG include (1) continuous ambulatory monitoring, which employs a Holter monitor, a transistorized tape recorder attached to chest leads.

Twenty-four-hour ECG recording is used to clarify the patient's symptoms and to document intermittent disorders of rate or rhythm which would not normally be picked up on the routine 12-lead ECG. (2) The exercise stress assessment, in which the ECG is monitored during the form of incremental levels of exercise, usually on a treadmill or bicycle. Various protocols have been devised but usually take the form of 3min of exercise at each stage, with the subject progressing to the next level until the target heart rate or expected level of submaximal or maximal work activity has been achieved. The information about the physical fitness, the

appearance of myocardial ischaemia and the occurrence of exercise-induced arrhythmias can be obtained from the exercise ECG. In many patients the resting electrocardiogram is normal, but abnormalities are brought out by physical exertion.

The ECG is an acknowledged sensitive screening tool for the common causes of sudden cardiac death in young athletes. Fuller et al. (1997) examined the cardiac function in 5,615 male and female high school athletes. Significant "abnormal" was detected in 1 per 255 athletes by echocardiography. The history of cardiac problems was unable to explain any of the detected "unusual" recordings.

Auscultation/inspection was found in ratio of 1:6,000 athletes, High blood pressure measurement was reported in a ratio of 1:1,000 athletes, and disorder of the ECG was in a ratio of 1:350. The results of the study suggested that the ECG was an effective screening tool in detecting cardiovascular function before approval for participation in sports was given.

# 2.2.4 Echocardiography

Echocardiography is one of the most frequently used tests for detecting cardiac function in children. This is a non-invasive method of imaging the heart using ultrasound waves. Recent improvements in echocardiographic techniques have made it increasingly possible to confirm the diagnosis without resorting to cardiac catheterization. It provides detailed information on cardiac structure and is a first-line investigation in many forms of heart disease. Its uses include:

- a) Clarification of cardiac anatomy and valve function.
- b) Left ventricular wall thickness.
- c) Ventricular function.
- d) Intracavity abnormalities.
- e) Pericardial diseases, Prosthetic valves and Aortic root.

Routinely, the echocardiogram will include measurements of the dimensions of the left atrium, left and right ventricles, aortic root and ventricular septal thickness.

Echocardiography involves the use of ultra-high-frequency sound waves to produce an image of the heart's structure. A transducer placed directly on the

chest wall delivers repetitive pulses of ultrasound and processes the returned signals.

There are basically two modes of Tran thoracic echocardiograms, A mode and M mode. Historically, cardiac ultrasound began with a single-crystal transducer display of the amplitude A of reflected ultrasound versus depth on an oscilloscope screen. With the time dimension shown explicitly on the horizontal axis and ultrasound beam converted to a corresponding gray scale level, a motion (M) mode display is produced. Motion mode provides a one-dimensional view of the heart and is useful in determining its size, the presence or absence of structures, and their relationship to one another. M-mode echocardiography allows measurement of ventricular internal dimensions and wall thickness throughout the cardiac cycle. The major advantage of M-mode echo is high time resolution, which facilitates recognition of endocardial borders. A potential disadvantage of M-mode dimensions is that overestimation will occur if the beam is oblique with respect to the long or short axis of the ventricle. Underestimation can occur if the M-line is not centered in the ventricular chamber. A two-dimensional (2D) echocardiographic image is generated from the data obtained by mechanically or electronically "sweeping" the ultrasound beam across the tomographic plane. A

two-dimensional (2D) echocardiogram provides information about spatial relationships between structures, for example, visual estimates of global and regional function from two-dimensional echocardiography images, quantitative ventricular volumes and ejection fractions based on endocardial border tracing. A pulse echocardiogram is primarily a velocity-sensing system and is generally used with 2-D echo to provide information about volume flow rate. Depending on the type of test, information can be obtained regarding the integrity of septa; chamber size; position and contractility; presence, position, size, and function of the valves; velocity of blood flow; and the relationship between, and size of the great vessels.

The echocardiogram is noninvasive, painless, and associated with no known side effects. Echocardiography therefore can be traumatic for young populations.

## 2.2.5 Maximal oxygen uptake (VO<sub>2</sub>max)

Maximal oxygen uptake is the most common and most important measurement derived from gas exchange data during exercise. Gas exchange techniques can provide a great deal of additional information regarding the capacity of the heart

and lungs to deliver oxygen to the working muscle during exercise. No other measure of work is as encompassing, precise, or reproducible as VO<sub>2</sub>max.

Maximal oxygen consumption is the maximal volume of oxygen that can be transported to, and consumed by, the working tissues. A high oxygen uptake is an important factor contributing to the endurance capacity in athletes competing in prolonged exercise. The more oxygen that can be transported to, and consumed by the exercising muscles, the better for the young endurance athlete. VO<sub>2</sub>max may reflect the cardiac capacity and the pulmonary capacity of oxygen transport of athletes. VO<sub>2</sub>max may also be directly related to cardiac output and arterial-venous oxygen saturation, oxygen muscular extraction and an activated aerobic metabolism. Bello (1995) studied the relationship between the Cardiac Output (CO) and the maximum oxygen uptake. He used echocardiograph testing with the evaluation of ergospirometric parameters for the analysis of ventilatory gas exchange during maximal exercise, in a group of 10 elite male cyclists (mean age: 25.1 years). A linear regression relationship was found between VO<sub>2</sub>max and CO, r = 0.84, p<0.0001. The links between oxygen output and the heart, oxygen delivery, were therefore reported to be strong. Changes in VO<sub>2</sub>max following training or detraining closely parallel changes in cardiac size and maximal cardiac

output. Clearly,  $VO_2$ max is directly related to the integrated function of several systems.

The determination of oxygen uptake requires the ability to measure three variables:

- a) The fraction of oxygen in the expired air.
- b) The fraction of carbon dioxide n the expired air.
- c) The volume of the inspired or expired air.

The volume of expired air is commonly collected with the use of a non-rebreathing valve. The difference in the concentration of oxygen in the ambient and exhaled air is measured using a metabolic system containing an oxygen analyzer. Oxygen uptake is the difference between the fraction of oxygen in the inspired and expired ventilation.

Aerobic capacity could be measured with the instruments, the bicycle ergometer and the motor driven treadmill. Boileau et al (1977) compared the relative merits of the bicycle ergometer and the motor driven treadmill in assessing the aerobic capacity of twenty-one healthy boys (aged 12.84±1.13 years) and related circulorespiratony responses to graded exercise. The VO<sub>2</sub>max mean values were

47.0 ml/kg.min on the bicycle and 50.5 ml/kg.min on the treadmill. The bicycle group mean VO<sub>2</sub>max was 7.9% lower than that observed during the treadmill test. The author suggested that (1)both forms of ergometry produced highly reliable values for VO<sub>2</sub>max and HRmax. (2)the difference of two VO<sub>2</sub>max values can be explained as that leg strength and subsequent local muscular fatigue may be limiting factors with the bicycle ergometer in children particularly when pedaling at high resistances to maximally tax the oxygen transport system.

## 2.3 Changes in Cardiac Shape and Function with Age

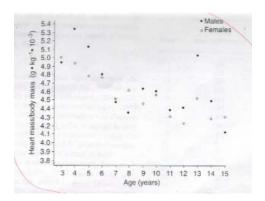
With the rise in energy requirements associated with growth during childhood, the cardiac structure and function develop continually.

## 2.3.1 Heart weight and size

The heart weight relative to body mass is high in newborn babies and declines steadily during growth. Rowland (1996) estimated the ratio of average heart mass to body mass during the childhood years, and postulasted that the ratio decreases approximately 20% from ages 3 to 15 years Therefore the size of the heart in relation to total body size is larger in infancy. The ventricle walls are more or less

equal in thickness at birth. With the growth, the left side becomes thicker than the right and pressures on the left side of the heart rise due to the increased demand of the postnatal peripheral circulation. An increase in heart size accompanies the adolescent growth spurt, with a resulting increase in blood pressure and decrease in heart rate.

Diagram 2.7 Estimated ratio of average heart mass to body mass during the childhood years.



(SOURCE: Rowland T.W. Developmental Exercise Physiology. Human Kinetics, Champangn IL, USA, 1996 page 121)

As stated previously, the left ventricular size also increases during growth. Henry et al. (1978) measured resting left ventricular end-diastolic dimension, using

echocardiograms, over the entire pediatric age span. The results suggested that left ventricular size is most closely related to body surface area (BSA) during growth.

## 2.3.2 Heart rate

Since the early of the 20th century, many researchers have studied age-related differences in basal heart rates and resting heart rates. These studies were reviewed by Rowland (1996). This suggested that both of basal heart rates and resting heart rates decrease progressively throughout the childhood years. Basal heart rates in subjects was obtained in standardized basal conditions (during the post absorptive state, at least 12 hr after the previous meal, and after lying quietly for at least 30 min). Resting heart rates were taken as participants were sitting in a chair 2-4 hr after their last meal. Table 2.1 describes an age-related differences in basal heart rates and resting heart rates.

Table 2.1 Heart rates in different ages and standard states

Years	Resting heart rates	Basal heart rates	Basal heart rates
	males and females	males	females
1	134	116	122

2	111	104	103
3	108	92	86
4	108	100	91
5	103	91	90
6	98	92	88
7	93	89	82
8	94	90	89
9	89	85	92
10	91	75	86
11	87	82	92
12	89	80	85
13	88	81	84
14	87	79	80
15	82	74	80

(SOURCE: Rowland T.W. Developmental Exercise Physiology. Human Kinetics, Champangn IL, USA, 1996 page 122)

Rowland (1996) suggested that the fall in basal and resting heart rate may have reflected heart reserve increasing progressively during the childhood years. The

endurance athletic performance. It also tends to parallel the decline in basal metabolic rate expressed relative to body size. An earlier study conducted by Malina and Roche (1983) demonstrated that the heart rate reserve in boys between the ages of 6 and 12 years increased 10% from 120 to 133 bpm.

A child's heart has the ability to pump more quickly than an adult's heart at any level of exercise. Children also have very high maximal heart rates, frequently over 200 beats per minute. Bailey et al. (1978) conducted an 8 years longitudinal study on the maximal heart rate in 51 boys during exercise in a running treadmill protocol. Mean maximal heart rate for the entire study was 196 bpm. At first year, the average peak rate of children was 193 bpm. Mean maximal rates on serial testing did not vary between years by more than 3 bpm. A study by Cumming et al. (1978), that used a treadmill protocol, for maximal testing revealed a mean maximal heart rate among 327 children of 200 bpm for both boys and girls.

Moreover, some studies have reported that maximal heart rates observed during cycle testing are usually less than those recorded with treadmill exercise. Boileau et al. (1977) reported that the mean maximal heart rate among 21 boys aged 11 to

14 years was 194 bpm on a walking treadmill test but only 186 bpm when the same boys performed exhaustive cycle exercise.

Maximal heart rate of children appears to decrease by approximately one beat per year as children age. Maximal heart rate may also be influenced by mode of exercise. The age-related decline in maximal heart rate is however, is independent of gender, fitness level or race.

## 2.3.3 Stroke Volume and Cardiac Output

The stroke volume of children is lower than that of adults due to smaller heart size and smaller blood volume in children. As children grow, resting stroke volume increases. The increases in the stroke volume are directly associated with increases in left ventricular size since myocardial contractility does not change, which in turn is directly related to BSA, lean body mass, or total body mass. At the same time, resting cardiac output rises. These changes in resting cardiac output parallel those of O<sub>2</sub> uptake, increasing in absolute value but declining relative to body mass. Since testing heart rate declines with age, Rowland (1996) indicates that increases in cardiac output at rest during childhood were accomplished

entirely by a rise in stroke volume. Despite age-related increases both submaximal and maximal cardiac output as well as stroke volume remain lower in children compared with adults. The higher maximal heart rate of children compared with adults does not fully compensate for the lower maximal stroke volume, and results in a lower cardiac output during exercise than adults. Maximal stroke volume during exercise increases in direct proportion to left ventricular and body size.

# 2.3.4 VO<sub>2</sub>max

An absolute rate (l.min<sup>-1</sup>) of  $VO_2$ max is much lower in children than in adults. Absolute  $VO_2$ max increases proportionately with growth until approximately 20 years. In children much of the growth-related change in  $VO_2$ max is related to changes in body composition. When adjusted for growth-related changes in body size (ml.kg.min<sup>-1</sup>), relative  $VO_2$ max is generally similar in children compared with young adults. Janz et al. (1996) studied an appropriate method to "normalize" oxygen uptake for body size in children and adolescents. The researchers measured the relationships between  $VO_2$ max, submaximal  $VO_2$ , body mass, height, body composition and sexual maturation in 126 children (mean age at baseline = 10.3yr) in a 5-yr longitudinal study. The results supported models using

fat – free body mass (FFM) was better for eliminating the effect of body size on  $VO_2$ . In boys a univariate model with a FFM exponent of 0.91 and in girls a univariate model with a FFM exponent of 0.87 satisfactorily normalized peak  $VO_2$ .

Anaerobic power is much lower in children compared with adults and increases throughout childhood and adolescence. The greatest changes in anaerobic power occur between the ages of 9 and 15 years. The difference in anaerobic power between children and adults persists even when values are adjusted for differences in body mass, muscle mass, or muscle size (Rowland, 1996).

Paul et al. (1977) studied the VO<sub>2</sub>max values by treadmill in 15 swimmers and 15 controls, which were 9 to 15 years old. The result indicated that after seven months of training the mean VO<sub>2</sub>max of the swimmers increased 8.11 ml/kg/min from 47.27 ml/kg/min to 55.38 ml/kg/min (15%). While that of the reference group increased 2.21 ml/kg/min from 46.81 ml/kg/min to 49.02 ml/kg/min (5%). This difference in increase between the two groups (5.90 ml/kg/min) was sufficient to be significant at the 0.01 level. That means that the training led to the increased in VO<sub>2</sub>max in children, as well as growth.

Again age related increases reflect increases in lean muscle mass during growth.

Most of the research has however been conducted on young males rather than

females. The relationship between anaerobic performance and age is not expected
to be as evident in young females who's increase in FFM is not as profound as
their male peers.

# Summary for the changes in Cardiac shape and function with age

In new born babies, the heart weight relative to body mass is high, as well as resting heart rate and basal heart rates is high. With growth, during childhood, the child's cardiac structure and function develop continually. These changes include:

- 1) The heart weight relative to body mass declines steadily.
- The left ventricular size and pressures increase and becomes thicker than the right side of the heart.
- 3) Both basal heart rates and resting heart rates decrease progressively. Maximal heart rate of children is higher than adult, and appears to decrease by approximately one beat per year as children age.

- 4) The stroke volume of a child being lower than that of an adult. However as children grow, resting stroke volume increase that directly associated with increases in left ventricular size. At the same time, resting cardiac output rises too.
- Both submaximal and maximal cardiac output as well as stroke volume remains lower in child compared with adults.
- 6) An absolute rate of VO<sub>2</sub>max is much lower in children than in adults, but increases proportionately with growth until approximately 20 years. However, relative VO<sub>2</sub>max is generally similar in children compared with adults.

# 2.4 Changes in Cardiac Structure and Function with Training in Adults

Exercise is the body's most common physiologic stress; it places major demands on the cardiopulmonary system. Maximal exercise can define the limits of performance in an individual. Virtually all body systems, including the cardiovascular, pulmonary, endocrine, neuromotor, and the thermoregulatory, are involved in the homeostatic adjustments required for acute exercise and in the chronic adaptations that occur with physical training. These adaptations allow the body to increase its metabolic rate up to 20 times the resting rate, during which cardiac output may increase as much as 6 times (Myers 1996). The magnitude of

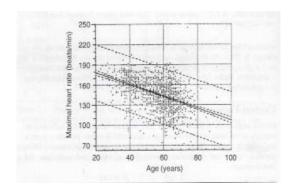
these adaptations is dependent upon an individual's age, sex, body size, fitness, and types of exercise. The finding of cardiovascular adaptations following intensive and long-term training in adults has been reported in numerous studies (Ikaheimo et al. 1979, Oakley. 1992, Potiron et al. 1989.). These adaptations include the changes of cardiac structure and function. It may be lower heart rate, left ventricular hypertrophy, higher blood pressure, and increase of stroke volume, cardiac output and maximal oxygen consumption during exercise.

#### 2.4.1 Heart rate

Sympathetic and parasympathetic nervous system influences underlie the cardiovascular system's first response to exercise, and increase in heart rate. Of the two major components of cardiac heart rate and stroke volume, heart rate is responsible for most of the increase in cardiac output during exercise, particularly at higher levels. Heart rate increases linearly with workload and oxygen uptake. The heart rate response to exercise is influenced by several factors, including age and type of activity due to endurance training reduces resting heart rate (Brooks et al. 1985).

Of these, perhaps the most important is age: children have higher maximum heart rates than adults, ranging from approximately 195 to 220 beats/min (Bale et al. 1992). And a decline in maximal heart rate occurs with increasing age (Jonathan 1996).

Diagram 2.8 the relationship between maximal heart rate and age.



(Source: Jonathan N. Myers. Essentials of cardiopulmonary exercise testing . 1996, U.S.A. Page 12.)

Maximal heart rate is unchanged or may be slightly reduced after a program of exercise training. At rest, heart rate will be lower in trained athletes than in untrained individuals due to changes in the heart's neural control. Stolt et al. (2000) recorded a resting 12-lead ECG in 60 elite endurance athletes (30 females aged  $24 \pm 4$ , 30 males aged  $25 \pm 3$ ) and 30 sedentary volunteers (15 females aged

26± 5, 15 males aged 26 ± 3). The result showed that the averaged resting heart rates in male and female athletes of 47 and 50 bpm, respectively and in the male and female control participants the resting heart rates averaged 68 and 66 bpm, respectively. A lower rest heart rate results in larger preload of heart at rest and subsequently enhances heart's reserve (Cameron et al. 1980). In contrast, maximal heart rate remains essentially unchanged by training (Brooks et al. 1985).

MacDougall (1987) reported that resting heart rates of body builders and weight lifters who do not do additional endurance training were not lower than those for an untrained population. MacDougall (1987) thus concluded that strength training does not result in the training bradycardia exhibited by endurance-trained athletes.

## 2.4.2 Type of training and cardiac structure and function

The effect of training on cardiovascular structures has been summarized as a "physiological" left ventricular hypertrophy that is due to growth in end-diastolic dimensions of the left ventricle, the left ventricular wall thickness, or both (MacFarlane et al. 1991, Maron. 1986, George et al. 1984). The blood volume increase that occurs in athletes has a direct effect on the cardiac pre-load which is a cause of growth in end-diastolic dimensions of the left ventricular (Adams.

1981, Bello et al. 1995). On the other hand, an increased afterload on the heart, i.e., increased resistance to pumping of blood, is a cause of growth in the left ventricular wall thickness (George et al. 1984). The "physiological" type of left ventricular hypertrophy (LVH) does not lead to an isocheim response during exercise, but results in left ventricular systolic and diastolic function improvement (Giorgi et al. 2000, Raskoff et al. 1976). Maximal stroke volume and cardiac output may also be modified by training (John et al. 1969). There is some evidence that after a short period of detraining, the size of the heart may show a reduction in elite athletes (Barry. 2002). This would indicate that the increase in size is a training adaptation.

Earlier studies have suggested that alterations in cardiac structure are mainly dependent on the type of training and or athletic activity. Athletes participating in dynamic-type endurance sports tend to develop mainly larger LV cavity dimensions, whereas athletes involved in static exertion and exposed to a pressure load are more likely to develop greater LV wall thickness without significant increase in cavity dimensions (Giorgi et al. 2000, MacFarlane et al. 1991, Morganroth et al. 1975).

Adams et al. (1981) assessed the effects of intense endurance training on cardiac structure and function in a group of healthy, college-age men (25 experimental and 11 control, mean age 22 years), by echocardiograph. The result indicated that intense endurance training results in a significant increase in resting left ventricular end-diastolic dimension and volume. This finding was supported by Stolt et al. (2000), who examined cardiac structures, using the echocardiography, in 30 male endurance athletes (aged  $25 \pm 3$ ) and 15 non-athletes (aged  $26 \pm 3$ ) at rest. As expected, the adult athletes group shown the greater left ventricular posterior wall thickness, left ventricular end-diastolic diameter and mass. At the same time, LV mass (g/BCA) and LV mass (g/height) were also significantly greater in athletes than in controls. None of the subjects showed any evidence of ventricular dysfunction. This study indicated that endurance sport not only develops left ventricular cavity dimensions, but also increases left ventricular wall thickness. In other words, the endurance-trained heart is, in a structural sense, an enlarged variant of normal. The following paragraphs outline the functional changes that have reported with endurance exercise training.

Bello (1995) studied the trend of left ventricular systolic and diastolic function and of VO<sub>2</sub>max during maximal exercise, using echocardiography and the

analysis of ventilator gas exchange in a group of elite male cyclists (mean age: 25.1). The researcher found the there was a linear regression relationship was found between VO<sub>2</sub>max and CO (r=0.84).

Stork et al. (1992) used 2-dimensional Doppler echocardiograph to evaluate left ventricular filling behavior in 25 endurance adult athletes (aged 31±9 years) of differently training levels at rest and during cycling. The results showed that the athletes with a higher training level had a more pronounced increase of left ventricular dimension, left ventricular mass and stoke volume index. This increase indicates a beneficial effect of additional training on LV systolic function. At the same time, all endurance-trained athletes showed a normal LV filling behavior at rest. However, arterial filling fraction was significantly higher and was more prolonged during exercise in less trained athletes than in top class athletes. Stork suggested that this paradox reflected an impairment of the energy-consuming process of early diastolic relaxation and postulated an imbalance of oxygen demand and supply in the myocytes. Stork et al.(1992) contended that the athletes with a higher training level have a more efficient LV filling behavior during exercise, possibly due to more refined relationship between myocardial oxygen demand and supply. The results of the study by Stork et al.(1992) indicates a more sophisticated cardiac structure and functional adaptation to physical stress and a more efficient diastolic performance during exercise in endurance athletes with a higher training level than their less trained peers.

Karjalainen et al. (1997) studied the relationship between left ventricular mass and exercise blood pressure in 32 male endurance athletes (aged from 22 to 31 year) and 15 age –matched controls. Left ventricular mass was evaluated using Doppler echocardiography. Participants also had their blood pressure measured during a maximal cycling exercise test. The authors found that left ventricular mass was not correlated with blood pressure at rest or in low-grade exercise. At peak exercise however, athletes had higher blood pressure than the sedentary control participants and there was a correlation between LV mass and systolic BP in endurance athletes. This may be the cause why endurance athletes display left ventricular mass thickness, as well as left ventricular chamber enlargement.

The aforementioned studies showed that athletes who embark on long-term endurance training generally exhibit increases in the size and stroke performance of the cardiac ventricles. These adaptations may cause increases in the amount of blood pumped with each heartbeat at rest and during exercise. At rest, cardiac

output does not change in endurance training athletes because heart rate decreases, in spite of a stroke volume increase. In contrast, maximal cardiac output may increase in endurance training athletes due to the large increase in stroke volume.

The increases in cardiac output will increase the supply of blood and oxygen to body during exercise.

Some studies have suggested that the cardiac adaptations to long-time exercise may differ among athletic groups. Ikaheimo (1979) studied the cardiac effects of training in 10 sprinters and 12 endurance runners using ECG, echocardiography and chest x-ray film. The results revealed that in both groups of athletes, the left ventricular end-diastolic volume, and the left ventricular wall thickness and mass were greater than normal. However, the left ventricular hypertrophy in the endurance runners was more apparent with the wall thickness and mass in the endurance runners higher than in the sprinters. The relative heart size presented by the chest X-ray examination in the endurance runners was also greater than in the sprinters. Ikaheimo (1979) concluded that endurance running training may cause left ventricular dilatation, left ventricular wall hypertrophy and improved systolic emptying of the left ventricle.

Elias et al. (1991) compared the differences of cardiac structure and function in adult weight trained athletes and endurance runners at rest using echocardiography. The results demonstrated that the endurance runners had significantly greater relative left ventricular end-diastolic dimension (LVEDd), left ventricular end-systolic dimension (LVEDs) and left ventricular diastolic posterior wall thickness (LVPW) than the weight trained athletes. From this study, it can be found that endurance athletes have—greater left ventricular wall thickness than strength athletes, although strength sports tend to develop greater LV wall thickness than in untrained people.

John and Allen (1999) also used ECG to report marked left ventricular hypertrophy in three college athletes compared with normative population. Two of these athletes were from a football background and one was from a basketball background, so no conclusions about the specificity of cardiovascular adaptations were possible from this anecdotal data.

Smith et al. (1994) examined myocardial adaptation and weight fluctuation of seven college wrestlers (above 18 years of age) during a competitive season. The results suggested that significant increases in myocardial mass occurred over the

course of a competitive wrestling season. Smith et al (1994) attributed most of the myocardial mass increase to increased left ventricular wall thickness. Du et al. (1987) reported that in sprint and strength athletes (aged 18-28 years) only the left ventricular mass and wall thickness indices were slightly higher than in the control group.

In general, it appears that significant cardiac adaptations can be found in adults from specific sports training backgrounds. These adaptations may differ among athletic groups as well as being different from less active normative populations.

# 2.4.3 ECG

Research using the 12-lead ECG presents a broad range of abnormal patterns in trained athletes. These variations include bradycardia, particularly increased QRS voltages, which are suggestive of left ventricular hypertrophy and repolarization abnormalities. These alterations have been attributed to the physiological cardiac adaptations that occur as a consequence of systematic physical training.

Some research has shown that left ventricular hypertrophy in the ECG was more apparent in the endurance runners than in non-athletes. Pelliccia et al. (2000) compared ECG patterns with cardiac morphology (as assessed by echocardiography) in 1005 consecutive athletes (aged  $24 \pm 6$  years, 75% male) who were participating in 38 sporting disciplines. In this study most athletes (60%) had a normal ECG or showed only minor alterations. A variety of abnormal ECG patterns occurred in 40% of the athletes. The criteria for abnormal ECG included the following: (1) striking increase in R or S wave voltage (≥35 mm)in any lead, (2) Q wave  $\geq$  4mm in depth and present in  $\geq$  2leads; (3) repolarization pattern with inverted T wave > 2mm in  $\ge 2$  lead, (4) left bundle branch block, (5) marked left ( $\leq$  -30°) or right ( $\geq$  110°) QRS axis deviation. A cardiovascular abnormality was identified clinically and/or by echocardiography in 53 athletes (5%). These abnormalities were mitral valve prolapse with mild regurgitation, a bicuspid aortic valve with regurgitation; atrial or ventricular septal defect, dilated cardiomyopathy, mild pulmonary artery stenosis, and myocarditis. Echocardiography analysis showed that LV end-diastolic cavity dimension, maximum wall thickness, mass index, and the left atrial dimension were each greater in athletes with distinctly abnormal ECGs than in those with normal ECGs, it might show that larger cardiac dimensions were associated with

abnormal ECG patterns. Abnormal ECGs were most frequently encountered in endurance athletes, male sex, and younger age (< 20 years of age).

# 2.4.4 VO<sub>2</sub>max

Yamazaki et al. (2000) made the echocardiography examination and an incremental treadmill exercise test for measuring peak oxygen uptake (VO<sub>2</sub>max) on 11 sedentary normal-weight students (mean age 21 years), 17 sedentary overweight students (mean age 20 years) and 215 athletic students (mean age 20 years). Results were that in sedentary students, absolute VO<sub>2</sub>max in the overweight students was slightly higher than that in normal-weight students (3,024 vs. 2,912 ml/min). Relative peak VO<sub>2</sub> (ml/min/kg) was highly negatively correlated with body mass index (kg/m2) in a total of 28 sedentary students. The correlation between absolute peak VO<sub>2</sub> and left ventricular dimension was weak in the sedentary overweight students. However, a correlation coefficient of 0.55 was obtained in athletic students. A stepwise multiple regression showed significant determinants of absolute peak VO<sub>2</sub> in athletic students for body surface area (45%), left ventricular dimension (7%), and certain sports (6%). In contrast to sedentary people, a tendency for LVDd or LV mass to rise with increases in both BSA and aerobic capacity was demonstrated in athletes.

Abernethy et al. (1996) indicated that after 6 weeks training of endurance, athletes would get a 20-40 per cent increase in VO<sub>2</sub>max due to changes in both the cardiovascular system and skeletal muscle cells. Cardiovascular adaptable changes may enhance oxygen delivery to skeletal muscle and the muscle's ability to extract and utilize oxygen to produce ATP during exercise. The extent to which VO<sub>2</sub>max improves after endurance training depends on initial fitness and previous training, age, genetics and the type of training program. Moreover, some weight-training programs have also resulted in small increases in VO<sub>2</sub>max. Gravelle et al. (2000) compared the change of VO<sub>2</sub>max in 19 active women after 11 weeks of strength training. Gravelle et al (2000) examined maximal oxygen uptake during a continuous, incremental test to exhaustion on a rowing ergometer. The results demonstrated that maximum oxygen uptake increased from pre- to post- strength training. The authors suggested that the increase in total oxygen consumption may have been due to increase in total muscle mass among the weight trainers. Specificity in endurance training however, may elicit more significant VO<sub>2</sub>max improvements.

# Summary of Changes in Cardiac Structure and Function with Training in Adults

Exercise is a physiologic stress which places major demands on the cardiopulmonary system. As a result, cardiovascular adaptations can occur following intensive and long-term training in adults. At rest heart rate will be lower in trained athletes than in untrained individuals due to changes in the heart's neural control. Cardiac output is maintained by a favorable increase in stroke volume. The changes of cardiovascular structures on trained adult may include "physiological" left ventricular hypertrophy that is due to increases in end-diostolic dimensions of the left ventricular, the left ventricular wall thickness, or both. The "physiological" type of left ventricular hypertrophy results in cardiac function improvement. Athletes participating in dynamic-type endurance sports tend to develop mainly larger left ventricular cavity dimensions associated with the blood volume increases that have direct effect on cardiac preload. On the other hand, strength trained athletes develop mainly larger left ventricular wall thickness due to an increase of pressure load. More recent studies indicated that the endurance sport not only develop increased left ventricular cavity dimensions,

but also increase left ventricular wall thickness which even be greater than strength athletes. At peak exercise, endurance athletes had higher blood pressure than the sedentary control. This may be the cause why endurance athletes display left ventricular mass thickness. The Cardiovascular adjustments were related to an increase of stroke volume, cardiac output and maximal oxygen consumption during exercise. Changes to the ECG were most frequently encountered in endurance athletes. These variations include bradycardia, particularly increased QRS voltayes, which are suggestive of left ventricular hypertrophy and repolarization abnormalities. Both of training of endurance and strength may resulted in increases in VO<sub>2</sub>max due to change in the cardiovascular system and skeletal muscle cells.

2.5 Changes in Cardiac Structure and Function with Training in Younger Population.

## 2.5.1 Cardiac structure and function

The adolescent growth period has been called a critical time for the development of maximal aerobic power. Adolescence provides the first opportunity for large

increases in physical capacity (George, 1985). Some recent research has shown that the cardiac structure and function of children may occur adaptation changes to exercise training as well as adults, although these changes might be quantitative differences in the absolute values during rest and exercise between children and adults.

Obert et al. (1998) used echocardiography to examine the cardiac structure and function of nine children (10-11 years old), who had taken part in long-term intensive endurance swimming training for at least 2 years. The results demonstrated that after intensive endurance training the cardiac function of the children resulted in adaptations that include increased stroke volume and enlarged left ventricular internal dimensions.

Rowland et al. (1998) studied stroke index (stroke volume relative to body surface area) and cardiac index (cardiac output in relation to body surface area) of eight trained male distance runners (mean age 11.5 years) and 14 untrained control males (mean age 10.9 years) during a progressive cycle exercise test to exhaustion. The results revealed that the maximal stroke index and cardiac index were greater in the trained runners than the control group during exercise, No

differences were observed between the groups at rest. The authors (1998) noted that in contrast to their findings on the child athletes, adult endurance athletes demonstrated a greater stroke volume than non-athletes at rest as well as during exercise. The above findings using echocardiography have suggested that there might be quantitative differences in the absolute values measured in some of the cardiac responses during rest and exercise between children and adults.

Additional comparative quantitative differences in the cardiac responses between athletes following specific types of exercise training are apparent in younger populations. Ricci et al. (1982) use echocardiography to compare the differences of left ventricular dimensions between three groups of adolescent male athletes (aged from 16 to 18 years),(endurance, sprint and strength). The result showed that after 8-20 weeks of specific training, the left ventricular mass of the endurance training group had significantly increased, (10%) compared with the strength group (4%). There were not increased in the sprint trained athletes. To date, comparative cardiac structure investigations in adolescent athletes have not extended beyond 20 weeks.

Some studies found that there is the higher prevalence of abnormal ECG in athletes < 20 years of age. The prevalence suggests that intensive training may be more likely to alter the ECG pattern when associated with body growth and maturation during adolescence. Sanjay et al. (1999) evaluated the resting ECG profile in 1000 junior elite athletes (18 years or under). The results revealed several observations: (1). ECG functions in junior elite athletes were similar to those in senior athletes. (2). Sinus bradycardia and sinus arrhythmia in the athletes were more common than for non-athletes. (3). Voltage criterion to determine the presence of left ventricular hypertrophy was present. (4). The athletes when compared with normative data had a prolonged PR interval, QRS and QT duration. (5). The ST segment elevation was more common in athletes than non-athletes. (6). Associated abnormalities that indicate pathological hypertrophy were absent in this sample of young athletes.

#### 2.5.2 Maximal oxygen uptake

It has been debated whether pre-pubescent children exhibit adaptations to exercise training beyond those expected from growth alone. Many recent studies

( Rowland el at, 1998; Unnithan et al, 1996 ) have shown improvements in

VO<sub>2</sub>max and endurance exercise performance in children when training was based on principles recommended to improve fitness in adults. These studies indicate that children show the same relative improvement as adults in aerobic power, endurance performance and cardiovascular system variables with appropriately prescribed exercise training. In children, VO<sub>2</sub>max may increase by 5-25 per cent with endurance training. Rowland et al. (1998) reported that child endurance athletes demonstrated a VO<sub>2</sub>max that was approximately 30% greater than their sedentary peers. Thus VO<sub>2</sub> peak is a consistent marker of cardiac adaptations to endurance training in young populations.

Cardiorespiratory fitness can be assessed by direct measurement of maximal oxygen uptake (VO<sub>2</sub>max). Studies in younger athletes usually refer to this measurement as VO<sub>2</sub> peak due to the frequent absence of a plateau in oxygen uptake (<0.1 l.min<sup>-1</sup>) with increasing workload towards the completion of the test. Unnithan et al. (1996) researched the difference in the results of the VO<sub>2</sub> peak tests between 15 endurance-trained males (aged 11yrs) and 18 non-trained males (age 11yrs). The results indicated that the mean VO<sub>2</sub> peak value for the endurance-trained group was higher than the control group (P<0.05). Rowland et al. (1997) studied cardiac responses using a maximal cycling test in 15 males

(mean age 10.9 years) and 16 adults. The result indicated there was a lower Q/VO<sub>2</sub> ratio during exercise in children compared with adults. In general there is an age-related increase in VO<sub>2</sub> peak with age in absolute terms (liters per minute) and relative terms (ml.kg<sup>-1</sup>min<sup>-1</sup>) in childhood. During adolescence, relative VO<sub>2</sub> peak plateaus in males and decreases in females. These sex-related changes in VO<sub>2</sub> peak performances are likely to at least partially reflect the adjustments in body composition during puberty.

# Summary of Changes in Cardiac Structure and Function with Training in Younger athletes

Early studies suggested that adolescent athletes, although improving their endurance performances with training, may not increase their maximal aerobic power to the same extent as do adults. There are however not many studies about specific cardiac adaptations during exercise in child and adolescent athletes, more recent research has shown that when the training dose is sufficient, cardiac responses to sustained steady-state exercise in children are similar to those observed in adults. These adaptive changes include lower resting heart rates, increased blood volume, and enlarged left ventricular internal dimensions, increase stroke volume and cardiac

output, faster heart rate recovery. The maximal stroke index and cardiac index were greater in the trained younger athletes than the control group during exercise. There is the higher prevalence of abnormal ECG in younger athletes. These abnormal ECG were similar to those in senior athletes. In children, VO<sub>2</sub>max may increase by 5-25 percent with endurance training. These changes will differ in children, depending on the intensity, duration and frequency of training, as well as genetic factors.

#### **CHAPTER 3**

#### **METHODS**

#### 3.1 Purpose

The purpose of the methods is to outline a) participant recruitment, b) testing procedures and c) statistical analyses.

#### 3.2 Participant recruitment

This study examined the cardiac structure and function of 13 endurance-trained adolescents. Another 7 non-active adolescents took part as a control group.

In this study twenty, 14 to 16 year old adolescent males, volunteered as participants for all testing of the study (Appendix3.1), after also gaining permission from their parents.

Participants were derived from two groups.

#### 3.2.1 Endurance training group

Endurance-trained adolescents were from an elite swimming squad, and athletics club. The participants had participated in endurance training at least three times per week for the past year.

For endurance-trained adolescents, each training session was taken for 1-2 hours. Work intensity was self-rated using a scale of 1 to 5: 1=light training that does not result in sweat and making you feel tired. 2=training that results in little sweat and feeling a little tired. Recovery happens in a few minutes. 3=training that results in much sweat, a heart rate increase (140-150 beat/min) and feeling obviously tired. Recovery takes a few minutes to fifteen minutes. 4=training that results in much sweat, a high heart rate increase (above 150 beat/min) and feeling more tired. Recovery takes more than fifteen minutes. 5=Hard work sessions are those that really make your heart rate increase (above 180 beat/min), sweat a lot and feel very tired. In this study, most of trained adolescents had work intensity above a scale of 3.

#### 3.2.2 Control group

The control group was obtained from peers of the endurance trained groups or some high schools in Melbourne. The volunteers did not belong to any athletics club and had not participated in intensive (above scale of 1 to 3) exercise training more than once per week for the past year.

The study was approved by the Human Research Ethics Committee of Victoria

University of Technology (Appendix 3.2), and written informed consent was obtained
from the participants and their parents (Appendix 3.3). All participants completed a

'Risk Factor Assessment Questionnaire' and filled the section entitled 'Family

Medical History' before the testing started (Appendix 3.4).

All participants were free of injuries at the time of testing and for three months prior to testing and absence of long-term use of medications that may affect exercise performance, e.g. beta blockers.

The participants were all informed that they could withdrawal from the study at any time without prejudice.

#### 3.3 The testing procedures for this project

This project requires participants to be tested at two different locations. Testing occurred at the Footscray Park Campuses of Victoria University as well as within the Department of Cardiology at the Royal Children's Hospital. All testing was completed in air-conditioned environments of the laboratory (22°C; not more than 50% relative humidity).

An overview of the procedures is as follows: the boys gave informed consent and documented their recent exercise training status, the boys attended Victoria University on two or three occasions and Royal Children's Hospital once. The testing consisted of anthropometric measurements and peak oxygen consumption test at Victoria University on the first day and repeated sprint test and skeletal muscle strength test at Victoria University on the second day, with three days in between. Six days after the Victoria University testing, the boys attended the Royal Children's Hospital for echocardiography.

#### 3.3.1 Testing at Footscray Park Campus of Victoria University

Following a familiarization session, the participants visited the Footscray Park

Campus of Victoria University for several tests on one or two days within

approximately one week. The testing involved (1) Anthropometrical

measurements, in the form of a sum of 5 skin fold sites, height and body mass. (2)

Peak oxygen consumption test (VO<sub>2</sub>max). (3) ECG recording at rest and during

exercise and recovery. (4) Repeated sprint test on a cycle ergometer. (5) Skeletal

muscle strength test.

On the testing day, the participants were required to avoid any vigorous exercise, and were requested to travel to the testing session by car. They were advised to eat a light meal 2-3 hours prior to the test, or as directed and to avoid coffee, tea, alcohol and non-prescription drugs for three hours prior to the test. During the exercise testing each participant was requested to wear running shoes and shorts.

The testing procedures were fully explained to the participants before testing.

At least half an hour rest between each test was given to provide participants with full recovery in order to avoid muscular fatigue and at the same time providing the maximal power potential.

1) Anthropometrics measurements: All participants were measured for height, weight and 5 site skin folds before other exercise testing. For assessing height, the participants were asked to stand on a flat surface against the wall, with no shoes on. A measuring tape attached to the wall (approximately 2 meters off the ground) could be brought down to the individual's top of head and a measurement to the nearest millimeter was recorded. Body mass was measured with portable digital scales. Body surface area was calculated from the height and weight of the participant by Body Composition Analysis program (I.H.Fairweather F.I.T. 1990., America).

Skin-folds measurements were assessed with 'Harpenden skin-fold clipers.'

The skin-fold sites included the triceps, biceps, subscapular, suprailiac and mid-abdominal. All measurements are made on the right side of the body.

Each of the five sites was measured twice. If the first two measurements were more than 1 mm apart a third measurement was taken. Body fat percentage was estimated using sum of four skin folds method on Body Composition

Analysis program (I.H.Fairweather F.I.T. 1990., America).

2) Peak oxygen consumption test (VO<sub>2</sub>max): Peak oxygen consumption tests are an established and accepted laboratory practice for determining maximal aerobic power in athletes of almost all ages. In this part of the laboratory testing, the participants were required to complete a VO<sub>2</sub> peak test on a cycle ergometer. The seat height on the bike was adjusted for each individual. Each participant was allowed for full leg extension and secured to the pedals using toe clips and straps. Before testing there was warm-up exercise around 3 minutes. Expired air was directed through a low-resistance valve (Hans Rudolph 2-way non-rebreathing, Hans Rudolph Inc., Kansas City, MO, USA) and air was analyzed for oxygen (Model S-3A/11, Anetek, Pittsburg, PA., USA) and carbon dioxide (Model CD-3A, Ametek, Pittsburg, PA., USA). The oxygen and carbon dioxide analyzers were calibrated just prior, and immediately following each exercise test, using standard gases of known concentrations ( $\pm 0.15\%$  and  $\pm 0.1\%$  precision for O<sub>2</sub> and CO<sub>2</sub>, respectively) and the ventilometer was calibrated using a three liter syringe (Hans Rudolph Inc., Kansas City, MO, USA). Oxygen consumption (VO<sub>2</sub>,STPD) was calculated for each 15s, and reported for the last 15s of each 60s period using standard equations (Consolazio, Johnson & Pecora, 1963) and Turbofit software (Vacumed, Ventura, CA, USA). The measurement of ventilation

during exercise requires that the participant's nose and mouth be sealed tightly with a clip and a mouthpiece. Participants performed continuous, incremental exercise to fatigue in the upright position. The initial workload on the cycle ergometer was 25 W with subsequent 25 W increments imposed each minute until the volunteers had reached volitional fatigue. The participant might request to stop exercise at any time before the test and in this case, the test was terminated. After the aerobic fitness test the participants felt fatigued, but recovered within several minutes. During recovery, the participants were monitored for signs and symptoms of vaso-vagal attack or other signs of exhaustion, and prevention / management measures were in place. There were no cases of adverse events requiring follow-up during the study.

3) ECG recordings: ECG was recorded during each exercise test in the project; these comprised VO<sub>2</sub>max, repeated cycle ergometer sprint tests. During each VO<sub>2</sub>max exercise test, perceived exertion (Borg Rating of Perceived Exertion 6-20 point scale, RPE; Borg, 1982) and heart rate (HR) using an telemetry electrocardiograph Model X-Scribe Stress Test System, (Mortara Instrument Inc., Milwaukee, WI, USA) were measured at the end of each minute. Cardiac

rhythm was monitored continuously. The ECG was also monitored and HR measured during all repeated sprint testing on a cycle ergometer and during skeletal muscle strength testing. Recovery heart rates (at end of each minute for 5 minutes) and the ECG were also monitored after each exercise test. To record the ECG, 10 disposable electrodes suitable for exercise testing (X-SCRIBE<sup>TM</sup>; Mortara, Milwaukee, USA) were placed on the participant's chest together with a transmitter device worn on the waist. Before exercise testing, each participant sat on a chair for at least 15 minutes before the resting ECG was recorded.

4) Anaerobic test: Anaerobic tests measure the power produced during a brief exercise test. This test was performed on a modified air-braked Repco Cycle ergometer (Series A, Repco, Melbourne, Australia). The protocol comprised of 5 bouts of 6 seconds of maximal sprint cycling with 24 seconds for recovery between each. Each participant was secured to the pedals using toe clips and straps and requested to stand up for each sprint. The participants were instructed to pedal as fast as possible and attempted to maintain maximum pedal speed throughout the 6 seconds of each bout. Peak power and mean power of each bout were measured from power output obtained during

the test. Peak power was the highest output during any one second interval of the anaerobic power test on a wind braked Repco front loaded cycle ergo meter, whilst mean power was calculated from the total work performed during the 6 second test period. Anaerobic test usually was arranged on a deferent day to the VO<sub>2</sub>max tests in order to provide participants with full recovery from muscular fatigue.

High intensity cycling test also resulted in the participants feeling very fatigued. Again, we minimized these effects through proper cool down procedures and offering water as well as careful monitoring of heart rate using the ECG.

5) Muscular strength: Muscle strength of two legs was tested using dynamic muscular contractions through a device that measures the force applied during a kicking action. A Cybex Isokinetic dynamometer measured each leg strength of a participant while a participant sits on a seat and undergoes knee extension/flexion against a relatively heavy resistance (ie at a constant angular velocity of 60 degrees per second through a range of approximately 90 degrees). Leg power was measured by asking the participant to undergo

knee extension/flexion five times against a lighter resistance (180 degrees per second).

In this test, the resistance was proportional to the dynamic tension produced by leg muscle. The axis of rotation of the Cybex machine was aligned with the anatomical axis of rotation in the knee joint. Each participant was seated on an adjustable chair, the thigh was stabilized with a velcro strap, and the lever arm was attached just proximal to the participant's malleolus. As the participant exerted muscular force throughout the 90 degrees arc motion, the produced force was registered as torque during the movement on the Cybex Dual-Channel Multi Strip chart Recorder.

The participant were motivated to perform kick-up and kick-down activities (knee extension/flexion) for maximal efforts against a resistance, throughout the range of muscular contraction. The rest period between trials was three minutes. The highest value of a minimum of the three trials was taken as a measure of each participant's maximum leg strength. For each participant the highest peak torque was measured as the maximal output.

# 3.3.2 Echocardiographic testing at the Department of Cardiology at the Royal Children's Hospital

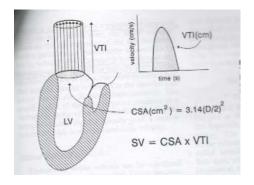
Echocardiography assessments was used on the participants to estimate cardiac structure and function. During this procedure a participant was asked to lie on a bed for a maximum of 60 minutes, with the left lateral decubitus position. A 3.5 MHz transducer was placed in the fourth or fifth intercostal space adjacent to the left sternal border. This procedure is low risk in that it involves the harmless emission of ultrasound waves. Standard Doppler Echocardiographic techniques was used to estimate the several dimensions of the cardiac structure that are not accessible using ECG technology. This procedure occurred only during rest.

Using the parasternal long axis view average 3 – M mode measurements of the interventricular septum (end diastole), left ventricular cavity size (end diastole and end systole) and left ventricular posterior wall (end diastole) were made.

Average 5 – M mode measurements if profound irregular heart rate (HR).

From the parasternal long axis view, left ventricular outflow tract (LVOT) diameter was measured by 2D imaging, using average 3 measurements within a millimeter of each other. The left ventricle ejects a volume of blood into the cylindrical aorta on each beat. The base of this cylinder is the systolic cross – sectional area of the aorta, while its height is the distance the average blood cell traveled during ejection for that beat. This distance is expressed as the velocity – time integral (VTI). From either the apical 5 chamber or apical 3 chamber view, LVOT VTI was measured using pulsed wave doppler, sample blood flow in the LVOT at the same the position as where the LVOT diameter was measured. VTI was taken to be the average of measurements from 3 consecutive heartbeats, except for irregular rhythms, where VTI was calculated from the average of five beats. VTI was also used in the calculation of stroke volume (SV) and cardiac output (CO).

Diagram 3.1. Doppler stroke volume calculation.



(source: Catherine M. O. et al., 1995. Textbook of Clinical Echocardiography.

98)

$$CO = SV * HR$$

Fractional shortening (%) of the LV was used assessment of LV systolic function.

Normal range = 
$$28 - 44\%$$
 (source – Henry, W.L. et al., Circulation62 (vol. 5):  $1054 - 1061$ , 1980).

#### **Summary of Procedures**

Testing in this project will occur at three different days (Appendix A). The testing is done over a three-week period with no more than three testing sessions involved in the study.

#### 3.4 Statistical analysis

Statistical analysis was performed using the statistical package SPSS.

Data was reported as means and standard errors of the mean (M±SEM). Difference in testing means values including height, weight, body composition, heart rate, VO<sub>2</sub> peak, anaerobic power, strength, power output and cardiac responses among two groups were analyzed through a series of one-way analysis of variance ANOVA. Post-hoc analysis tests were conducted where appropriate, to determine differences among the two groups. For all testing, the alpha level of (P<0.05) was accepted as significant.

#### **CHAPTER 4**

#### RESULTS

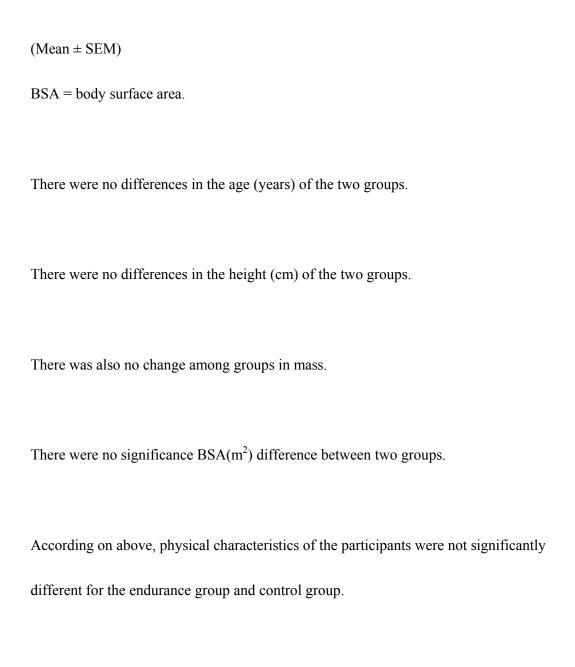
This chapter presents the results of the study, highlighting the differences between the endurance training and control groups through the exercise tests, echocardiography assessments and participant descriptive tests.

#### 4.1 Participant characteristics

Participants' characteristics are presented in Table 4.1. The average age at the participant were 15.15 years for control group, 15.33 years for the endurance training group.

TABLE 4.1 Subject Descriptive Characteristics.

	Endurance	Controls
Height (cm)	174.18±2.81	171.21±3.29
Weight (kg)	60.77±2.91	58.65±3.32
BSA (m <sup>2</sup> )	1.73±0.2	1.69±0.17



#### 4.2 Body composition

There were no significance differences in the body composition (fat %) and lean body mass of the two groups. However, the mean of body fat (%) was higher in control

group (11.80) than endurance, and the mean of lean body mass (%) was lower in control.

Table 4.2. present of total body fat, lean body mass.

	Total body fat (%)	Lean body mass (%)
Endurance	8.89±0.67	91.15±0.64
Control	11.80±1.65	88.20±1.65

 $(Mean \pm SEM)$ 

### 4.3 Peak oxygen consumption test

Table 4.3. Results for the peak oxygen consumption test.

	VO <sub>2</sub> (l/min <sup>-1</sup> )	$VO_2(l/min^{-1})$ $VO_2(ml/kg^{-1}.min^{-1})$ $VO_2(ml/kg^{-1}.min^{-1})$		Peak HR
	absolute	per kg body wgt	per kg *LBM	(beat/min)
Endurance	3.30±0.19	*54.41±1.79	*59.69±2.0	194.38±3.66
Control	2.70±0.19	*45.83±1.60	*52.19±2.40	188.71±5.50

 $(Mean \pm SEM)$ 

<sup>\*</sup> P<0.05

<sup>\*</sup>LBM = lean body mass.

The results of the peak oxygen consumption test are presented in table 4.3. There was no indication that in absolute peak  $VO_2$  values (l/min<sup>-1</sup>) there were any significant differences between the two groups. However there were significant (P< 0.05) differences of relative peak  $VO_2$  (ml/min/kg) and (ml/min/per kg LBM) between two groups. The endurance group had bigger relative peak oxygen consumption than the control group. Furthermore, there also were significant (P<0.05) differences of testing time between two groups. The mean testing time is longer in endurance group (12.85  $\pm$  0.69 minutes) than control group (10.43  $\pm$  0.75 minutes). There was a significant relationship (r= 0.51) between the length of test and peak oxygen uptake.

There wasn't any significant difference (P>0.05) of peak heart rate of  $VO_2$ max test between both groups.

#### 4.4 Sprint testing and Muscular strength

The results of sprint testing are summarized in table 4.4.1. On this study, There was no significant difference (P>0.05) of peak power of sprint testing test between both groups.

Table 4.4.1 peak power

	Peak power (Watts)
Endurance	$843.92 \pm 61.84$
Control	$750.43 \pm 62.08$

 $(M \pm SEM)$ 

As the result summarized in table 4.4.2, there was no significant difference (P>0.05) of muscular strength between both groups.

Table 4.4.2 muscular strength

	CFLR(Nms)	CFLL	CELR	CELL
Endurance	$54.84 \pm 5.97$	$63.47 \pm 5.46$	66.17 ±7.11	$73.69 \pm 7.43$
Control	$68.08 \pm 7.10$	$50.53 \pm 10.52$	$94.18 \pm 13.53$	$64.55 \pm 12.97$

 $(M \pm SEM)$ 

CFLR = concentric flexors of leg right

CFLL = concentric flexors of leg left

CELR = concentric extonsors of leg right

CELL = concentric extonsors of leg left

#### 4.5 Heart size and function

Table 4.5.1. Rest heart rate, blood pressure of the two groups

	Rest heart rate	Systolic blood pressure	Diastolic blood pressure
	(beat/min)	(mmHg)	(mmHg)
Endurance	$61.00 \pm 1.19$	$118.15 \pm 2.61$	$74.77 \pm 2.51$
Control	$60.00 \pm 3.50$	$110.57 \pm 1.62$	$66.29 \pm 1.66$

 $(Mean \pm SEM)$ 

There were no significant difference for both resting heart rates and systolic blood pressure between the two groups. The endurance group had significantly higher diastolic blood pressure than the control group (P<0.05).

Table 4.5.2. Cardiac dimensions: left ventricular dimensions during diastole and systole

	LVDd(mm)	LVDd/BSA(mm/m <sup>2</sup> )	LVDs(mm)	LVDs/BSA(mm/m <sup>2</sup> )
Endurance	$5.26 \pm 0.13$	$3.05 \pm 0.02$	$3.59 \pm 0.12$	$2.08 \pm 0.02$
Control	$5.38 \pm 0.20$	$3.12 \pm 0.02$	$3.40 \pm 0.23$	$1.96\pm0.02$

 $(Mean \pm SEM)$ 

LVDd = left ventricular end-diastolic dimension.

LVDs = left ventricular end-systolic dimension.

Table 4.5.3. Cardiac dimensions: posterior wall thickness and fractional shortening

	PW(mm)	PW/BSA(mm/m <sup>2</sup> )
Endurance	$0.80 \pm 0.02$	$0.47 \pm 0.02$
Control	$0.79 \pm 0.02$	$0.46 \pm 0.02$

 $(Mean \pm SEM)$ 

PW = left ventricular diastolic posterior wall thickness.

Table 4.5.4. Indices of cardiac function: stroke volume and cardiac output

	SV(ml)	SVindexed(l/min/m <sup>2</sup> )	CO(l/min)	CO indexed(l/min/m²)
Enduranc	ce $91.23 \pm 5.41$	$52.35 \pm 2.48$	$5.89 \pm 0.37$	$3.40 \pm 0.17$
Control	$92.42 \pm 3.95$	$54.72 \pm 2.30$	$5.51 \pm 0.19$	$3.25 \pm 0.18$

 $(M \pm SEM)$ 

There was no significant difference (P>0.05) of left ventricular end-diastolic, left ventricular end-systolic dimension and left ventricular diastolic posterior wall thickness between both groups.

There was no significant difference (P>0.05) of stroke volume, stroke volume indexed, cardiac output and cardiac output indexed at rest between both groups.

#### **CHAPTER 5**

#### DISCUSSION

This study investigated the effects of a long – term endurance program on resting and exercising cardiac responses, maximal aerobic power, and muscle power in male adolescents. This chapter is structured to describe the major findings of the present study and how these findings relate to previous studies of similar ages and training groups.

The results of this study confirm earlier reports that endurance-trained male adolescents had higher value of maximal oxygen uptake than untrained male adolescents (Naughton et al., 2000). The improvements in maximal oxygen uptake were associated with longer exercise time. It most likely be that endurance-trained adolescents have higher value of maximal oxygen uptake than untrained adolescents due to they had the ability to exercise at a high fraction of VO<sub>2</sub>max for prolonged periods.

Previous studies found a linear regression relationship between VO<sub>2</sub>max and maximal cardiac output (Bello et al., 1995). On the other hand, some investigators have reported that at rest stroke volume and cardiac output were similar for trained subjects and untrained (Rowland et al., 1998; John., 1969). This study is in agreement with that. In contrast, endurance-trained adolescents did not appear expected greater left ventricular internal dimension and left ventricular wall thickness than untrained.

From the results of this study, it may be suggested that the improvements of maximal oxygen uptake were not comparable with greater cardiac structure and function. Other organic physiological capacity, as well as mental capacity to resist the symptoms and physical manifestations of fatigue to a greater degree, might be related to the endurance ability.

#### 5.1 Subjects' Descriptive Characteristics.

In adolescence, increased age is with hormonal changes and substantial growth of the cardiorespiratory and musculoskeletal systems, so the aerobic and cardio improvements may be related to maturation, body mass, body composition, as well as

training. The velocity of physical growth during the adolescent years makes research with a group of young athletes particularly difficult.

In this study, both of groups appear a similar in age, height, weight and body surface area. The endurance group shown lower mean value in body fat (%) (8.89%) than control group (11.80%), even though a significant difference could not be demonstrated. Previous investigations indicated that the magnitude of the change in body composition appears to be a function of the training intensity (Christian 1989). Although body composition changes considerably during normal growth, particularly during adolescence, physical training further modifies body composition. Specifically, regular training results in an increase in lean body mass – primarily muscle tissue – and a decrease in fat tissue in children.

## 5.2 The Comparison of Maximal Aerobic Power ( $VO_2$ max) in Different Training Adolescent Males.

The ability to perform aerobic exercise is influenced by the individual's maximal oxygen uptake, which is widely recognized as one of the most important indexes of aerobic fitness (Pettersen, 2001). A number of studies have demonstrated great

percentage improvements in maximal oxygen uptake (VO<sub>2</sub>max) in trained adults (Abernethy et al., 1996; Gravelle et al., 2000 and Yamazaki et al., 2000), due to changes in both the cardiovascular system and skeletal muscle cells. Several previous investigators (Unnithan et al., 1996; Rowland et al., 1998 and Obert et al., 2000) had also provided the evidence that indicated prepubertal endurance-athletes would get a greater VO<sub>2</sub>max than untrained prepubertal children. Some researcher reported an average training improvement in VO<sub>2</sub>max in approximately 10% in adolescents (Naughton et al., 2000). The present study show there was a greater relative VO<sub>2</sub>max (ml/kg/min) in endurance-trained male adolescents than untrained male adolescents, but no significant different of absolute VO<sub>2</sub>max (l/min) in endurance-trained boys and untrained boys. Improvements in maximal aerobic power from endurance training are contingent on adequate intensity, duration, and frequency of exercise (continuous activity of large muscle groups, three to five sessions a week for 20 to minutes, at an intensity eliciting a heart rate 60 to 90% of maximum) (Rowland. 1992). However, a previous study found that there was no change in VO<sub>2</sub>max (ml/kg/min) in growing boys over 22 months of running training (Daniels et al., 1971). Some study suggested that studies in which children failed to improve aerobic power quantitatively or qualitatively compared to adults might not have provided sufficient exercise intensity (Rowland, 1992). A previous study (Cameron et al., 1980) shown that training during

the adolescent growth spurt failed to accelerate the development of  $VO_2$ max beyond the level expected from growth alone.

Previous investigators have speculated that changes in body composition during puberty might result in different associations between body size and VO<sub>2</sub>max. VO<sub>2</sub>peak does not increase in proportion to body mass in children and adolescents during exercise, rather that VO<sub>2</sub>peak is inversely related to body mass (Pettersen. 2001). During endurance performance, the weight load added by excessive body mass (i.e., body fat) would negatively influence endurance fitness (Janz et al., 1996). Previous study indicated that the mean value of VO<sub>2</sub>max exhibited significant negative relationship with body fat percent (r= -0.55) (Al-Hazzaa et al., 1994). The use of fat-free mass (FFM) to normalize VO<sub>2</sub> is more appropriate for research questions examining physiologic changes during growth and maturation rather than questions examining endurance performance in weight-bearing activities. The results of the present study confirm the greater VO<sub>2</sub> of FFM (ml/min/per kg lean body mass) occurs in trained male adolescents.

In particular, VO<sub>2</sub>max is directly related to maximal cardiac output, arterial-venous oxygen saturation, the pulmonary capacity of oxygen transport, the oxygen muscular

extraction, and an activated aerobic metabolism. The increase in peak VO<sub>2</sub> with training results primarily from an increase in maximal cardiac output, not from an increase in the arterio-venous (A-V) O<sub>2</sub> difference (Yamazaki et al., 2000). A previous study found a linear regression relationship between VO<sub>2</sub>max and maximal cardiac output(r=0.84) (Bello et al., 1995). In this study, the maximal heart rate during VO<sub>2</sub>max testing is similar in both of groups. The peak VO<sub>2</sub> values are may expected to be due to increase in maximal stroke volume.

Furthermore, recent work from our study showed that the length of the trial during VO<sub>2</sub>max was longer in endurance-trained children than untrained children and there was a stronger relationship between the length of test and peak oxygen uptake. Basing on the mean testing time, the max aerobic work is around 300W in endurance-trained children, while is around 250W in untrained children. Endurance capacity may be increased as a result of training by increasing the time for which an exercise task requiring a given VO<sub>2</sub> can be sustained or by increasing the work rate that can be sustained for a given time (Maughan. 1992). Successful endurance performance requires a high VO<sub>2</sub>max, but also the ability to exercise at a high fraction of VO<sub>2</sub>max for prolonged periods. On the other hand, differences in endurance ability between individuals may be related not only to their organic physiological capacity, but also to

mental capacity to resist the symptoms and physical manifestations of fatigue to a greater degree than those individuals with lesser endurance ability (Alan et al., 2001).

5.3 The Comparison of Muscle power and Muscle strength in Different Training Adolescent Males.

Increases in muscle mass are associated with the muscle group engaged in the training and are a function of training intensity. In the present study, the endurance-trained boys didn't show higher lean mass than the control group. Based on this finding, the lack of improvement in anaerobic work capacity and muscle strength after endurance training may be explained by the lack of increase in lean mass. Earlier studies also showed that the skeletal muscle adaptation in adolescent boys occurs in the sprint trained group only (Geraldine, 2000). Endurance training generally has no effect on the cross-sectional area of muscle fibers, although may result in the increased capillarization of trained muscle and the increases in the size of the Type 1 (slow twitch, highly oxidative) fibers that have been reported after a 6 – month endurance training regimen (Maughan, 1992). The increased capillarization of trained muscle, together with the absence of any change in muscle fiber size, will result in a decreased

diffusion distance and increased surface area for exchange between muscle and blood which may result in the improvement of the ability to perform aerobic exercise.

5.4 The Comparison of the Cardiac Function and Structure in Different Training Adolescent Males.

Cardiovascular adaptations related to endurance training are well known among male adult athletes. Endurance training may causes left ventricular enlargement due to higher blood volume and greater wall hypertrophy, as well as improved systolic emptying of the left ventricle (Ikaheimo et al., 1979; Giorgi et al., 2000). Many factors influencing left ventricular preload and afterload can affect left ventricular end-diastolic dimension, physical training could significantly increase left ventricular internal diameter by reductions in heart rate or by an increase in central blood volume (Ricci et al., 1982). The blood volume increase that occurs in athletes has a direct effect on the cardiac pre-load and on left ventricular chamber dilatation, so stroke volume can be modified by training. Some studies have demonstrated that improvements in maximal cardiac output result entirely from increased stroke volume, as little change occurs in maximal heart rate (Rowland, 1992).

In concordance with previous studies, endurance-trained adult athletes showed elevated values for LV chamber dimension, LV mass, LV mass index and stroke volume index when compared to normal subjects. The athletes with a higher training level showed an even more pronounced increase of LV mass, LV mass index and stroke volume index. The increased stroke volume index in top athletes when compared to controls indicates a beneficial effect of additional training on LV systolic function (Stork et al., 1992). Compared to lesser trained individuals, extremely trained athletes show more complete LV filling during exercise, possibly due to a better relation between myocardial oxygen demand and supply and reduced exercise induced myocardial hypoxia. Whether LVIDs and LVEDV are the critical factors that determine exercise SV remains to be seen.

Based on previous studies, most of cardiovascular adaptations related to endurance training appeared in adults. However, whether or not children improve their cardiac size and function with exercise is controversial. The problem is that, particularly in young children, it is difficult to isolate the effect of exercise on cardiac factors from the influence of growth and maturation and of heredity. In the present study, however, a significant difference could not be demonstrated in left ventricular end-diastolic dimension, ventricular end-systolic dimension and left ventricular posterior wall

thickness between endurance-trained male adolescents and untrained male adolescent. Previous studies found that if long-term training is a stimulus for cardiac hypertrophy, it is at best a mild one. As Bale (1992) comments, exercise in adolescents between 14 to 18 years produces improvements similar to those of adults. The degree of improvement again depends upon the level and type of exercise undertaken and the initial fitness of the children. Ricci et al., (1982) found that after 8 weeks endurance, there were not significant increases in left ventricular internal dimension and left ventricular wall thickness in adolescent boys (16 years old). Obert et al., (1998) used echocardiography to examine the cardiac structure and function of nine children (10-11 years old), who had taken part in long-term intensive endurance swimming training for at least 2 years. The results demonstrated that after two years intensive endurance training the cardiac function of the children resulted in adaptations that include increased stroke volume and enlarged left ventricular internal dimensions. Therefore, endurance training need enough stimulus, including the intensity, duration and frequency of training, as well as genetic factors for significant cardiac adaptation. Our subjects in training group might lack enough intensity, duration or frequency of training, so didn't shown greater left ventricular internal dimensions, wall thickness, peak heart rate and resting heart rate data. On the other hand, the endurance-trained adolescent males exhibited higher VO<sub>2</sub>max levels than untrained adolescent males,

but without greater left ventricular internal dimension and left ventricular wall thickness. This suggested that there are other factors, not only the cardiovascular system, which may be important in determining endurance capacity, would result in the increased VO<sub>2</sub>max. Apparently other extrinsic cardiac factors including increased muscle mass, increased oxidative capacity of skeletal muscle, increased muscle blood flow, and possibly increased total blood volume play a primary and more important role than cardiac factors in the development of maximal gas transport capacity in children (Cameron et al., 1980).

Furthermore, in children, it might be difficult to get more obvious cardiovascular improvement in trained children than untrained children, because most "untrained" children are also active, either through recreation or school sport or physical education. Bale (1992) comments that in most studies, the effects of exercise or training on children are only assessed in weeks or months, yet in adults the effects may be monitored over several years. Thus, the assumption that physical activity, exercise or training explains the differences between nonactive and athletic children in functional capacity over and above growth and maturation may not be a valid one.

It is true that athletes have a left ventricular wall thickness slightly larger than normals; however, it is not clear whether this is due to long-term training or to genetic factors. On the other hand, previous studies suggest that factors other than the type of training may be more important in determining the geometry of the left ventricle in adaptive hypertrophy. Here genetic predisposition may be significant (Stolt et al., 2000). Familial aggregation of LV dimensions has been reported and it has been concluded that > 60% of the variability in LV mass can by explained by heritable factors (Karjalainen et al., 1997).

Other investigators have reported that stroke volume and cardiac output were similar for trained subjects and untrained at rest (Rowland et al.,1998; John, 1969). The results of this study are in agreement with that. On the other hand, the work of John et al. (1969) showed that trained subjects demonstrated significantly higher maximal values for cardiac output and stroke volume during exercise than untrained subjects, with similar maximum heart rate.

Earlier studies suggest that exercise blood pressure (BP) is more strongly associated with LV mass in subjects with LV hypertrophy than in subjects with normal-sized hearts, static exercise increased BP by increasing stroke volume, with no change in

the systemic vascular resistance. Greater LV mass is associated with larger stroke volume (Karjalainen et al., 1997).

A previous study has indicated that no significant or only a slight negative relationship between resting blood pressure and physical fitness (Al-Hazzaa et al., 1994). In this study, there was no significant difference of systolic blood pressure between both groups, but there was significant difference of diastolic blood pressure. The finding of cardiovascular adaptations following intensive and long-term training in adults has been reported in numerous studies (Ikaheimo et al. 1979, Oakley, 1992, Potiron et al. 1989.). These adaptations include the changes of cardiac structure and function. It may be lower heart rate, left ventricular hypertrophy, higher blood pressure. Stolt et al.(2000)'s study indicated that endurance sport not only develops left ventricular cavity dimensions, but also increases left ventricular wall thickness and an increased afterload on the heart, i.e., increased resistance to pumping of blood, is a cause of growth in the left ventricular wall thickness (George et al. 1984). The increased peripheral resistance results from increases in sympathetic outflow to the arterioles in many vascular beds. The endurance group in this stusy had higher diastolic blood pressure. It might be due to endurance-trained male adolescents had higher sympathetic nervous activity than control group. Nervous system adaptations

following training might occur more early than cardiovascular adaptations. This needs other research.

### **CHAPTER 6**

### CONCLUSIONS AND RECOMMENDATIONS FOR FURTHER RESEARCH

### **6.1 Conclusions**

In conclusion, the endurance-trained male adolescents had higher maximal oxygen uptake during exercise testing than untrained adolescent males, even though the height, weight, body composition and body lean mass were similar on both of trained and untrained male adolescents. In addition, the endurance-trained adolescent males could sustain a longer exercise time than untrained male adolescents. There was a stronger relationship between the length of test and peak oxygen uptake. This suggests that endurance capacity may be increased as a result of training by increasing the time for which an exercise task requiring a given peak oxygen uptake can be sustained or by increasing the work rate that can be sustained for a given time.

On the other hand, this study found that the endurance-trained adolescent males did not exhibit greater stroke volumes, cardiac output at rest or cardiac dimensions than untrained male adolescents, as was hypothesized. This observation suggests that

endurance training stimulus may need to be of greater intensity, duration and frequency than was evident for this cohort. Alternatively or in addition, the control group may have been engaged in sufficient exercise as to blur any differences that otherwise may have occurred. A third possibility is that genetic factors may be more important in determining cardiovascular development and performance in adolescents, than was previously thought.

### **6.2 Recommendations For Further Research**

A limitation of this study is the fact that only male adolescents volunteers were studies, so care must be taken in extrapolating the findings to the whole population. The endurance-trained subjects need to be recruited from several different specific sport clubs in the further investigation. The long-distance runner might get enough stimuli, including the intensity, duration and frequency of training for significant cardiac adaptation.

Further study is needed to determine whether the superior aerobic capacity of the young endurance athlete is as a result of training or genetic predisposition or a combination of both.

Furthermore, whether functional performance of children in response to exercise adapts in a similar way to that of adults has not been completely resolved (Bale, 1992). The intensity, duration and frequency of exercise program are important factors which may result in cardiac adaptation. It could not be said that the untrained children in this study did no exercise, even though they did not participate in athletic training. It is likely that the difference in intensity of exercise between trained children and untrained children might be smaller than between trained and untrained adults. These need to be further researched. On the other hand, if there is a difference of cardiovascular adaptation between endurance-trained adolescents and untrained adolescents, it might be mild one. In this study, the limitations of having small number of control group might affect the findings as well. Therefore, it will need larger sample size for showing the significant difference.

The cardiac function at rest could not reflect the cardiac reserve capability.

Comparing the difference of cardiac function and structure between trained and untrained adolescents at high workloads should therefore expected provide valuable insights whether there is the improvements of cardiac reserve in trained adolescents.

### **REFERENCES**

Adams, T. D., Yanowitz, F. G., Fisher, A. G., Ridges, J. D., Lovell, K., & Pryor, T.A. (1981). Noninvasive evaluation of exercise training in college-age men. <u>Circulation</u>. 64(5), 958-965.

Alan, S. C. G., Michael, I. L., & Timothy, D. N. (2001). Neural Control of Force

Output During Maximal and Submaximal Exercise. Sports Medicine, 31(9), 637-650.

Al-Hazzaa, H. M., Sulaiman, M. A., Al-Matar, A. J., & Al-Mobaireek, K. F. (1994).

Cardiorespiratory Fitness, Physical Activity Patterns and Coronary Risk Factors in

Preadolescent Boys. International Journal of Sports Medicine, 15, 267-272.

Anguilla, A., Macchia, C., Magliari, F., Bacca, F., & Lusiani, L.(1986).

Echocardiographic study of training induced heart modifications in a group of sport beginners. Medicina dello Sport, 39(6), 539-545.

Arts, F. J. P., & Kuipers, H. (1994). The Relation Between Power Output, Oxygen

Uptake and Heart Rate in Male Athletes. <u>International Journal of Sports Medicine</u>, <u>15</u>,

228-231.

Bailey, D. A., Ross, W. D., Mirwald, R. L., & Weese, C. (1978). Size dissociation of maximal aerobic power during growth in boys. <u>Med. Sport.</u> 11, 140-151.

Bale, P. (1992). The Functional Performance of Children in Relation to Growth,

Maturation and Exercise. Sports Medicine, 13(3), 151-159.

Barry, J. M., (2002). Hypertrophic Cardiomyopathy - Practical Steps for Preventing Sudden Death. <u>The Physician and Sportsmedicine</u>, 30(1), 19-24.

Bello, V. D., Talarico, L., Muro, C. D., Santoro, G., Bertini, A., Caputo, M. T., Bianchi, M., Cecchini, L., & Giusti, C. (1995). <u>International Journal of Sports</u>

<u>Medicine</u>, 16, 498-506.

Brooks, G. A., & Fahey, T. D.(1985). Exercise Physiology. Macmillan, New York.

Cantwell, J. D., (1996). ECG Quiz: Can This College Athlete Compete? <u>The Physician and Sportsmedicine</u>, 24(4).

Cantwell, J. D., & Dollar, A. L. (1999). ECG Variations in College Athletes. <u>The</u>
Physician and Sportsmedicine. 27(9), 68-74.

Carre, F., & Chignon, J. C. (1991). Advantages of Electrocardiographic Monitoring in Top Level Athletes. International Journal of Sports Medicine, 12, 236-240.

Cameron, J. R. B., Cunningham, D. A., & Nichol, P. M. (1980).Gas transport capacity and echocardiographically determined cardiac size in children. <a href="https://doi.org/10.1001/jhear.2007/">The American</a>
<a href="https://doi.org/10.1001/jhear.2007/">Physiological Society, 994-999.</a>

Christian, W. Z., Michael, G. M., & Jan, Melichna. (1989). Physiological Considerations in Training Young Athletes. <u>Sports Medicine</u>. <u>8</u>(1),15-31.

Cumming, G. R., Everatt, D., & Hastman, L. (1978). Bruce treadmill test in children: normal values in a clinic population. <u>Am.J. Cardiol.</u> 41, 69-75.

Daniels, J., & Oldridge, N. (1971). Changes in Oxygen Consumption of Young Boys

During growth and running training. Medicine and Science in Sports, 3, 161-165.

Du, H. (1990). A preliminary study on changes of cardiac performance in different categories of elite athletes during static exercise. Chinese Journal of Sports Science (Beijing), 10(1), 46-51.

Du, H., Lin, F., & Zhang, Z. (1987). The comparing research of left cardiac structure and function with top-ranking endurance, strength and sprint-trained athletes and untrained men. Chinese Journal of Sports Medicine(Beijing), 6(3), 147-150.

Eberle, T., Hessling, G., Ulmer, HE., & Brockmeier, K. (1998). Prediction of normal QT intervals in children. <u>Journal of Electrocardiology</u>, <u>31</u>, 121-125.

Elias, B. A., Berg, K. E., Latin, R. W., Mellion, M. B., & Hofschire, P. J. (1991).

Cardiac structure and function in weight trainers, runners, and runner/weight trainers.

Research Quarterly for Exercise and Sport, 62(4), 326-332.

Fenici, R., Ruggieri, M. P., Brisinda, D., & Fenici, P. (1999). Cardiovascular adaptation during action pistol shooting. <u>The journal of Sports Medicine and Physical Fitness</u>, <u>39</u>(3), 259-266.

Fleck, S. J., Henke, C., & Wilson, W. (1989). Cardiac MRI of Elite Junior Olympic Weight Lifters. <u>International Journal of Sports Medicine</u>, 10, 329-333.

Fuller, C. M., Mcnulty, C. M., Spring, D. A., Arger, K. M., Bruce, S. S., Chryssos,
B. E., Drummer, E. M., Kelley, F. P., Newmark, M. J., & Whipple, G. H. (1997).
Prospective Screening of 5,615 High School Athletes for Risk of Sudden Cardiac
Death. Medicine & Science in Sports & Exercise. 29(9), 1131-1138.

Fredriksen, P. M., Ingjer, F., Nystad, W., & Thaulow, E., (1998). Aerobic endurance testing of children and adolescents-a comparison of two treadmill-protocols.\_

Scandinavian Journal of Medicine & Science in Sports, 8, 203-207.

Giorgi, D., Bello, V. D., Bertini, A., Talini, E., Valenti, G., Gioppi, A., Precisi, S., Pallini, M., Moretti, L., Caputo, M. T., & Giusti, C. (2000). <u>International Journal of Sports Medicine</u>, 21, 616-622.

Glover, D. W., Maron, B. J., & Matheson, G. O. (1999). The Preparticipation Physical Examination. <u>The Physician and Sports Medicine</u>, <u>27</u>(8),

Goldberg, L., Williams, M., Hurley, B., Daniels, W. L., MacDougall, D., & Longhurst, J. C. (1987). Cardiovascular effects of weight training. <a href="National Strength">National Strength & Conditioning Associaton Journal, 9(2), 10-21.</a>

Gravelle, B.L., & Blessing, D. L. (2000). Physiological adaptation in women concurrently training for strength and endurance. <u>Journal of Strength and</u>

Conditioning Research, 14(1), 5-13.

Hanne-Paparo, N., Drory, Y., Schoenfeld, Y., Shapira, Y., & Kellermann, J. J. (1976).

Common ECG changes in athletes. <u>Cardiology</u>, <u>61</u>(4), 267-78.

Henry, W. L., Ware, J., Gardin, J. M., Hepner, S. I., McKay, J., & Weiner, M. (1978). Echocardiographic measurements in normal subjects. Growth-related changes that occur between infancy and early adulthood. <u>Circulation</u>. <u>57</u>, 278-285.

Hurley, B. F., Seals, D. R., Ehsani, A. A., Cartier, L. J., Dalsky, G. P., & Hagberg, J.
M. (1984). Effects of high intensity strength training on cardiovascular function.
Medicine and Science in Sports and Exercise (Indianapolis), 16(5), 483-488.

Ikaheimo, M. J., Palatsi, I. J., & Takkunnen, J. T. (1979). Noninvasive evaluation of the athletic heart:sprinters versus endurance runners. Am J Cardiol, 44(1), 24-30.

Janz, K. F., Burns, T. L., Witt, J. D., & Mahoney, L. T. (1998). Longitudinal analysis of scaling VO2 for differences in body size during puberty: the muscatine study.

Medicine & Science in Sports & Exercise, 1436-1444.

Jensen-Urstad, K., Saltin, B., Ericson, M., Storck, N., & Jensen-Urstad, M. (1997).

Pronounced resting bradycardia in male elite runners is associated with high heart rate variability. Scandinavian Journal of Medicine & Science in Sports, 7, 274-278.

John, R. M., & Andersen, K. L. (1969). Pulmonary diffusing capacity and cardiac output in young trained Norwegian swimmers and untrained subjects. Medicine and Science in Sports, 1(3), 131-139.

Karjalainen, J., Mantysaari, M., Viitasalo. M., & Kujala, U. (1997). Left ventricular mass, geometry, and filling in endurance athletes: association with exercise blood pressure. <u>Journal of Applied Physiology</u>, <u>82</u>(2), 531-537.

MacFarlane, N., Northridge, D. B., Wright, A. R., Grant, S., Gargie, H. J. (1991). A comparative study of left ventricular structure and function in elite athletes. <u>British Journal Sport Medicine</u>, <u>25</u>(1), 45-48.

Malina., R. M,. & Roche, A. F. (1983). Manual of physical status and performance in childhood. <u>Physical performance</u>. <u>2</u>, New York.

Maron, B. J. (1986). Structural features of the athlete heart as defined by echocardiography. Journal of the American College of Cardiology, 7(1), 190-203.

Maughan, R., J. (1992). Aerobic Function. Sport Science Review. 1, 28-42.

Mayo, J., Kravitz, L. (1999). A review of the acute cardiovascular responses to resistance exercise of healthy young and older adults. <u>Journal-of-Strength-and</u>

<u>Conditioning Research</u>, <u>13</u>(1), 90-96.

Melhim, A. F. (2001). Aerobic and anaerobic power responses to the practice of taekwon-do. <u>British Journal of Sports Medicine</u>.35(4), 231-235.

Myers, J. N. (1996). Essentials of Cardiopulmonary Exercise Testing, USA.

National Strength & Conditioning Association. (1987). Cardiovascular effects of weight training. <a href="National Strength">National Strength</a> & Conditioning Association Journal. 9(2), 10-20.

Naughton, G. A., Carlson, J. S., Iuliano, S., Gibbs, M., & Snow, R. J. (1998).

Cardiorespiratory Responses and Circulating Metabolite Concentrations in Male and

Female Adolescents During a Simulated Duathlon. <u>International Journal of Sports</u>

Medicine. 19, 303-309.

Naughton, G., Farpour-Lambert, N. J., Carlson, J., Bradney, M., & Praagh, E. V. (2000). Physiological Issues Surrounding the Performance of Adolescent Athletes.

Sports Medicine. 30(5), 309-325.

Oakley, C.M. (1992). The electrocardiogram in the highly trained athlete. Cardiol Clin,

Obert, P., Stecken, F., Courteix, D., Lecoq, A. D., & Guenon, P. (1998). Effect of long-term intensive endurance training on left ventricular structure and diastolic function in prepubertal children. <u>International Journal of Sports Medicine</u>. <u>19</u>, 149-154.

Osborne, G., Wolfe, L. A., Burggraf, G. W., & Norman, R. (1992). Relationships between Cardiac Dimensions, Anthropometric Characteristics and Maximal Aerobic Power (VO2max) in Young Men. <u>International Journal of Sports Medicine</u>, <u>13</u>, 219-224.

Paul, E., Sehgal, R., Chrystof, D., Neches, W. H., Webb, C. L., Duffy, C. E., Shulman, S. T., & Chaudhry, F. A. (1995). Feasibility of exercise stress echocardiography for the follow-up of children with coronary involvement secondary to Kawasaki disease.

<u>Circulation</u>, 91(1), 122-128.

Paul, V., David, H. C., (1977). Cardiorespiratory alterations in 9 to 11 year old children following a season of competitive swimming. Medicine & Science in Sports.

Pelliccia, A., Maron, B. J., Culasso, F., Di Paolo, F. M., Spataro, A., Biffi, A., Caselli,G., & Piovano, P. (2000). Clinical Significance of Abnormal ElectrocardiographicPatterns in Trained Athletes. <u>Circulation</u>, 3, 278-284.

Pettersen, S. A., Fredriksen, P. M., & Ingjer, F. (2001). The correlation between peak oxygen uptake (VO2peak) and running performance in children and adolescents.

Aspects of different units. Medicine & Science in Sports. 11.223-228.

Potiron-Josse, M., & Bourdon, A. (1989). Athletic heart. Science et Sports (Paris). 4(4), 305-316.

Raskoff, W. J., Goldman, S., & Cohn, K. (1976). The "athletic heart".Prevalence and phydiological significance of left ventricular enlargement in distance runners. <u>The Journal of the American Medical Association</u>, <u>236</u>(2), 158-62.

Ricci, G., Lajoie, D., Petitclerc, R., Peronnet, F., Ferguson, R. J., Fournier, M., & Taylor, A. W. (1982). Left ventricular size following endurance, sprint, and strength training. Medicine and Science in Sports and Exercise, 14(5), 344-347.

Ross, D. S., & Cooper, G. (1999). Acute Onset of Chest Pain in a Soccer Player. <u>The Physician and Sportsmedicine</u>, 27(12), 101-104.

Roos, R. (1989). Cardiac work load may peak in weight training. <u>The Physician and Sportsmedicine</u>, <u>17</u>(6), 34.

Rowland, T., Goff, D., Deluca, P., & Popowski, B. (1997). Cardiac Effects of a Competitive Road Race in Trained Child Runners. Pediatrics, 100(3), 2.

Rowland, T., Goff, D., Popowski, B., Deluca, P., & Ferrone, L. (1998). Cardiac Responses to Exercise in Child Distance Runners. International Journal of Sports Medicine, 19, 385-390.

Rowland, T., Popowski, B., & Ferrone, L. (1997). Cardiac responses to maximal upright cycle exercise in healthy boys and men. Official Journal of the American

College of Sports Medicine. 5, 1146-1151.

Rowland, T.W.(1992). Trainability of the Cardiorespiratory System During Childhood.

<u>Can. J. Spt. Sci.</u> 17, 259-263

Roy, A., Doyon, M., Dumesnil, J. G., Jobin, J., & Landry, F. (1988). Endurance vs. strength training: comparison of cardiac structures using normal predicted values.

Journal of Applied Physiology (Bethesda, Md.), 64(6), 2552-2557.

Salke, R. C., Rowland, T. W., & Burke, E. J. (1985). Left ventricular size and function in body builders using anabolic steroids. Medicine and Science in Sports and Exercise (Indianapolis), 17(6), 701-704.

Sharma, S., Whyte, G., Elliott, P., Padula, M., Kaushal, R., Mahon, Niall., & Mckenna, W. J. (1999). Electrocardiographic changes in 1000 highly trained junior elite athletes.

British Journal of Sports Medicine, 33, 319-324.

Smith, S. A., Humphrey, R. H., & Wohlford, D. L F. (1994). Myocardial Adaptation and Weight Fluctuation in College Wrestlers. <u>International Journal of Sports Medicine</u>,

Stolt, A., Karjalainen, J., Heinonen, O. J., & Kujala, U. M. (2000). Left ventricular mass, geometry and filling in elite female and male endurance athletes. <u>Scandinavian</u>

Journal of Medicine & Science in Sports, 10, 28-32.

Stork, T., Mockel, M., Muller, R., Eichstadt, H., & Hochrein, H. (1992). Left ventricular filling behaviour in ultra endurance and amateur athletes: a stress doppler-echo study. <u>International Journal of Sports Medicine</u>, <u>13</u>, 600-604.

Treuth, M. S., Hunter, G. R., Pichon, C., Figueroa-Colon, R., & Goran, M. I. (1998). Fitness and energy expenditure after strength training in obese prepubertal girls.

Medicine & Science in Sports & Exercise, 1130-1136.

Unnithan, V. B., Timmons, J. A., Brogan, R. T., Paton, J. Y., & Rowland, T. W. (1996). Submaximal running economy in run-trained pre-pubertal boys. <u>Journal of Sports Medicine & Physical Fitness</u>, <u>36</u>(1), 16-23.

Urhausen, A., & Kindermann, W. (1989). One and two dimensional echocardiography

in body builders and endurance trained subjects. Naughton, G., Farpour-Lambert, N. J., Carlson, J., Bradney, M., & Praagh, E. V. (2000). Physiological Issues Surrounding the Performance of Adolescent Athletes. Sports Medicine. 30(5), 309-325.

Vincent, G. M. (1998). Sudden Death in a Young Athlete. <u>The Physician and Sportsmedicine</u>, <u>26</u>(7), 59-62.

Wolfe, L. A., Cunningham, D. A., & Boughner, D. R. (1986). Physical conditioning effects on cardiac dimensions: review of echocardiographic studies. <u>Canadian Journal</u> of Applied Sport Sciences, 11(2), 66-79.

Yang, X., Telama, R., Leino, M., & Viikari, J. (1999). Factors explaining the physical activity of young adults: the importance of early socialization. <u>Scandinavian Journal of Medicine & Science in Sports</u>, *9*, 120-127.

Yamazaki, H., Onishi, S., Katsukawa, F., Ishida, H., & Kinoshita, N. (2000). Peak Aerobic performance and left ventricular morphoogical characteristics in university students. Clinical Journal of Sport Medicine, 10, 286-290.

Yetman, A. L., Hamilton, R. M., Benson, L. N., & McCrindle B. W. (1998).

Long-term outcome and prognostic determinants in children with hypertrophic cardiomyopathy. <u>Journal of the American College of Cardiology</u>, <u>32</u>(7), 1943-1950.

Zauner, C. W., Maksud, M. G., & Melichna, J. (1989). Physiological considerations in training young athletes. Sports Medicine (Auckland). 8(1), 15-31.

# APPENDLX A

SUBJECT INFORMATION AND INFORMED CONSENT STATEMENTS

# Appendix 3.1

Volunteers are needed for a research project comparing the effects of strength and endurance (aerobic) training on the heart in young males.



We are looking for males aged 14-16 years who fit in to ONE of the following three groups:



- Strength trained males: must be doing
   strength training at least three times per
   week and endurance training less than one
   time per week for the past year.
- 2. **Endurance** trained males: must be doing **endurance training at least three times per week** and strength training less than one time per week for the past year.
- 3. **Untrained** males: not more than one session per week of intensive sports training for the past year.

Boys must also be free of injuries for three months prior to testing and generally not be on any prescribed medications.

# If you become involved, you will undergo the following tests and get information

### on:

Test	Equipment	Benefit to you
VO <sub>2max</sub>	Cycle ergometer	Aerobic (endurance) fitness
Exercise	ECG machine	How your heart works during the VO <sub>2max</sub>
ECG		test
Muscle	Cybex machine	Best test for measuring strength for knee
strength		extension and flexion
Sprint testing	Cycle ergometer	Anaerobic (sprint) fitness
Body	DEXA scan	How much muscle and fat you have, and
composition		your bone density
Heart size	Ultrasound	How big your heart is

The testing is done over a three week period with no more than four visits in all.

There are no blood samples or muscle biopsies in this research project!!

For further information, please contact:

# Dr. Steve Selig

School of Human Movement, Recreation & Performance

Victoria University of Technology

Phone: 9688 4421 mobile 0418 570 772

Fax: 9688 4891

Email: <u>Steve.Selig@vu.edu.au</u>

# Appendix 3.2

### Approved by the Human Research Ethics committee of VUT

### Victoria University of Technology

PO Box 14428 Melbourne City MC 8001 Australia Telephone: (03) 9688 4710 Facsimile: (03) 9687 2089

Footscray Park Campus

Office for Research 6 Geelong Road Footsarry



TO:

Dr Steve Selig

School of Human Movement, Recreation and Performance

Footscray Park Campus

COPY:

Ms Jian Rong Shi

School of Human Movement, Recreation and Performance

Footscray Park Campus

FROM:

Dr Neale Yates

Research Information Officer

Office for Research

DATE:

20 September 2000

SUBJECT:

Application HRETH FHD 027/00 involving human subjects

The University Human Research Ethics Committee at its meeting on 14 September 2000 assessed your application for project:

HRETH FHD 027/00:

Cardiac Structure and Function in Young Athletes

During Exercise

(HREC 00/121; HREC 00/99; HREC 00/90 previously

circulated)

It was resolved to approve application HRETH FHD 027/00 (HREC 00/121; HREC 00/99; HREC 00/90) from 15 June 2000 to 1 December 2001.

If you have any further queries please do not hesitate to contact me on ext. 4710.

Dr Neale Yates

Neale Leter

# Appendix 3.3

### **Victoria University**

### **Consent Form for Subjects Involved in Research**

### **INFORMATION TO PARENTS:**

Dear parent/guardian and participant

Your son is being invited to take part in a study entitled

Cardiac Structure and Function, Body Composition and Bone Health of Young

**Athletes during Exercise** 

We are wanting to compare the effects of a long-term intensive endurance and strength training program on the <u>cardiac structure and function</u>, <u>body composition and bone health</u> in adolescents.

The research project is being conducted at Victoria University by: Dr. Geraldine

Naughton, Dr. Steve Selig and Ms Jian Rong Shi

To be eligible for participation into the project I understand that my son will need to meet the following criteria:

- Male
- Aged between 14 and 16 years
- A training history of at least twice weekly <u>endurance training</u> for the past 12 months

OR

A training history of at least twice weekly <u>strength training</u> for the past 12 months

OR

• Not been involved in serious sports training over the past 12 months

# **Summary of Procedures**

Testing in this project will occur at three different venues on up to four different days:

Test	Site of Test	Equipment	Benefit to your son
VO <sub>2max</sub>	Victoria University:	Cycle ergometer	Aerobic (endurance)
	Footscray campus		fitness testing to be
			compared to others of the
			same age
Exercise	Victoria University:	ECG machine	How your heart works
ECG	Footscray campus		during the VO <sub>2max</sub> test
Muscle	Victoria University:	Cybex machine	Knowledge about
strength	Footscray campus		strength and power
			during knee extension
			and flexion
Sprint testing	Victoria University:	Cycle ergometer	Anaerobic (sprint) fitness
	Footscray campus		shows how much power
			you can produce during
			an "all out" cycle over a
			very short period of time

Body	Victoria University:	Skinfolds	How much fatty tissue
composition	Footscray Campus		there is just underneath
			the surface of your skin
Heart size	Royal Childrens'	Ultrasound	How big your heart is
	Hospital		

The testing is done over a three weeks period with no more than four testing sessions involved in the study.

# Testing at Footscray Park Campus of Victoria University

# 1. Aerobic Fitness Test (VO<sub>2</sub> peak)

Aerobic Fitness tests are commonly used for working out the maximal endurance ability in people of all ages. Specifically, in this part of the laboratory testing, my son will be required to perform an exercise test on a stationary cycle up to his maximal ability. The exercise will commence at a comfortable load and increase every minute until my son reaches fatigue. He may request to stop exercise at any time before the end of the test and in this case, we will stop the test. This will not compromise my son in any way. If any unusual signs or symptoms appear, we will also stops the test.

### 2. Cardiograph (ECG)

During the aerobic exercise test, my son's heart rate and rhythm will be monitored continuously using a standard cardiograph (ECG). This involves the placing of 10 sticky electrodes on the chest and my son will wear a transmitter device (the size of a mobile phone) on his waist.

# 3. Muscular strength

Muscle strength will be tested using a device that measures the force applied during a kicking action. My son will sit on a seat and perform kick-up and kick-down activities (knee extension/flexion) for three maximal efforts against a resistance.

Muscular power will also be measured using the same movement pattern for five repetitions at a lighter resistance but a higher speed.

### 4. Sprint/power (anaerobic) testing

Anaerobic tests measure the power produced during a brief but high intensity exercise test. The anaerobic component tested in this project will require my son to perform 5 repeats of 6 seconds of maximal sprint cycling with a 24 second recovery between each sprint. This test will be performed on a Repco cycle ergometer – which is a cycling machine that allows my son to stand up as he cycles to produce a maximal power output. This test has often been conducted previously in the Exercise Physiology laboratory with various sporting populations.

### 5. Body composition by Skinfold Measurements

These are measurements of the size of the fold of skin over a specific site on the body eg biceps, triceps, underneath the shoulder blade and just next to the belly button. The feeling when the callipers are used will be no more than a soft pinch of the skin that lasts about 2-3 seconds. The skinfold measurements will be taken along with height and weight during the visit to the Footscray Park Campus of Victoria University.

# Testing at the Department of Cardiology, Royal Children's Hospital

# 1. Echocardiography to determine heart size

Echocardiography assessments will be used on my son to estimate cardiac structure and function. During this procedure my son will be asked to lie fairly still on a bed for a maximum of 60 minutes. This procedure is very low risk in that it involves the harmless emission of ultrasound waves. Standard Doppler Echocardiographic techniques will be used to estimate the size of the heart and how much blood flows into the heart when it is filling, and out of the heart when it contracts. This procedure will occur only during rest.

### What are the risks?

### Aerobic (VO2 peak) Test

Risks include: (i) Cardiac arrest and sudden death: minimised and managed by

prior screening, adequate staffing (competent and/or qualified in dealing with the medical emergencies that may arise in the laboratory). (ii) vasovagal (fainting) attack: university procedures will be followed for prevention and management of this. (iii) heat illness.

For the last 1-3 minutes of the aerobic fitness test my son may feel very fatigued. The researchers will try to minimise these effects through proper warm up and cool down procedures as well as careful monitoring of heart rate and consistently paying attention to the feelings of my son. If my son becomes distressed or there are unusual signs or symptoms, the test will be stopped immediately. Water and glucose-based drinks will be offered during recovery. Recovery from the test takes usually only a few minutes.

### **Sprint/power testing**

High intensity cycling test will also result in my son feeling very fatigued. There is also a small risk of thigh and calf muscle strain from exercising at high intensity. A vasovagal (fainting) attack is possible following this test, but the researchers are skilled at preventing or dealing with this problem. Again, the researchers will try to minimise these effects through proper warm up and cool down procedures as well as careful monitoring of heart rate using the ECG. Water and glucose-based drinks will be offered during recovery.

### What are the Benefits to Me?

My son will have access to his results of of cardiac structure and function testing, aerobic fitness, anaerobic fitness and body composition profiles. My son will receive feedback regarding the results soon after the test.

I understand that my son will also be contributing extremely useful information to the sporting community. Athletes will benefit from the knowledge that this study provides, because the results of this study may help other researchers, doctors, coaches and adolescent athletes to understand cardiac structure and function, body composition and bone health and how changes may occur in association with training.

Individual results will remain strictly confidential and will not be provided to other participants and their families.

I certify that I have had the opportunity to have any questions answered and that I understand that my son can withdraw from this experiment at any time and that this withdrawal will not jeopardise my son in any way.

I have been informed that the information I provide will be kept confidential.

### WRITTEN CONSENT

I certify that the objectives of the experiment, together with any risks to me associated

with the pro-	cedures listed hereunder to be carried out in the experiment, have been
fully explain	ned by:
either Dr. St	teve Selig, Dr. Geraldine Naughton or Ms Jian Rong Shi
and that I f	reely consent to my child's participation involving the use of these
procedures.	
CERTIFIC	ATION BY PARENT OF PARTICIPANT
I,	
1,	
Of	
(address)	
	am the parent /guardian of

and that I am voluntarily giving my consent for my son to participate in the
experiment entitled:
PARENT / GUARDIAN AND PARTICIPANT CONSENT
I have read this form and I understand the procedures involved and the conditions
under which the tests will be conducted. My child is under the age of 18, consents to
the test and, additionally, I give formal consent for my child to participate in the
testing procedures outlined above <b>WITHOUT</b> medical supervision.
Name of Parent/Guardian Signature of Parent/Guardian
Date
Child should print his/her name here Signature of adolescent
Date

Any queries about your participation in this project may be directed to the researcher (Name: Dr.Steve Selig ph. 96884421 ). If you have any queries or complaints about the way you have been treated, you may contact the Secretary, University Human Research Ethics Committee, Victoria University of Technology, PO Box 14428 MCMC, Melbourne, 8001 (telephone no: 03-9688 4710).

[\*please note: where the subject/s is aged under 18, separate parental consent is required; where the subject is unable to answer for themselves due to mental illness or disability, parental or guardian consent may be required.]



#### VICTORIA UNIVERSITY OF TECHNOLOGY

# INFORMED CONSENT FOR AN EXERCISE TEST FOR VOLUNTEERS UNDER THE AGE OF 18 YEARS

Parents or guardians need to explain the following form to the child who is to perform the exercise test. The child should print his/her name on the form as a means of giving consent, before the parent gives formal consent on behalf of the child. Note: teachers, club leaders, etc. are NOT permitted to give consent for an exercise test on behalf of the children under their care. Please return this <u>Consent Form</u>.

#### 1. EXPLANATION OF THE GRADED EXERCISE TEST

You will perform a graded exercise test on a bicycle ergometer, motor-driven treadmill, rowing ergometer, upper body ergometer or step test. The exercise intensities will begin at a level you can easily accomplish and will be advanced

in stages, depending on your functional capacity. We may stop the test at any time if signs or symptoms occur or you may stop whenever you wish to because of personal feelings of fatigue or discomfort. We do not wish you to exercise at a level which is abnormally uncomfortable for you; for maximum benefit from the test, exercise as long as is comfortable.

#### 2. RISK AND DISCOMFORTS

There exists the possibility of certain changes occurring during the test. They include abnormal blood pressure, fainting, disorders of heart beat, and in very rare instances, heart attack, stroke or death. Every effort will be made to prevent these by preliminary screening and careful monitoring during the test. Should you feel any symptoms of discomfort of any kind, indicate this to us and we will terminate the test immediately.

#### 3. RESPONSIBILITIES OF THE PARTICIPANT

Information you possess about your health status or previous experiences of unusual feelings with physical effort may affect the safety and value of your exercise test. You are responsible to fully disclose such information on the accompanying sheets or when requested by the testing staff. Furthermore you are expected to disclose any feelings of discomfort during the exercise test. The staff will take all reasonable precautions to ensure the safety and

value of your exercise test but we can not be held responsible in the event that you fail to disclose important information to us.

#### 4. BENEFITS TO BE EXPECTED

The results obtained from the exercise test assist in the evaluation of the types of physical activities you might engage in with no or low hazards.

#### 5. INQUIRIES

Any questions about the procedures used in the graded exercise test or in the estimation of functional capacity are encouraged. If you have any doubts or questions, please ask us for further explanations.

#### 6. MEDICAL SUPERVISION

Normally it is not necessary for someone under the age of 35 to need a medical practitioner present for an exercise test. However if your cardiovascular risk factor and medical history indicate the need for medical coverage, we will arrange for a medical practitioner to be present. Alternatively, we will arrange for a medically supervised test if you prefer it that way.

#### 7. FREEDOM OF CONSENT

Your permission to perform this graded exercise test is voluntary. You are free to <u>deny</u> <u>consent now or withdraw consent at any time</u> (including during the exercise test) if you so desire.

#### PARENT'S / GUARDIAN'S CONSENT

**Date** 

I have read this form and I understand the procedures involved and the conditions under which the tests will be conducted. My child is under the age of 18, consents to the test and, additionally, I give formal consent for my child to participate in the exercise test **WITHOUT** medical supervision.

Child should print his/her name here	Signature of child (if able to sign)
omia snouta print mayner nume neve	Signature of ciniu (if usic to sign)
Date	
Name of Parent/Guardian	Signature of Parent/Guardian

## **VUT human Performance UNit**

## RISK FACTOR ASSESSMENT QUESTIONNAIRE

### Please return this form to:

Name: _Dr.Steve Selig or Jian Rong Shi_	Telephor	ie:	9688 44	21	
School of Human Movement, Recreation & Performance					
Victoria University of Technology	Email: ste	ve.sel	ig@vu.e	du.au	
PO Box 14428					
MELBOURNE MC8001	Fax:	9688	4891		
=					
NAME: DATE:	••••••	•••••	•••••	SEX:	M/F
AGE: WEIGHT:	•••••	.(kg)			
HEIGHT:(cm).					
ADDRESS:	•••••				
POSTCODE:					
TELEPHONE: Work: Ho	ome:	•••••	••••••	•••••	••••
FAX:					
MEDICAL HISTORY:					

# In the past have you ever had (tick No or Yes)

	NO '	YES	NO	YES		
Stroke			Congenital Heart Disease			
Myocardial infarction (heart atta	nck)		Disease of Arteries/Veins			
Angina Pectoris			Asthma			
Heart Murmur			Other Lung Disease (eg.emphysema)			
Heart Rhythm Disturbance			Epilepsy			
Rheumatic Fever			Injuries to back, knees, ankles			
List any prescribed medications being taken: Other current illness (give details):  Other previous illnesses						
ALLERGIES: Do you have any allergies NO  YES  If yes, give details:						
SYMPTOMS DURING OR A	FTER	EXERC	CISE			
As a result of exercise, have you	As a result of exercise, have you ever experienced any of the following:					
	N	NO YES		NO YES		
Pain or discomfort in the chest, l	back,	<b>-</b> -	Palpitations (heart rhythm disturbate	nce) 🗆 🗖		
arm, or jaw			or racing heart rate			

Severe shortness of breath or problems	_		Pain in the legs during mild exertion $\Box$	
with breathing during mild exertion				
Dizziness, nausea or fainting	Ì		Severe heat exhaustion (ie heat stroke)	
CARDIOVASCULAR RISK FACTO	RS	<b>5:</b>		
Do you have (tick No, Yes or circle?)	NO	O YES	S DON'T KNOW	
High Blood Pressure		<u> </u>	?	
High Blood Cholesterol/Triglycerides		<b>.</b>	?	
Smoking Habit			Ex-smoker Average/day	
Diabetes			?	
Do you drink alcohol regularly		) C	Average/day	

Please turn over and provide the information requested overleaf.

# Appendix 3.4

#### **FAMILY MEDICAL HISTORY:**

Have members of your immediate family ever had any of the following conditions:								
(tick No, Yes or circle?).								
If you answer Yes or ?, write beside this the member of the family affected (F=father,								
M=mother, B=brother, S=sister, G	M= gı	randmoth	ner, GF=grand	father).				
	NO	YES	FAMILY	AGE	ALIVE			
			MEMBER	(Years)	NOW?			
					(Y/N)			
Myocardial infarction (heart attack	x)□	<b>□</b> ?						
Angina Pectoris		<b>□</b> ?						
Stroke		<b>□</b> ?						
High Blood Pressure		<b>□</b> ?						
High Blood Cholesterol/Triglyceri	ides□	<b>□</b> ?						
Diabetes		<b>□</b> ?						
Cancer		<b>□</b> ?						

## PERSONAL LIFESTYLE:

## A. Exercise (histoty of training over the past 12 months)

	How many	How long	Type of	*Work intensity
	sessions per	was each	exercise	An a scale of 1 to 5 (1=light
	week were	session	performed	or easy training
	your training			5=very heavy or hard
	outside of			training)
	school			
12 moths ago				
(July –				
September of				
2000)				
9 months ago				
(October–				
December of				
2000)				
6 months ago				
(January –				

March of		
2001)		
Currently		
(April – June		
of 2001)		

#### • Work intensity:

1=light training that does not result in sweat and making you feel tired.

2=training that results in little sweat and feeling a little tired. Recovery happens in a few minutes.

3=training that results in much sweat, a heart rate increase (140-150 beat/min) and feeling obviously tired. Recovery takes a few minutes to fifteen minutes.

4=trsining that results in much sweat, a high heart rate increase (above 150 beat/min) and feeling more tired. Recovery takes more than fifteen minutes.

5=Hard work sessions are those that really make your heart rate increase (above 180 beat/min ), sweat a lot and feel very tired.

#### B. Nutrition

List a typical day's eating pattern.

Breakfast	Lunch	Dinner	Snacks	Drinks

How many hours sleep do you usually have? hours
On average how much time do you spend each day on passive hobbies or
just relaxing minutes/hours.

Do you feel that you usually get sleep or enough

C. Rest/Recreation

time to relax? NO YES

OFFICE USE ON
CLEARANCE TO UNDERGO AN EXERCISE 1
This person has been cleared to undergo a fitness
test:
☐ Without medical supervision
☐ With medical supervision
A fitness test is not advisable at this time
Signed: Dr/Mr/Mrs/Ms
(Circle appropriate title:
Physician/exercise physiologist)

Please turn over and provide the information requested overleaf.

# **APPOINTMENT SHEET: Cardiac Structure and Function in Young Athletes**

## **During Rest and Exercise**

Name:	Email: _	
DI.	4	
Phone:	_ (home)	Mobile:
Postal Address:		
Pcode:		

Test	Instructions	Date	Time	Place
VO <sub>2max</sub>	1. Do not exercise on day of test.			305 room, L build
& ECG	2. If exercising on day before test then			ing, Footscray
	make it light exercise.			Park Campus,
	3. Eat a light meal 2-3 hours prior to the			Victoria
	test, or as directed. Avoid coffee, tea,			University of
	alcohol and non-prescription drugs for			Technology.
	three hours prior to the test.			
	4. Bring running shoes and shorts, or			

	tracksuit.
	5. Wear a T-shirt over the top.
	6. Change and shower facilities are
	available (please bring towel.).
Muscle	As above
strength	
Sprint	As above
testing	
Body	Do not exercise on day of test.
composit	2. If exercising on day before test then
ion	make it light exercise.
	3. Eat a light meal 2-3 hours prior to the
	test, or as directed. Avoid coffee, tea,
	alcohol and non-prescription drugs for
	three hours prior to the test.

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oital,
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•

#### **Dear Nash Kovacevic**

We have make appointment of testing time for you on 12/July. If you have any further queries regarding the above, please do not hesitate to contact me on mobile: 0402 128 843 or <a href="mailto:xsirau@hotmail.com">xsirau@hotmail.com</a>. Fax: 9688 4891

Regards,

Jian

## RESULT REPORTING

Name:	Age:
<b>Body Composition</b>	
Weight kg	Percentile ranking th
	(50th percentile = average for age)
Height cm	Percentile ranking th
	(50th percentile = average for age)
%Body fat	
(average = 15%)	
Endurance (VO <sub>2</sub> peak test)	
Score of m	nl.kg <sup>-1</sup> min <sup>-1</sup>
$35-45 \text{ ml.kg}^{-1} \text{min}^{-1} = \text{average}$	e for adolescent male
$45-55 \text{ ml.kg}^{-1}\text{min}^{-1} = \text{good}$	
55-60 ml.kg-1min-1 = very §	good
60+ = elite	

Peak Power

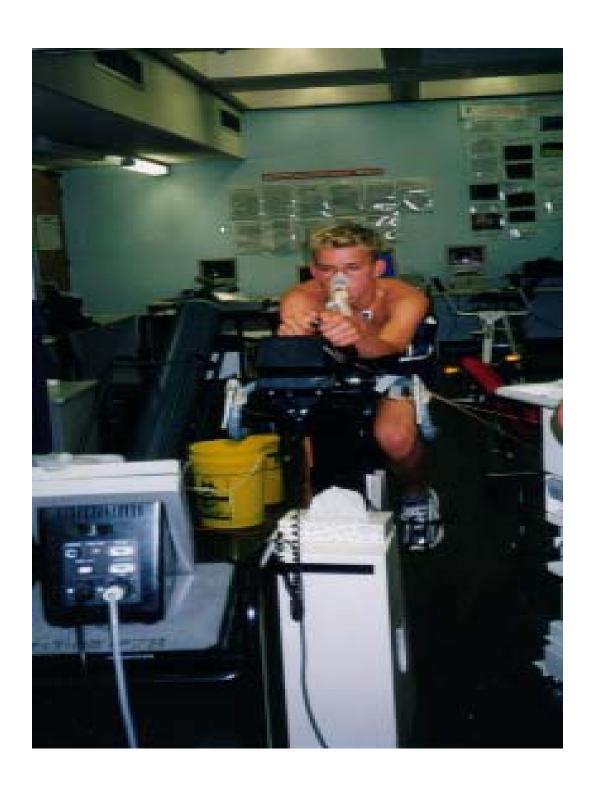
(Above 600 Watts is very good)

**Anaerobic bike sprints** 

Leg Strength
Score
Electrocardiogram
Normal Yes or No
Echocardiogram

Normal Yes or No





APPENDIX B

**RAW DATA** 

# VO<sub>2</sub>max-hr detail (endurance)

	PRE-	TEST	TESTING=HR							
	HR	1	2	3	4	5	6	7	8	
Patrick.S	110	131	136	137	148	159	162	171	180	
Ryan.B	51	85	85	90	100	107	115	122	138	
Liam.A	69	108	106	121	130	147	163	173	182	
Evan.C	69	112	100	106	107	117	125	137	139	
Trent.G	86	121	114	127	131	157	137	144	180	
Russ.G	69	118	118	127	137	149	162	171	178	
Evan.R	62	108	108	117	119	124	129	135	140	
Andrew.W	63	84	89	95	103	104	112	118	127	
Matthew.P	69	81	100	107	113	125	133	140	149	
Damon.E	61	100	111	115	126	132	143	153	166	
Matthew.L	59	108	118	120	131	138	149	158	166	
Nathan.W	62	91	89	103	105	110	113	130	137	
Samuel.Mc	81	125	126	130	139	142	153	167	175	
Nash.K	56	113	125	133	158	161	169	179	186	

	TEST	TESTING-HR									
	9	10	11	12	13	14	15	16			
Patrick.S	191	197	197								
Ryan.B	150	160	171	182	189						
Liam.A	188										
Evan.C	143	157	164	171	178	186	194				
Trent.G	167	163	165	177	188	192	197	202			
Russ.G	185	190									
Evan.R	148	143	146	174	183						
Andrew.W	134	145	146	136	160	139	144	202			
Matthew.P	159	169	174	179	184	180					
Damon.E	176	185	190	198							
Matthew.L	164										
Nathan.W	145	156	164	173	179	168					
Samuel.Mc	179	189	192	198	202	205	211				
Nash.K	192										
	RECO	DE-HR									
	1	2	3	4	5						

Patrick.S	167	159	160				
Ryan.B	136	99	100	98	54		
Liam.A	156	134	129	118	103		
Evan.C	131	115	106	93	102		
Trent.G	180	158	149	145	148		
Russ.G	151	128	112	118	113		
Evan.R	160	138	135	165	106		
Andrew.W	149	122	150	142	96		
Matthew.P	148	118	109	99	98		
Damon.E	169	153	154	146	138		
Matthew.L	150	133	118	95	99		
Nathan.W	129	118	117	115	99		
Samuel.Mc	190	161	163	155	154		
Nash.K	163	153	137	116	122		

# VO<sub>2</sub>max-hr detail (control)

	PRE-	TEST	ΓESTING-HR							
	HR	1	2	3	4	5	6	7	8	
Jarrod.B	94	123	116	119	124	133	145	158	164	
David.S	100	148	143	134	135	149	147	173	175	
Shane.F	81	111	114	113	115	112	109	113	114	
Robert.B	64	108	107	112	117	139	155	167	176	
Nick.C	71	105	116	119	130	141	150	162	134	
Gregar.E	62	122	121	128	139	146	160	162	178	
	TEST	ING-HI	R							
	9	10	11	12	13	14	15	16		
Jarrod.B	172	187	197	184						
David.S	166	180	195	198						
Shane.F	138	146	163	178						
Robert.B										
Nick.C										
Gregar.E	187	193	197	201						

	RECOE-HR						
	1	2	3	4	5		
Jarrod.B	173	148	128				
David.S	175	165	153				
Shane.F	125	99	113	86	101		
Robert.B	160	122	115	116	102		
Nick.C	103	101	94	90	80		
Gregar.E	124	101	95	115	103		

# **Testing Detail (endurance)**

Name	Height	Weight	BSA	Bady	Bady	LBM	LBM/kg	SBP
	cm	kg	$M^2$	Fat %	Fat/kg	%		mmHg
Patrick.S	172	63	1.75	8.96	5.64	91.04	57.36	122
Evan.C	192	78.56	2.08	10.09	7.92	89.91	70.64	130
Trent.G	177.2	71.18	1.88	10.64	7.57	89.36	63.61	118
Russ.G	164	48.69	1.51	7.52	3.66	92.48	45.03	108
Evan.R	181	61.52	1.79	7.52	4.63	92.48	56.89	126
Ryan B	168.5	53.2	1.60	7.65	4.07	92.35	49.13	96
Liam.A	152.5	38.3	1.29	5.61	2.15	94.39	36.15	110
Andrew.W	180	63.34	1.81	7.14	4.52	92.86	58.82	130
Matthew P	175.4	61	1.74	11.36	6.93	88.64	54.07	124
Matthew.L	170.5	57.22	1.66	7.14	4.08	92.86	53.14	118
Damon.E	168	56.95	1.64	7.14	4.06	92.86	52.89	118
Samuel.Mc	186.7	72.43	1.97	14.06	10.19	85.94	62.24	116
Nathan.W	176.5	64.6	1.80	10.19	6.58	89.81	58.02	120
Evan.G								

	DBP	VO <sub>2</sub> max	VO <sub>2</sub>	VO <sub>2</sub>	Peak	Peak	Leg Stren	gth
Name	mmHg	ml/kg/m	max	max	HR	Power	(Peak Tor	que)
		in	L/mi	Per kg				
			n	LBM				
							RCF	LCF
Patrick.S	88	44.36	2.79	48.64	203	839		
Evan.C	78	55.21	4.34	61.44	189	1178	33.2	58.6
Trent.G	80	54.85	3.89	61.15	189	1262	58.6	58.6
Russ.G	68	51.63	2.51	55.74	195	666		
Evan.R	78	55.8	3.43	60.29	206	817	47.5	43.6
Ryan B	68	56.47	3	61.06	192	660	63.1	64.7
Liam.A	70	58.22	2.23	61.68	204	407	38.1	59.2
Andrew.W	80	65.34	4.14	70.38	216	740	76.5	91.1
Matthew P	82	53.89	3.29	60.85	185	868	66.9	68.5
Matthew.L	54	39.81	2.28	42.91	198	740		
Damon.E	80	59.36	3.38	63.91	166	924		
Samuel.Mc	66	57.98	4.2	67.48	179	998		
Nathan.W	80	54.41	3.51	60.50	211	872		
Evan.G								

		T						
Name	RCE	LCE	Rest	IS	LVEd	LVEd/	LVEs	LVEs/
			HR			m <sup>2</sup>		m <sup>2</sup>
Patrick.S			60	0.85	4.98	2.85	3.7	2.12
Evan.C	58	74.8	62	0.74	6.11	2.94	4.65	2.24
Trent.G	66.7	89.5	65	0.86	5.4	2.88	3.7	1.97
Russ.G			62	0.6	4.61	3.05	3.11	2.06
Evan.R	80.7	70	72	0.82	5.1	2.84	3.48	1.94
Ryan B	52.4	80	50	0.96	5.7	3.56	3.8	2.37
Liam.A	35.6	33.3	61	0.73	4.6	3.55	3.2	2.47
Andrew.W	88.2	75	65	0.58	5.8	3.21	3.7	2.05
Matthew P	81.6	93.2						
Matthew.L			66	0.69	5.5	3.30	3.8	2.28
Damon.E			69	0.71	5.1	3.10	3.2	1.95
Samuel.Mc			65	0.58	5.46	2.78	3.69	1.88
Nathan.W			61	0.93	5.13	2.85	3.76	2.09
Evan.G			67	0.93	4.83	2.73	2.93	1.65

Name	PW	PW/m <sup>2</sup>	SV	SVind	СО	COind	FS%
				exed		exed	
Patrick.S	1.02	0.58	71.1	40.74	4.26	2.47	25.6
Evan.C	0.82	0.39	118	56.84	7.29	3.57	24
Trent.G	0.74	0.39	86	45.79	5.57	3.00	31
Russ.G	0.97	0.64	87	57.58	7.18	4.00	33
Evan.R	0.71	0.40	92.1	51.37	6.64	3.80	32
Ryan B	0.84	0.52	110	68.74	5.50	3.50	34
Liam.A	0.69	0.53	69.1	53.38	4.22	3.35	30.4
Andrew.W	0.74	0.41	123	68.04	8.00	4.60	37
Matthew P							
Matthew.L	0.75	0.45	87.5	52.56	5.80	3.50	31
Damon.E	0.66	0.40	69	41.98	4.75	2.90	38
Samuel.Mc	0.60	0.31	104	52.92	6.78	3.50	32
Nathan.W	0.93	0.52	78	43.40	4.72	2.64	26.7
Evan.G	0.91	0.51	83.7	47.23	5.60	3.16	40

# **Testing Detail (control)**

Name	Height	Weight	BSA	Bady	Bady	LBM	LBM/kg	SBP
	cm	kg	$M^2$	Fat %	Fat/kg	%		mmHg
Jarrod.B	172.5	64.5	1.77	18.22	11.75	81.78	52.75	108
David.S	178	64.1	1.80	13.53	8.67	86.47	55.43	112
Shane.F	159.5	43.6	1.41	10.84	4.72	89.16	38.88	104
Robert.B	183	59.76	1.78	10.04	6	89.96	53.76	110
Nick.C	164.5	52.78	1.57	8.14	4.30	91.86	48.48	110
Gregar.E	177	70	1.86	16	11.20	84	58.80	112
Nash.K	164	55.76	1.60	5.83	3.25	94.17	52.51	118
	DBP	VO <sub>2</sub> max	VO <sub>2</sub>	VO <sub>2</sub>	Peak	Peak	Leg Strength	
Name	mmHg	ml/kg/m	max	max	HR	Power	(Peak Torque)	
		in	L/mi	Per kg				
			n	LBM				
							RCF	LCF
Jarrod.B	72	47.76	3.08	58.39	200	722	81.9	58.9
David.S	68	49.46	3.17	57.19	198	954	75.5	71.8

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70	48.35	2.11	54.28	190	433	49.3	22.2
62	44.50	2.66	49.48	176	886	65.6	49.2
60	36.87	1.95	40.22	162	752		
64	47.21	3.31	56.29	201	768		
68	46.63	2.60	49.51	194	738		
RCE	LCE	Rest	IS	LVEd	LVEd/	LVEs	LVEs/
		HR			$m^2$		m <sup>2</sup>
92.2	77	54	0.83	5.90	3.27	3.9	2.16
91.7	85.7						
63.4	27.1	57	0.79	5.29	2.96	3.63	2.03
129.4	68.4	66	0.73	4.95	3.16	2.7	1.72
		54	0.77	5.76	3.09	3.75	2.01
		62	0.78	4.98	3.11	3.02	1.89
		67	0.93	4.83	3.02	2.93	1.83
PW	PW/m <sup>2</sup>	SV	SVind	СО	COind	FS%	
			exed		exed		
0.72	0.40	108	59.90	5.8	3.3	33	
	62 60 64 68 RCE 92.2 91.7 63.4 129.4 PW	62 44.50 60 36.87 64 47.21 68 46.63  RCE LCE  92.2 77 91.7 85.7 63.4 27.1 129.4 68.4  PW PW/m²	62 44.50 2.66 60 36.87 1.95 64 47.21 3.31 68 46.63 2.60  RCE LCE Rest HR  92.2 77 54  91.7 85.7 57  129.4 68.4 66  14.50 54  62  PW PW/m² SV	62 44.50 2.66 49.48 60 36.87 1.95 40.22 64 47.21 3.31 56.29 68 46.63 2.60 49.51  RCE LCE Rest IS HR  92.2 77 54 0.83  91.7 85.7	62	62 44.50 2.66 49.48 176 886 60 36.87 1.95 40.22 162 752 64 47.21 3.31 56.29 201 768 68 46.63 2.60 49.51 194 738  RCE LCE Rest IS LVEd LVEd/ HR	62       44.50       2.66       49.48       176       886       65.6         60       36.87       1.95       40.22       162       752         64       47.21       3.31       56.29       201       768         68       46.63       2.60       49.51       194       738         RCE       LCE       Rest       IS       LVEd       LVEd/       LVEs         HR       m²       m²       3.27       3.9         91.7       85.7       0.79       5.29       2.96       3.63         129.4       68.4       66       0.73       4.95       3.16       2.7         54       0.77       5.76       3.09       3.75         62       0.78       4.98       3.11       3.02         PW       PW/m²       SV       SVind       CO       COind       FS%         exed       exed       exed       exed

David.S								
Shane.F	0.86	0.48	100	56.03	5.72	3.3	31.3	
Robert.B	0.87	0.56	91.1	58.13	5.98	3.9	45	
Nick.C	0.66	0.35	87.4	46.90	4.69	2.52	34.81	
Gregar.E	0.83	0.52	84.3	52.66	4.25	3.3	39.2	
Nash.K	0.91	0.57	83.7	52.31	5.6	3.16	40	