USING BALLISTOCARDIOGRAPHY TO EVALUATE CARDIAC PERFORMANCE IN TRAINED MALE ICE HOCKEY PLAYERS

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ABSTRACT

During exercise the demand on the heart increases considerably. Evidence suggests that long-term exercise training causes adaptations to the heart that are not present in the sedentary, and is commonly referred to as athlete's heart. It is not unusual for the cardiac adaptations to mimic certain pathological conditions. Therefore, being able to differentiate the athletic heart from the pathological heart has important implications for trained athletes. The presence of cardiovascular disease permits disqualification from competition or cessation of training to prevent progression of the disease or sudden cardiac death. Commonly used diagnostic tools to evaluate cardiac performance (e.g., echocardiography) can be time consuming and costly, especially for mass screening of athletes.

Ballistocardiography (BCG) is a non-invasive technology that has been used to record ultra-low frequency vibrations of the heart allowing for the measurement of important cardiac cycle events including timing and amplitudes of contraction. Recent developments in BCG have made this technology simple to use, as well as time- and cost-efficient in comparison to other more complicated and invasive techniques used to evaluate cardiac performance.

Therefore, the following studies in this thesis project have attempted to (a) demonstrate the utility of using BCG as a screening device, and (b) determine any differences occurring in the athletic heart. The timing and amplitude of cardiac events in trained ice hockey players as well as a recreationally active control group were evaluated and compared using independent sample *t*-tests. Results found in the following studies demonstrated the utility of using simple, non-invasive BCG to measure cardiac

performance, particularly for mass screening. As well, significant differences in cardiac performance were found between trained participants and the control group, thus allowing for the conclusion that regular exercise training leads to physiological changes of the heart.

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LIST OF ABBREVIATIONS, SYMBOLS, AND NOMENCLATURE

BCG	Ballistocardiography
ECG	Electrocardiography
mG	Milligravity
MRI	Magnetic resonance imaging
SCG	Seismocardiography

THESIS GOALS AND STRUCTURE

In this thesis, the utility of ballistocardiography (BCG) to assess athletes' cardiac performance at rest was demonstrated through the testing of both trained ice hockey players and a recreationally active control group. It has been noted that cardiovascular screening tools can play an important role in evaluating the athlete's heart during routine medical exams (Corrado et al., 2005). Diagnostic tools such as electrocardiography (ECG), echocardiography and cardiac magnetic resonance imaging (MRI) are currently used to evaluate the hearts of the athletes. However, because of advances in micro-processor and computer technology, BCG has the potential to become an important screening tool for athletes. As a result, the goals of this thesis included the following:

1) To demonstrate the utility of using BCG to assess and evaluate cardiac performance in trained ice hockey players.
2) To compare the hearts of trained athletes to a recreationally active control group, and determine if differences are present with high levels of exercise training.
3) To determine what specific differences occur to the timing of cardiac events in trained ice hockey players compared with recreationally active adults.
4) To determine what specific differences occur to the amplitude of cardiac events (a surrogate of contractility force) in trained ice hockey players compared with recreationally active adults.
5) To provide a thorough review of traditional and more commonly used cardiovascular diagnostic devices as well as an in-depth review of the field of BCG.

This thesis constitutes a paper format thesis. A collection of three prepared manuscripts which represent individual chapters in this thesis, have been developed in an attempt to address the above goals. Through these included papers, the following general hypothesis will be addressed:

H = That BCG would be able to assess and evaluate the differences that occur in the athletic heart and determine the general and specific changes of the cardiac cycle in trained male ice hockey players by analyzing the different waveforms of the BCG.

Additionally, an attempt to address several specific hypotheses related to the general hypothesis will be made as well. These hypotheses include:

$\mathbf{H_1}$ = There will be a significant difference in measured cardiac timing events between trained ice hockey players and recreationally active adults.

$\mathbf{H_2}$ = There will be a significant difference in the contractility of the heart as reflected by the amplitude of the different measured waveforms, between the trained ice hockey players and recreationally active adults.

Chapter 1 provides a thorough review of various cardiovascular diagnostic devices as well as an in-depth review of the history of technological advances in the field of BCG. **Chapter 2** describes the athletic heart as characterized by the timing events of the cardiac cycle recorded by BCG. It provides normative data that helps to differentiate and compare the athletic heart with that of the control group. Annotated and analysed BCG waveforms and cardiac events are discussed as well as any differences or patterns that occurred in the trained athletes. **Chapter 3** examines the amplitude of recorded BCG

cardiac cycle events to explore the contractile nature of the athletic heart. This study provides additional information on the mechanical function of the athletic heart.

THESIS INTRODUCTION

Vigorous exercise training that is regularly endured by athletes can lead to a number of physiological adaptations, particularly in cardiac function (Fazel, Roberts, Brooks, & Graybum, 2009). The study of cardiac function and performance resulting from exercise training has been an area in research since the early 1900s. The cardiovascular system plays an important role in the human body, especially during exercise in athletic populations. Its main role is to provide energy through oxygenated and nutrient-rich blood to the body's tissues, and therefore plays a fundamental role in optimal exercise performance (Chummun, 2009; Esch, Bredin, Haykowsky, Scott, & Warburton, 2007). Under strenuous exercise conditions the demand on the heart increases considerably. During exercise, there are numerous physiological changes that occur, all of which occur in an integrative and coordinated manner. These changes not only affect the cardiovascular system, but also the entire body. Various organ systems are affected and can have an influence on the cardiovascular system's response to exercise (Charlton & Crawford, 1997).

The long term effect on the cardiovascular system and the body is dependent on the type of exercise as well as the intensity, duration, and the frequency of which training is performed. Exercise can be separated into primarily dynamic or static exercise (Charlton & Crawford, 1997). Athletes involved in sports with predominately static work such as shot putting, have a disproportionate increase in wall thickness relative to cavity size. Those involved in predominately dynamic sports such as long-distance running, have an increase in cavity size and a modest increase in wall thickness (Bossone, Vriz, Bodini, & Rubenfire, 2004). These adaptations, amongst others, are commonly referred

to as athlete's heart (Bossone et al., 2004). As a result of the volume of exercise training that is performed, only high intensity competitive sports and/or intense exercise training leads to the development of athlete's heart (Rost & Hollmann, 1992).

Athlete's heart has been an area of interest since the 19th century. However, it remains inconclusive whether the athletic heart is physiologically adapted, extremely effective and healthy, or a heart that is sick and borderline pathological. The differentiation between physiological and pathological features has important implications for athletes. Exercise may aggravate minor cardiac conditions that may not be of concern during everyday activity, and can result in serious cardiac disease or possibly even a fatal incident during a sports event (Maron, 2009; Maron & Pelliccia, 2006; Rost & Hollmann, 1992). Therefore, the following studies in this thesis project attempted to determine what cardiac differences are present in the athletic heart as compared to a recreationally active control group. These studies observed cardiac performance in highly trained ice hockey players who performed a combination of moderate static and high dynamic exercise in their sport (Bossone et al., 2004).

Cardiovascular Function and Types of Exercise

Dynamic Exercise. Dynamic exercise is often used synonymously with endurance or aerobic training (Charlton & Crawford, 1997; Fagard, 1997). Using long distance running as an example, dynamic aerobic exercise requires more oxygen to support increased musculature activity than static exercise. How much energy or oxygen that is needed is dependent on the intensity and duration of the dynamic activity. Therefore, a primary acute response with aerobic exercise is an increase in oxygen consumption (VO_2) (Charlton & Crawford, 1997; Plowman & Smith, 2003). Maximal

oxygen consumption, or VO_2max, is a product of cardiac output (total amount of blood released from the left ventricle per minute) and arteriovenous oxygen difference (A-VO_2). VO_2max can easily be tested and provides a measurement of both circulatory and respiratory capacity (Cunningham, Telford, & Swart, 1976). Increases in VO_2max characterize adaptations to exercise training.

A-VO_2 is the difference between the amount of oxygen returned in venous blood and the amount originally carried in arterial blood (Charlton & Crawford, 1997; Plowman & Smith, 2003). During exercise, blood flow is redistributed so that it is reduced to abdominal organs and increased to the working muscles. A-VO_2 can increase up to three times due to an increase in both oxygen extraction across active muscle capillary beds and a redistribution of cardiac output (blood) to the active muscles.

Cardiac output is a product of heart rate (frequency of contraction per minute) and stroke volume (the amount of blood ejected with each heart beat). During maximal dynamic exercise cardiac output can increase up to four times, heart rate can triple, and stroke volume can double. Cardiac output increases primarily from an increase in heart rate which is triggered by activation of the sympathetic nervous system in response to exercise intensity (Rowell, 1986). However, stroke volume also contributes, as it can increase with improved venous return caused by the active muscles pumping blood back to the heart during exercise. With an improvement in stroke volume from training, a lower heart rate is required to provide adequate cardiac output for the workload and VO_2. Evidence suggests that in trained athletes stroke volume can continue to increase until task failure (Gledhill, Cox, & Jamnik, 1994). Overall, improvements in all of these

parameters result in an increase in VO_2max which helps support the demands of the aerobic system during dynamic exercise (Charlton & Crawford, 1997).

Static Exercise. With static exercise, force is developed with no or minimal movement and is also referred to as anaerobic or power exercise. Static exercise involves activities such as weightlifting, shot putting, handgrip exercises, and wrestling. The effect static exercise has on the cardiovascular system is dependent on the intensity of muscle contraction (Charlton & Crawford, 1997; Plowman & Smith, 2003). During static exercise, increases in cardiac output occur primarily as a result of increased heart rate. The intensity of exercise and activation of the sympathetic nervous system are responsible for increasing the rate of the heart (Charlton & Crawford, 1997; Plowman & Smith, 2003).

Stroke volume remains fairly constant during low-intensity contractions and decreases during high-intensity contractions. Upon cessation of static activity there is a remarkable increase in stroke volume, particularly with high-intensity contractions (Charlton & Crawford, 1997; Lind, Taylor, Humphreys, Kennelly, & Donald, 1964; Plowman & Smith, 2003; Smith, Misner, Bloomfield, & Essandoh, 1993). This is the result of a decreased preload (volume of blood returned to the heart) and increased afterload (resistance presented to the contracting ventricle). Decreases in preload occur because of high intrathoracic pressure that compresses the vena cava, reducing venous return. Static exercise triggers a rapid increase in both systolic and diastolic pressure, thus there is an increase in mean arterial blood pressure (afterload) (Donald et al., 1967; Lind et al., 1964; Seals, Washburn, & Hanson, 1985; Tuttle & Horvath, 1957). Increased

afterload or resistance on the contracting ventricle, results in less blood ejected at a given force of contraction (Plowman & Smith, 2003).

As with any physical activity, static exercise increases the demand on the active muscle, however it also results in an increase in intramuscular pressure during muscular contraction. This increase in pressure results in constriction of blood vessels, which impedes muscle blood flow (Charlton & Crawford, 1997; Freund, Gobbs, & Rowell, 1979; Plowman & Smith, 2003; Sjogaard, Savard, & Juel, 1988). As soon as the contraction ceases, the occlusion to the muscle is released and there is a remarkable increase in blood flow which compensates for the reduced flow during muscle contraction (Lind et al., 1964; Rowell, 1993). Because there is minimal blood flow to the muscles, the anaerobic system provides energy to perform static work. During dynamic exercise, occlusion of blood vessels leading to the muscles also occurs. However, there are alternating periods of muscle contraction and relaxation, allowing for blood flow through the venous system (Plowman & Smith, 2003). In contrast to dynamic exercise, static work primarily results in skeletal muscle adaptations and has a minimal effect on cardiovascular responses as much of the energy used during static exercise is consumed by anaerobic mechanisms (Charlton & Crawford, 1997).

Any adaptations that occur in the cardiovascular system depend on the type of exercise, frequency, duration, and intensity of the training. Engaging in predominantly static exercise will result in minimal cardiovascular adaptations. Conversely, performing mainly dynamic exercise for 30 to 60 minutes, three to four times a week at 60-70% of maximal VO_2 during the exercise session will provide cardiovascular adaptations, that lead to an increase in VO_2 (Charlton & Crawford, 1997; Clausen, 1977). However, it is

important to note that most exercise is not purely dynamic or static, as most training programs are a combination of the two forms and tend to overlap. For example, long distance running is an ideal example of dynamic exercise, but it does also require some static muscular activity to maintain upright position (Charlton & Crawford, 1997; Fagard, 1997).

Structural Changes and Hemodynamic Load

In response to chronic dynamic or static training, enlargement of chambers or hypertrophy of the heart muscle can occur. Hypertrophy simply refers to an increase in muscle mass. Enlargement of the heart is the dilatation of a particular chamber, to hold an increased amount of blood (Thaler, 2010). These adaptations occur to accommodate increases in cardiac output. Through an increase in cardiac output, the heart improves its efficiency and capability to meet the demands placed on the cardiac system during exercise.

During dynamic exercise, the vasculature of large muscles involved vasodilate and increase venous return to the heart which can cause volume overload. This volume overload can lead to eccentric hypertrophy of the heart, which is characterized by chamber enlargement as well as change in ventricular wall thickness. Concentric hypertrophy can also occur, but it is in response to static exercise. The type of hypertrophy that occurs is the result of the amount of blood the heart has to pump into the body during exercise. With dynamic exercise the body requires greater amounts of oxygen to perform than static exercise. Static activities such as weightlifting involve developing muscular tension against resistance with little or no movement. This causes a pressure load on the heart rather than volume overload and results in concentric

hypertrophy. Ultimately, the effect on the heart is dependent on whether the activity produces a pressure or a volume load (McMullen & Jennings, 2007). Both types of hypertrophy whether eccentric or concentric hypertrophy, can lead to a condition termed hypertrophic cardiomyopathy.

Occasionally, long-term participation in intense physical activity or sports can result in hypertrophic cardiomyopathy which can be associated with sudden cardiac death (Maron & Pelliccia, 2006; Rost & Hollmann, 1992). There are two types of hypertrophic cardiomyopathy. One type is characterized by adaptations to cardiac tissues and the cardiovascular system as a result of chronic exercise. These adaptations allow for participation in intense exercise such as long distance running or intermittent sports such as basketball or hockey. The other form of hypertrophic cardiomyopathy, that is less common in athletes and has a prevalence of 0.2% in the general population, is the result of a genetic disorder and is pathological. However, both types, if undetected, can result in sudden cardiac death (Maron & Pelliccia, 2006; Rost & Hollmann, 1992). Without adequate screening of the heart hypertrophic cardiomyopathy can easily go undetected, as it is almost always asymptomatic even though a number of cardiac abnormalities may be present (Maron & Pelliccia, 2006; Rost & Hollmann, 1992). It is important to screen those involved in continual physical activity, especially highly trained athletes.

The Athletic Heart

Dr. Salomon Henschen, a Swedish physician, first studied the effect of physical activity on the heart during the last decades of the 19th century (Rost, 1982; Rost & Hollmann, 1983). During a basic physical examination, he performed careful percussion on the chests of cross-country skiers and found signs of an enlarged heart. Cardiac

enlargement was later confirmed through radiography testing as well as from evidence discovered during autopsy. The development of echocardiography in the 1970s further allowed clinicians to gain better insight on the athletic heart and also permitted the study of wall thicknesses, internal dimensions and functions, as well as estimations of left ventricular mass. There is evidence that long-term training in athletes produces adaptations in the heart, which are not present in sedentary individuals and is frequently referred to as athlete's heart (Fagard, 1997; Kervancioglu & Hatipoglu, 2007).

Athlete's heart is a physiological adaptation that occurs in response to habitual exercise and typically results in left ventricular cavity enlargement and increased wall thickness, as well as mitochondrial biogenesis (Caso et al., 2006; Rimbaud, Garnier, & Ventura-Clapier, 2009). These structural changes, along with electrical adaptations are commonly observed on electrocardiograph (ECG) readings (Corrado, Biffi, Basso, Pelliccia, & Thiene, 2009). It is not uncommon for athletes to display abnormal ECG patterns and cardiac dimensions falling outside clinically accepted values (Maron & Pelliccia, 2006). Eighty percent of highly trained athletes show ECG pattern changes such as sinus bradycardia, first-degree atrioventricular block, and early repolarization. These pattern changes result from physiological adaptations of the cardiac autonomic nervous system, such as increased vagal tone and/or withdrawal of sympathetic activity, and occur in response to strenuous training conditions (Corrado et al., 2009).

It is important to be able to differentiate and characterize these physiological adaptations that occur in response to training from the less common patterns that occur in 5% of the population and are unrelated to training conditions. Some of the unrelated changes can include ECG patterns such as ST-T repolarization abnormalities,

pathological Q-waves, intraventricular condition defects, ventricular pre-excitation, long and short QT interval, and Brugada-like repolarization changes. These changes to the pattern may indicate the presence of underlying cardiovascular disorders, such as inherited cardiomyopathies or cardiac ion channel diseases which can predispose and put the athlete at risk for sudden cardiac death (Corrado et al., 2009; Maron & Pelliccia, 2006). It is important that ECG abnormalities associated with athletic adaptations and underlying cardiovascular disease are properly identified. However, not all cardiac abnormalities are detected with ECG (Maron, 2009).

It is currently difficult to differentiate between an athlete's heart and a pathological heart using traditional diagnostic techniques such as ECG. Being able to identify this difference has important implications for high performance athletes. High-risk individuals are not recommended to participate in intense competitive sports and can even be disqualified to prevent death or progression of the disease (Caso et al., 2006; Maron & Pelliccia, 2006; Neary, MacQuarrie, Jamnik, et al., 2009; Sharma et al., 2002). Differentiation between athletic and pathological adaptations can also help reduce unnecessary distress and costs associated with additional testing to exclude any pathological problems (Corrado et al., 2009). Diagnostic dilemmas arise when an athlete's heart mimics certain pathological conditions (Maron & Pelliccia, 2006). For this reason, it is important to use appropriate diagnostic tools that are able to characterize and provide accurate values of athletes' cardiac performance.

Recently, BCG has been proposed as a simple, non-invasive technique to assess cardiac function in athletes (Neary, MacQuarrie, & Busse, 2009), making it possible for athletic teams to screen their players and observe if any remarkable abnormalities appear

in the waveform. If abnormalities are noted, the athlete can be sent for follow-up testing if necessary. Each diagnostic tool has its advantages, but when combined with other screening technology such as BCG, it may offer a more thorough as well as reliable diagnosis (Neary, MacQuarrie, Jamnik, et al., 2009). More importantly, BCG may aid in decreasing costs associated with echocardiography as well as other more expensive diagnostic tests. It also has the potential to help with the diagnostic dilemma that exists today. Furthermore, BCG may aid in preventing sudden cardiac death or the progression of other pathological cardiac conditions and ultimately saving the lives of some athletes.

The development and the science of BCG leading up to the advent of digital BCG, a newly re-developed technology, along with other diagnostic devices will be discussed in a thorough literature review in the next chapter of this thesis. However, prior to introducing the diagnostic tools that have been developed and used to evaluate cardiovascular activity, it is important to have a good understanding of the cardiovascular system, both anatomical and physiological. The structure of the heart plays an important role in its function and adaptations, whether a result of athletic activity or from pathological conditions, can affect cardiovascular performance.

Anatomy of the Cardiovascular System

Chambers. The human heart has four chambers – two upper chambers called atria and two lower chambers called ventricles. The atria are separated by the interatrial septum and the ventricles are separated by the interventricular septum. The heart is divided into right and left halves through the separation of the septum. The left side of the heart distributes blood to the entire body and is referred to as systemic circulation and is responsible for supplying nutrient and oxygen-rich blood to every organ of the body

(Saladin, 2007). The right side of the heart distributes blood to the lungs and is referred to as pulmonary circulation. It is responsible for carrying the blood to the lungs for gas exchange and returns the blood to the heart (Saladin, 2007). The atria receive blood and the ventricles eject the blood through contraction of the myocardium.

Cardiac Valves. To effectively pump blood, the heart needs unidirectional flow valves (Saladin, 2007). Their primary purpose is to prevent regurgitation of blood. Valves open and close depending on the pressure caused from the blood (Chummun, 2009). The heart has four one-way valves (Chummun, 2009; Plowman & Smith, 2003). There are two semilunar valves, aortic and pulmonary, at the ventricular outflows. Both the aortic and pulmonic valves have three flaps and are responsible for controlling the blood flow from the ventricles. Their names foreshadow the opening in which they are situated. The pulmonic valve opens into the pulmonary artery and the aortic valve opens into the aorta. The heart also has two atrioventricular valves which separate the atrium from the ventricle – on the left side of the heart is the bicuspid (or mitral valve) and on the right is the tricuspid valve (Green, 1987; Plowman & Smith, 2003). There are three flaps on the right side and two flaps on the left side.

Heart Wall. The heart wall has three different layers – the epicardium, myocardium, and the endocardium. The external surface of the heart is covered by the epicardium which splits into another external surface called the pericardium. The pericardium is a double-walled sac that holds serous fluid and prevents friction during contraction (Saladin, 2007). The interior heart chambers are lined by the endocardium. The layer that sits between the epicardium and the endocardium is referred to as the myocardium. Myocardium is composed of cardiac muscle and is considered the

contractile muscle of the heart (Plowman & Smith, 2003; Saladin, 2007). The heart wall is primarily composed of cardiac muscle. Its composition is thicker in the ventricles, as they are responsible for contractility and ejection of the blood (Green, 1987). The myocardium/ventricles squeeze the hearts' chambers causing the blood to flow into their respective outlets. The force of contraction is based on Frank-Starling's Law. The greater the venous return, the greater the heart will expand (acting like an elastic band) within limits, and a stronger myocardial muscular contraction will occur (Chummun, 2009).

Physiology of the Cardiovascular System

Electrophysiology. The heart generates electrical activity within each individual cardiac muscle fibre. Ions responsible for this activity include sodium, potassium, calcium, and chloride. The most important ions are potassium and sodium and the concentration differences of these two ions across the cardiac muscle cell's membrane help determine the resting potential, which is between -70 to -90mV (Green, 1987). Contraction of the heart, or depolarization, occurs when there is a change in the resting potential through an increase in permeability of sodium. Return towards resting potential, or repolarization, occurs when there is an increase in potassium permeability. However, hyperpolarization can occur if there is a significant influx of potassium permeability (Green, 1987). The sequence of depolarization and repolarization varies, but tends to follow two patterns. The first pattern is referred to as the pacemaker potential and generates the heartbeat and is located in the sinoatrial and atrioventricular nodes. The sinoatrial node is the electrical impulse-generating tissue known as the pacemaker region, and is located on the posterior part of the heart where the superior vena cava joins the right atrium. The atrioventricular node is part of the electrical system which controls

heart rate. It is located in the septal wall of the right atrium and electrically connects the atrium and ventricle. Most importantly, it delays electrical impulses to ensure that the atria have ejected the blood into the ventricles before contraction (Green, 1987). The second pattern is referred to as the non-pacemaker potential and is found in all other cardiac muscle fibres. The result of the heart's sequenced electrical activity produces what is known as the cardiac cycle (Green, 1987).

Mechanical Function of the Heart

Cardiac Cycle. The cardiac cycle consists of both contraction and relaxation of the heart. Contraction of the heart is referred to as systole and occurs when blood is ejected from the heart. Systole is defined as the segment of the cardiac cycle from the closing of the mitral valve to the closing of the aortic valve and lasts for approximately 250 – 290 milliseconds (ms) (Otto, 2004). Relaxation is referred to as diastole and is when the heart fills with blood. Diastole is the interval from the closing of the aortic valve to the closing of the mitral valve and lasts approximately 500 ms (Otto, 2004). A complete sequence of systole and diastole is what encompasses the cardiac cycle. Several hemodynamic events occur throughout the cardiac cycle (Green, 1987). During ventricular systole, isovolumic contraction and ventricular ejection occur. Isovolumic contraction occurs from mitral valve closure to the opening of the aortic valve and lasts approximately 40 ms (Otto, 2004). Rapid or ventricular ejection is the duration from the opening to the closure of the aortic valve and lasts approximately 80 ms (Otto, 2004). Ventricular diastole consists of four different phases, isovolumic relaxation, the early rapid filling phase, diastasis, and late diastolic filling due to atrial contraction (Berne & Levy, 1986; Otto, 2004). Isovolumic relaxation is the duration between aortic valve

closure and mitral valve opening and lasts approximately 80 to 100 ms. The rapid filling phase begins when the mitral valve opens, allowing blood to flow from the left atrium to the left ventricle. As the ventricle fills, the pressures between the atrium and ventricle equalize and result in a phase called diastasis. Diastasis is the duration in which there is minimal movement of blood between the chambers. Following diastasis, late diastolic ventricular filling occurs during atrial contraction (Otto, 2004).

Stroke Volume. Stroke volume is the amount of blood ejected from the ventricles with each beat. It is determined by various factors that play major roles in cardiac performance (Green, 1987; Plowman & Smith, 2003). These factors include, but are not limited to: (a) the volume of the blood that returns to the heart and the initial stretch of the cardiac muscle (preload); (b) the resistance that the heart must overcome and pump against (afterload); (c) heart rate – the greater the frequency, the lower the stroke volume, and (d) the force of contractility. Stroke volume is greater in highly trained individuals than those who are untrained or sedentary. In untrained individuals, stroke volume plateaus at sub-maximal work rates around 40 – 50% of their VO_2max. This plateau occurs because at high heart rates, the time available for ventricular filling is decreased which results in less end-diastolic volume (Powers & Howley, 2007). In trained endurance athletes, stroke volume continues to progressively increase beyond sub-maximal and up to maximal work rates (Gledhill et al., 1994). Highly trained athletes have improved ventricular filling during heavy exercise because of increased venous return. The greater the venous return, the greater the end-diastolic volume which results in a stronger ventricular contraction and an increase in stroke volume. This physiological

response is known as Frank-Starling's Law of the heart (Chummun, 2009; Powers & Howley, 2007).

Heart Rate. Heart rate represents the number of beats per minute and can vary depending on the body's need for oxygen. During exercise, heart rate increases due to the amount of energy required to perform the activity. Heart rate in resting and maximal exercise conditions varies amongst trained and untrained individuals. At rest the untrained individual's heart rate may be around 72-75 beats per minute versus the trained at 45-55 beats per minute. During maximal exercise conditions the untrained individual's heart rate, which is age dependent, may average around 200 beats per minute whereas the trained is approximately the same or slightly lower at 190 beats per minute (Powers & Howley, 2007). Heart rate is controlled by the sinoatrial node, which is highly influenced by neural innervation from both the parasympathetic and sympathetic divisions of the autonomic nervous system (Green, 1987). The parasympathetic system inhibits activity decreasing the heart rate through the release of the neurotransmitter acetylcholine. The sympathetic system increases heart rate through the release of the neurotransmitter norepinephrine (Chummun, 2009). The release of these excitatory and inhibitory neurotransmitters is what controls and regulates the heartbeat. Both acetylcholine and norepinephrine are considered to be forms of extrinsic stimulators as they reach the heart through the blood (Green, 1987). Other hormones also influence the cardiovascular system. Some of these hormones include adrenocortical hormones, thyroid hormones, insulin, glucagon and anterior pituitary hormones (Berne & Levy, 1986).

Cardiac Output. The total volume of blood released from the left ventricle in one minute is referred to as cardiac output. It is a product of heart rate and stroke volume.

During exercise, cardiac output can increase up to sevenfold to meet the metabolic demands. This increase is primarily attributed to an increase in heart rate. However, stroke volume also contributes through increased venous return caused by the contracting muscles during exercise (Charlton & Crawford, 1997). Training has the potential to increase stroke volume (at both rest and during exercise), thus a lower heart rate is needed to maintain cardiac output (Charlton & Crawford, 1997). Cardiac output can also be influenced by factors that include emotion, stress, alcohol, and chemicals such as drugs. Each factor has the potential to influence peripheral resistance, heart rate and stroke volume (Chummun, 2009).

Summary

The structure and the function of the heart can adapt in response to regular exercise training. However, as stated the adaptations that occur depend on the type and frequency of exercise and whether predominately static or dynamic exercise (or a combination of the two) was performed. Athletes who have undetected cardiac conditions or those who undergo extreme cardiac adaptations (e.g., hypertrophic cardiomyopathy) in response to exercise training may be putting their life at risk when engaging in physical activity. Cardiovascular conditions are a leading cause of death in society and can also affect the athletic population. Many cardiovascular conditions are preventable and reversible through proper screening and timely interventions. However, screening and assessment, particularly mass screening of athletes, is highly dependent on the non-invasive techniques that are available and technical ability to objectively qualify acquired data (e.g., images).

Current diagnostic tools have certain advantages but when used in conjunction with other techniques such as BCG, may offer a more thorough as well as reliable diagnosis to the patient (Neary, MacQuarrie, Jamnik, et al., 2009). More importantly, BCG may aid in decreasing costs associated with echocardiography as well as other more expensive diagnostic tests (e.g., cardiac MRI). It has the potential to accurately characterize the heart and recognize cardiac abnormalities, thus can become an important screening tool for evaluating cardiac function. The history of BCG leading up to the advent of digital BCG, a newly re-developed technology, along with a brief overview of other more commonly used diagnostic devices will be discussed throughout the following review.

CHAPTER I – USING BALLISTIOCARDIOGRAPHY TO MEASURE CARDIAC PERFORMANCE: A BRIEF REVIEW OF ITS HISTORY AND FUTURE SIGNIFICANCE

ABSTRACT

Ballistocardiography (BCG) is a non-invasive technology that has been used to record ultra-low frequency vibrations of the heart allowing for the measurement of cardiac cycle events including timing and amplitudes of contraction. Recent developments in BCG have made this technology simple to use, as well as time- and cost-efficient in comparison to other more complicated and invasive techniques used to evaluate cardiac performance (i.e., echocardiography, positron emission tomography scan, radionuclide (MUGA) angiography scans). Recent technological advances are considerably greater since the advent of microprocessors and laptop computers. In this paper the history of BCG is reviewed. As well, the present and future potential benefits of using BCG to measure cardiac cycle events, and its application to clinical and applied research are discussed.

INTRODUCTION

Cardiovascular disease is a leading cause of death in society. It is both a preventable and a reversible condition through proper screening and timely interventions. However, screening and assessment is dependent on the non-invasive techniques that are available and technical ability to objectively analyze acquired data (e.g. images). Each method used to screen the heart has its advantages, but not all of these tools are readily accessible and can often be very expensive and time consuming.

Throughout history several tools have been developed and used to evaluate cardiovascular function. In 1899, enlargement of the heart caused by athletic activity was recognized by performing a basic physical examination with careful percussion (Maron & Pelliccia, 2006; Rost, 1997). Following the discovery of chest radiography, echocardiography and computed tomography scanning were introduced and confirmed these findings (Maron & Pelliccia, 2006; Rost, 1997). Echocardiography has been the primary diagnostic tool used in many studies to examine the heart and more recently, cardiac MRI, has been used to show superior imaging scans (Maron & Pelliccia, 2006; Sharma et al., 2002). ECG is also utilized, as it is able to detect abnormalities in cardiovascular function (Maron, 1997).

Recently, it has been suggested that ballistocardiography (BCG) or seismocardiography (SCG), a measure of the seismic activity of the heart, may provide additional information to characterize cardiac performance or function in a practical and cost-efficient manner (MacQuarrie, Gebhardt, & Neary, 2011). BCG is a newly re-developed technology that has potential to contribute to the existing procedures that are used to examine the heart. Where ECG represents the electrical activity generated by the heart, BCG represents the mechanical function of the heart. Thus, it has been suggested that BCG provides an advantage over the basic ECG as it can determine all timing events and contractile forces of the cardiac cycle (Pinheiro, Postolache, & Girao, 2010). Even before advancements in BCG technology had progressed (with advanced computer and micro-processor technology), it was discovered that despite the presence of cardiovascular disease the ECG appeared to be normal, meanwhile the BCG was abnormal (Phibbs, Lowe, & Holmes, 1967). Furthermore, it was discovered that the ECG

was unable to detect any abnormalities during the primary stages of disease. However BCG, a more sensitive technique of cardiac adaptations, can detect abnormalities during the early stages of disease (Lowenstein, Arbeit, & Rubin, 1962).

Each diagnostic tool has its advantages but when combined with other techniques such as BCG, it may offer a more thorough as well as reliable diagnosis to the patient (Neary, MacQuarrie, Jamnik, et al., 2009). More importantly, BCG may aid in decreasing costs associated with echocardiography as well as other more expensive diagnostic tests. It has the potential to accurately characterize the heart and recognize cardiac abnormalities, thus can become an important screening tool for evaluating cardiac function. A recent study by Pinheiro et al. (2010) described the main characteristics of BCG waveforms and analysis and therefore will not be discussed in detail here. The history of BCG leading up to the advent of current research using BCG, along with a brief overview of other cardiac diagnostic devices will be discussed in this review.

Diagnostic Devices for Cardiac Performance

With the evolution of time and advancements in cardiovascular physiology, new and advanced diagnostic tools have been developed to evaluate cardiovascular function because of disease. The most commonly used diagnostic tools in examining cardiovascular performance are summarized in Table 1. Other diagnostic devices include the single average ECG, stress test, transesophageal echocardiogram, thallium scans or myocardial perfusion scan, holter monitor, and coronary angiogram (Bigi & De Chiara, 2005; Brandes & Bethge, 2008; Clark & Beller, 2005; Desjardins & Cahalan, 2009; Green, 1987; Grubb, Temesy-Armos, Hahn, & Elliott, 1991; Wijeysundera, Beattie, Austin, Hux, & Laupacis, 2010). These tests have been used either for daily monitoring

Table 1

Summary of diagnostic devices used to assess cardiac performance

Device		Description
Electrocardiography	(Green, 1987)	A simple and useful diagnostic technique that can evaluate the electrical activity of the heart through the measurement of the potential difference between two points on the body using electrodes.
Echocardiography	(Otto, 2004)	A non-invasive test that uses piezoelectric crystal ultrasound transducers which both generates and receives ultrasound waves, creating an image of the heart's motion in the chambers and valves.
Cardiac MRI	(Pennell, 2010)	A technique that creates images from atomic nuclei with uneven spin using radiowaves in the presence of a magnetic field. Evaluates the heart and blood vessels.
Positron Emission Tomography Scan	(Heller, Calnon, & Dorbala, 2009)	Perfusion imaging is performed using several available radiotracers and is captured by camera technology. Evaluates the function and blood flow to the heart.
Radionuclide (MUGA) Angiography Scans		Involves an injection of an imaging agent followed by a resting scan. Primarily performed to determine the heart's ejection fraction.

of cardiac function or for very specific cardiac anomalies performed in the emergency room or catheterization laboratory. Echocardiography, ECG, and cardiac MRI are some of the most common tools to assess and measure cardiac performance. These tools have played an important role in diagnosing cardiac function, both in the past and present, and will be discussed in detail below.

Echocardiograph. The development of echocardiography began in 1842 when Christian Doppler, an Austrian researcher who studied astronomy, came across ideas that eventually became important principles of ultrasound diagnostics. In 1954, the first recording of cardiac events using ultrasound was performed. However, clinical use of Doppler echocardiography did not occur until the late 1970s. Primarily, Doppler echocardiography has two functions. Its original function was to determine blood flow velocities, direction, and characteristics in the heart and great vessels. Another more recent function is to determine the velocity of myocardial tissue wall motion using Doppler imaging. Through the determination of blood flow velocities, Doppler echocardiography allows various hemodynamic evaluations both in the heart and the vascular system (Anavekar & Oh, 2009). As well, it has the ability to observe and determine timing events of the cardiac cycle (MacQuarrie et al., 2011; Otto, 2004).

Presently, echocardiography is one of the most commonly used diagnostic tools for cardiovascular disease. However, it is expensive and less practical for mass assessment, although it is commonly used in some European countries to assess their athletes (Pelliccia & Maron, 1995). As a result, twelve-lead ECG has been proposed as a more cost-effective and practical method for screening the heart (Corrado et al., 2009; Corrado et al., 2005).

Electrocardiograph. ECG is a simple and useful diagnostic technique commonly used to evaluate the electrical activity of the heart through the measurement of the potential difference between two points on the body (Green, 1987). It has the potential to detect underlying cardiovascular conditions including abnormalities such as arrhythmia, early repolarization, hypertrophic and dilated cardiomyopathy, coronary artery disease and various other electrophysiological conditions (Corrado et al., 2009; Corrado et al., 2005). Changes in ECG patterns commonly occur in up to 80% of trained athletes as a result of regular exercise training (Holly, Shaffrath, & Amsterdam, 1998). Adaptations to the cardiac autonomic nervous system, such as increased vagal tone and/or withdrawal of sympathetic activity contribute to ECG changes (Holly et al., 1998).

It is important that ECG abnormalities caused by exercise training and those possibly associated with cardiovascular disease are properly identified as additional testing to confirm the presence of pathology may be necessary. Le et al. (2010) illustrated the effectiveness of using ECG in determining athletes at risk for cardiac abnormalities. 653 athletes were subjected to a resting ECG test during pre-season medical exams. 63 of these athletes were determined to have abnormal ECG findings suggestive of a variety of cardiac conditions. They also indicated the need for a more cost-effective screening tool in the athletic setting. ECG abnormalities are quite common in trained athletes, but fortunately the incidence of fatal cardiovascular disease in young trained athletes is extremely rare (less than 1%) (Pelliccia & Maron, 1995). Therefore, there is an on-going debate as to whether or not mass screening is worth the associated expense to detect the small number of athletes at risk (Pelliccia & Maron, 1995). Although ECG is one of the most important and common tools used in cardiology today, the logistics of screening

large numbers of individuals remains a concern (Le et al., 2010; Neary, Len, MacQuarrie, Jamnik, et al., 2008), and not all cardiac conditions are detected using ECG (Maron, 2009).

Cardiac MRI. The concept of cardiac MRI began in the 1970s through experimental research with iodinated contrast media and prototype cardiac computed tomography scanners. Throughout the 1970s and 1980s it was perceived that echocardiography and nuclear imaging provided the most information of non-invasive cardiac imaging (Higgins, 2009). During the early 1980s the first cardiac magnetic resonance images were published (Alfidi et al., 1982). Cardiac MRI allows for a complete and non-invasive evaluation of cardiac function, morphology and tissue characterization (Germans & van Rossum, 2008). It is able to provide information on both anatomical and physiological abnormalities of the cardiovascular system (Herfkens et al., 1983). As well, cardiac MRI provides superior image quality in comparison to echocardiography.

The use of cardiac MRI to evaluate the athletic heart is increasing (Prakken, Velthuis, Cramer, & Mosterd, 2009). It can help identify the presence of heart conditions such as hypertrophic cardiomyopathy, coronary artery anomalies, and myocarditis amongst several others (Prakken et al., 2009). However, there are contraindications to its use which include but are not limited to metal devices, pacemakers, claustrophobia, and radiation exposure (Prakken et al., 2009). Furthermore, the differentiation between physiological adaptations and cardiovascular disease is difficult as there is such a great overlap. For example, 2% of athletes have left ventricular wall thickness of 13-15 mm, and 15% have left ventricular cavity enlargement of 60 mm or more, showing overlap

with mild cardiomyopathy (Maron, 2005; Prakken et al., 2009). This emphasizes the importance of establishing specific reference values to help determine athletes at risk. However, the practicality of performing mass screening using cardiac MRI is low as it is a costly and time consuming diagnostic technique (Karamitsos, Arnold, Neubauer, & Petersen, 2007).

Displacement Cardiography

Many researchers and clinicians have demonstrated an interest in the study of cardiac vibrations, which are believed to be caused by myocardial wall motion (Crow, Hannan, Jacobs, Hedquist, & Salerno, 1994). The mechanical movements of the heart during diastole and systole cause vibrations that travel toward the sternum where they can be recorded by an accelerometer. The instrumentation to record mechanical function through cardiac vibrations is referred to as displacement cardiography. Several displacement cardiography devices have been developed over the years and are summarized in Table 2.

Ballistocardiography. The BCG is a non-invasive technique used to evaluate cardiovascular function (Alametsa, Viik, Alakare, Varri, & Palomaki, 2008; Eblen-Zajjur, 2003; Pinheiro et al., 2010; Scarborough, 1955; Starr, 1965). Today, it is simple and inexpensive in comparison to other more complicated, expensive and invasive techniques previously discussed, such as echocardiography or cardiac catheterization (Eblen-Zajjur, 2003). BCG studies the motion of the body with each heartbeat. The motion is thought to arise from contraction of the heart as well as from ejection and

Table 2

Summary of displacement cardiography devices

Device	Description
Apexcardiograph	Places a pressure transducer over the apex of the heart.
Kinetocardiograph	Places a cylinder over the sternum to detect movement of the air column within the cylinder by a pressure transducer placed on top of the cylinder. Device is very complex and sensitive, as a result is limited in its ability to accurately capture and record events that occur throughout the cardiac cycle.
Ballistocardiograph	Uses an accelerometer to detect motion of a platform in response to motion of the body due to cardiac activity.
Seismocardiograph	Uses a sensitive piezoelectric accelerometer to identify low-frequency vibrations through placement on the lower sternum.
Cardiokymograph	Uses a capacitor to induce an electromagnetic field that penetrates the chest wall near the apex and detects distortion of the area due to cardiac movement.
Phonocardiograph	Uses a chest microphone or a miniature sensor that is introduced to the blood vessels and into one of the heart chambers. Creates a graphic record of the sounds/murmurs produced by contraction of the heart, including its valves and associated great vessels.
Impedance Cardiography	The placement of four disposable electrodes is used to transmit and detect electrical and impedance changes in the thorax. These impedance changes can be used to measure and calculate hemodynamic parameters.

Note: All information presented in Table 2 is adapted from Salerno et al. (1991) and Crow et al. (1994).

movement of the blood toward the periphery (McKay, Gregson, McKay, & Militzer, 1999; Pinheiro et al., 2010). During systole when ejection of the blood into the great vessels occurs, the center of gravity of the blood moves towards the head of the body. As the blood moves towards the periphery and accumulates further away from the heart in the peripheral vessels, the center of gravity moves towards the feet (Noordergraaf, 1961; Starr, 1965). This shift consists of components attributable to cardiac activity (circulatory events), respiration, and body movement (Alametsa et al., 2008). A BCG waveform is created from this shifting of the centre of mass of the body, as the blood distribution changes throughout the cardiac cycle (Starr, 1965). The waveform represents different events and phases that occur as a result of the body motions produced during cardiovascular action (Alametsa et al., 2008; McKay et al., 1999; Pinheiro et al., 2010).

In 1877, the ballistic displacements of the body were studied and first published (Gordon, 1877). A subject was placed on a bed suspended by ropes from the ceiling (pendulum bed) and its motion was recorded synchronously with the subject's heartbeat. However, its measurements were impractical and inaccurate. The amplitude of the movement of the bed caused by respiration was greater than that caused by circulatory events (Alametsa, Varri, Viik, Hyttinen, & Palomaki, 2009). In the 19th century, BCG was re-discovered. The majority of the early devices constructed tracked movement using a special bed (McKay et al., 1999). In 1939, Starr developed a BCG, a modification of the pendulum bed. His bed was suspended by cables from the ceiling, constrained lateral movements, and recorded the displacement of the bed. Adjustments were made to his design to minimize the effect of respiration. The Dock direct body BCG was made by

attaching one end of a transducer to a concrete block and the other end to a wooden bar clamped across the subject's shins (Dock & Taubman, 1949). However, it became clear that Isaac Starr's design and the Dock direct body system among other designs were all unacceptable. The manner in which the body was supported interfered with the recording. Their devices were cumbersome and required regular maintenance (Dock & Taubman, 1949). From 1952 to 1965, there were several advancements in the instrumentation of the BCG and in its theory. Starr (1955, 1965) had discovered that BCG signals were an indication of the strength of myocardial contraction. In particular, the application of Newton's laws of mechanics to estimate the strengths or weaknesses of cardiac performance (Starr, 1965).

The main focus of BCG is the observation of ballistic effects such as force, impact, velocity and momentum patterns of the body and blood, as well as respiratory displacement (Talbot, 1958). The heart is responsible for the cause of mechanical forces that are produced throughout the cardiac cycle. Through contraction of the myocardial wall, certain forces are generated which produce very powerful acceleration and deceleration forces (Phibbs et al., 1967). The primary goal in the development of the BCG was to apply Newton's laws of mechanics to cardiovascular function (Starr, 1965). The contraction of the heart causes motion, a concept accurately described by Newton's laws. In particular, Newton's third law of motion, in which it states that for every acting force, there is an equal and opposite reacting force. Displacement, velocity and acceleration are all important aspects of motion (Starr, 1965). Of these three, acceleration (of ejected blood) is the best predictor of cardiac function. Acceleration can detect the beginning of weakness, whereas displacement (cardiac output) cannot detect problems

until they have already occurred (Starr, 1965). Therefore, the acceleration of blood is the most important aspect to study and can be observed through the forces generated, as force is equal to mass times acceleration ($F = m \cdot a$). Common sense would indicate that any degeneration of the myocardium would likely result in a change of magnitude and characteristics of the forces generated (Phibbs et al., 1967). It is impossible to determine the effect of the activity in the right and left ventricle as both sides of the heart contribute to the BCG. The recorded waveforms are not a pure force, but rather the resultant of vectors (Gubner, Rodstein, & Ungerleider, 1953). For example, if one were to lay perfectly still in the supine position with controlled breathing and place a glass of fine wine on the sternum of the chest, in time the fine wine would oscillate within the glass in rhythm with the flow of blood through the heart and cardiovascular system. The resulting waveform created corresponds to the BCG.

The BCG was studied extensively up until the late 1960s. The interest in the heart as a muscle slowly declined as an appeal towards the ECG developed (Starr, 1965). ECG is far less complicated than BCG. However, unlike BCG, it is unable to evaluate the force of the heart's contractility.

ECG is a relatively simple procedure that observes the electrical activity of the heart (Green, 1987). One advantage of ECG is that electrical signals unrelated to the heart can be avoided. With BCG it is difficult to avoid unrelated forces, such as coughing (Talbot & Harrison, 1955). Alongside recordings of cardiovascular function, the BCG also captures respiratory and body movement related to motion (Eblen-Zajjur, 2003). Consequently, muscular movements or vibrations from other sources can interfere in obtaining an accurate recording (Starr, 1965).

It is imperative to provide detailed and specific instructions prior to testing, stating that it is important to relax, breathe normally and avoid muscular movements (Eblen-Zajjur, 2003). Gubner et al. (1953) state that the BCG waveform will be poor unless the subject is completely relaxed. It is for this reason it is difficult to record a BCG in an upright position. Extra movement may also occur in the elderly, as they may find it difficult to remain in the same position (Gubner et al., 1953). However, non-myocardial vibrations such as body and respiratory movement can be eliminated by frequency filtering (Bombardini, Gemignani, Bianchini, Venneri, Petersen, Pasanisi, Pratali, Pianelli, et al., 2008).

BCG is also able to provide more information than ECG. In 1967, Phibbs et al. discovered that despite the presence of cardiovascular disease the ECG appeared to be normal, meanwhile the BCG was abnormal. Lowenstein et al. (1962) found that the ECG is unable to detect any abnormalities during the primary stages of disease. The BCG, a more sensitive device to record the mechanical function of the heart, can detect abnormalities during the early stages of disease. Researchers have suggested that the BCG is a useful measurement device for clinicians in determining fundamental aspects of the heart's function (MacQuarrie et al., 2011; Rosenblatt, 1957). Due to the cumbersome nature and maintenance required of early models, the BCG had difficulty maintaining its status as an effective tool for analyzing cardiac function (Inan, Etemadi, Paloma, Giovangrandi, & Kovacs, 2009). As computer and micro-processor technology has progressed, these concerns were addressed and the consistency of the device has improved.

Those individuals without past or present cardiovascular abnormalities have a fairly consistent and normal BCG waveform (Alametsa et al., 2008). The standard for a normal BCG waveform was determined by the waveforms produced in young, healthy individuals (Baker, 1968). Strong contractility produces large waveforms, whereas weak contractility results in abnormal and small waveforms (Scarborough et al., 1952). The difference observed between waveforms is due to the condition of the cardiovascular system (e.g., healthy versus a diseased heart) (Alametsa et al., 2008; Pinheiro et al., 2010). Both age as well as participation in physical activity can also have an effect on the amplitude of BCG waveforms and the timing of cardiac cycle events (Alametsa et al., 2008). Typically those over the age of 50 display abnormal BCG waveforms (e.g., decrease in waveform amplitude) even in the absence of cardiovascular abnormalities (Rosenblatt, 1957).

Abnormal waveforms, related to cardiac disease, diabetes, and obesity are difficult to identify and annotate their specific cardiac events. There is no consistency between waveforms (e.g., timing of cardiac cycle events) and evaluation becomes extremely challenging. To aid in properly annotating the BCG waveform, a simultaneous recording of an ECG can be used. Through the attachment of a lead(s), it is simple to synchronously record the BCG as well as ECG. The QRS complex of the ECG is recorded just before systolic contraction and provides a reference to help with analyzing the BCG (Gubner et al., 1953). A recent study by Pinheiro et al. (2010) described the main characteristics of BCG waveforms and analysis and therefore will not be discussed in detail here.

Seismocardiography. The term SCG was established in the 1960s replacing the term BCG (Dock & Zoneraich, 1967). In 1964, an accelerometer was attached directly to the skin over the sternum to study the motion of the heart, and thus the process was referred to as SCG (Bayevski, Egerov, & Kasarjan, 1964). It is a measure of the acceleration caused by the movement of the heart (Poliac, Zanetti, & Salerno, 1991). The SCG is collected non-invasively through an ultra-low frequency transducer placed on the chest. A single-channel ECG is then simultaneously recorded. SCG is able to record very-low-frequency compression waves that are transmitted to the surface of the thorax (Crow et al., 1994). These compression waves are primarily produced by both myocardial wall motion and blood flow (Zanetti, Poliac, & Crow, 1991).

SCG differs from other cardiography instruments as several improvements were made to help better record low-frequency vibrations of the heart. Some of these improvements included: (a) a more sensitive piezoelectric accelerometer; (b) the use of modern microprocessors and the most important improvement was (c) its simplicity to use and interpret (Crow et al., 1994). SCG is able to provide a view of the mechanical function of the heart through the summation of forces generated by each cardiac event (Crow et al., 1994). SCG is both a convenient and reliable measure of cardiac function both at rest and immediately after exercise (Zanetti et al., 1991). Like BCG and other displacement cardiographs, SCG must be measured under controlled resting conditions to accurately record cardiac cycle waveforms.

Validity of Displacement Cardiography

Neither SCG or BCG technology have gained widespread clinical use because of technological limitations including analysis of waveforms and non-myocardial movement

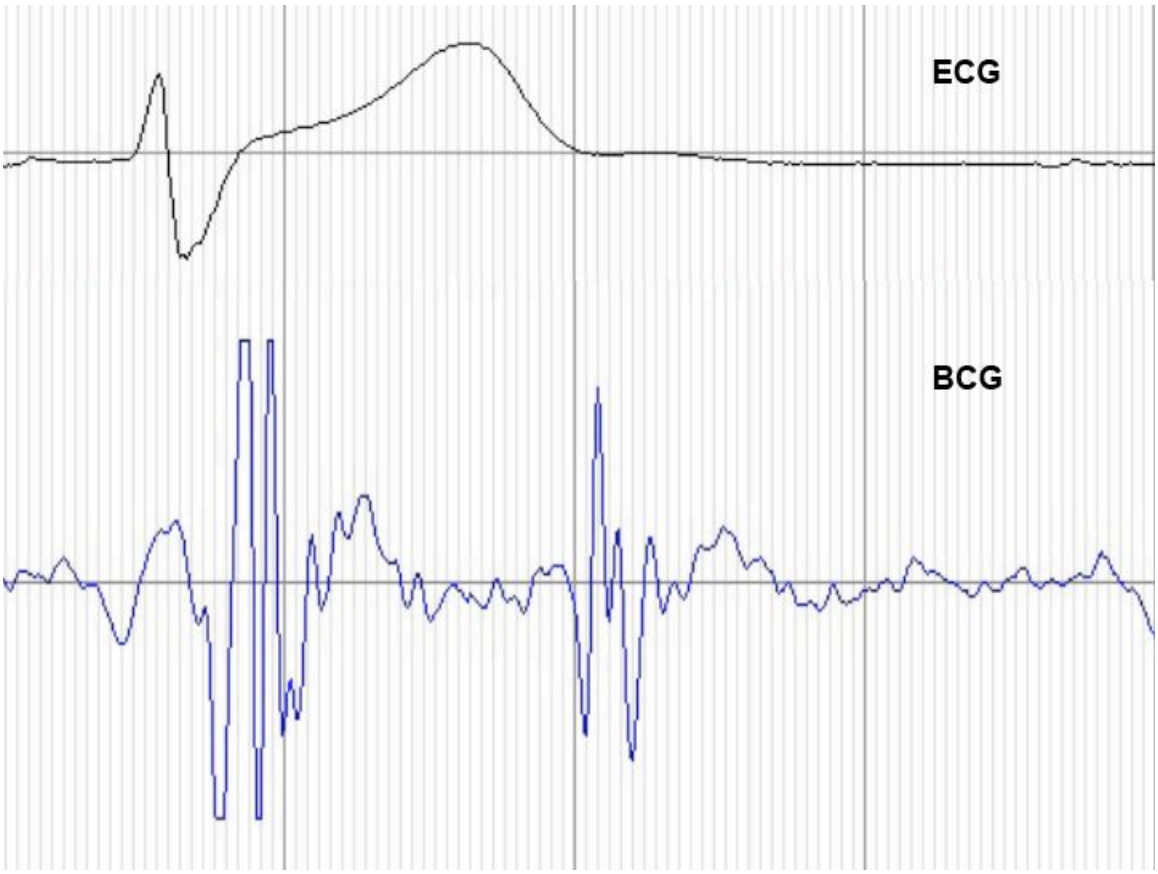

Figure 1. Illustration of simultaneous electrocardiogram (ECG) and the ballistocardiogram (BCG) waveform.

as well as other competing devices such as echocardiography and ECG (Ngai et al., 2009). Although there are differences between the various methods used to evaluate cardiac performance there are also many commonalities. Figure 1 provides a graphical comparison of an ECG and a BCG waveform. Ngai et al. (2009) also provide a side-by-side comparison of ECG, BCG and SCG. Before a diagnostic tool such as BCG and SCG can be considered a valid means to measure cardiac performance, it must be validated against an established norm. Zanetti et al. (1991) observed whether cardiac events would coincide using SCG, ECG, M-mode and pulsed Doppler echocardiography. It was determined that the agreement between SCG and echocardiography was very good. For systolic SCG points, the mean absolute difference from echocardiography for all subjects was 7.9 ± 8.1 ms, and for diastolic SCG points the mean absolute difference was 11.8 ± 11.5 ms.

Later, Crow et al. (1994) evaluated the relationship between SCG and echocardiography and found that they were both equally accurate in measuring cardiac time intervals. They determined that SCG was just as reliable as echocardiography as both methods were able to identify the presence of longer cardiac intervals in subjects with cardiomyopathy. Among all systolic measurements except mitral valve closure, the SCG point occurred slightly earlier than the echocardiography point. This is likely the reflection of the fact that echocardiography measures flow, and SCG measures motion. The mean absolute difference for systolic points was 7.6 ms. SCG preceded the echocardiography point for all diastolic measurements. The mean absolute diastolic difference was 6.8 ms.

Furthermore, previous unpublished research from our laboratory has shown BCG was found to be a reliable method to monitor differences in the cardiac cycle over a period of days (Neary, MacQuarrie, & Busse, 2009). Additional research has also been performed to compare BCG with echocardiography to determine whether BCG could provide a valid measure of the timing events of the cardiac cycle (MacQuarrie et al., 2011), and support previous research by Crow et al. (1994) and Zanetti et al. (1991). The results showed that the timing events of the cardiac cycle were not significantly different between BCG and echocardiography, and that BCG was simple to use, and can monitor cardiac performance both in an applied and clinical setting.

Digital Ballistocardiography

Digital BCG is a new technology that is based on the BCG developed in the 19th century, as well as advancements that have been made to SCG. The technology consists of a sensor, a digitizing transceiver unit, as well as proprietary software used to capture and analyze the ballistocardiogram (Figure 2). The technology generally uses a highly sensitive accelerometer with a frequency range of 0.1 Hz to 4 kHz and a dynamic range that is linear (± 2%) ranging from 0.05 mm/s^2 to 20 km/s^2 to measure the low-frequency vibrations caused by the contraction of the heart. The BCG accelerometer captures the cardiac forces in three waveforms – each one representing an anatomical axis (x-axis measures from head to foot; y-axis depicts right to left; z-axis measures back to front). More specifically, the accelerometer captures the seismic forces present at the sternum of the chest, which is caused by myocardial contraction. These vibrations are processed digitally – the transceiver unit digitizes and transmits the recorded data using Bluetooth® technology to connect to proprietary software on a laptop

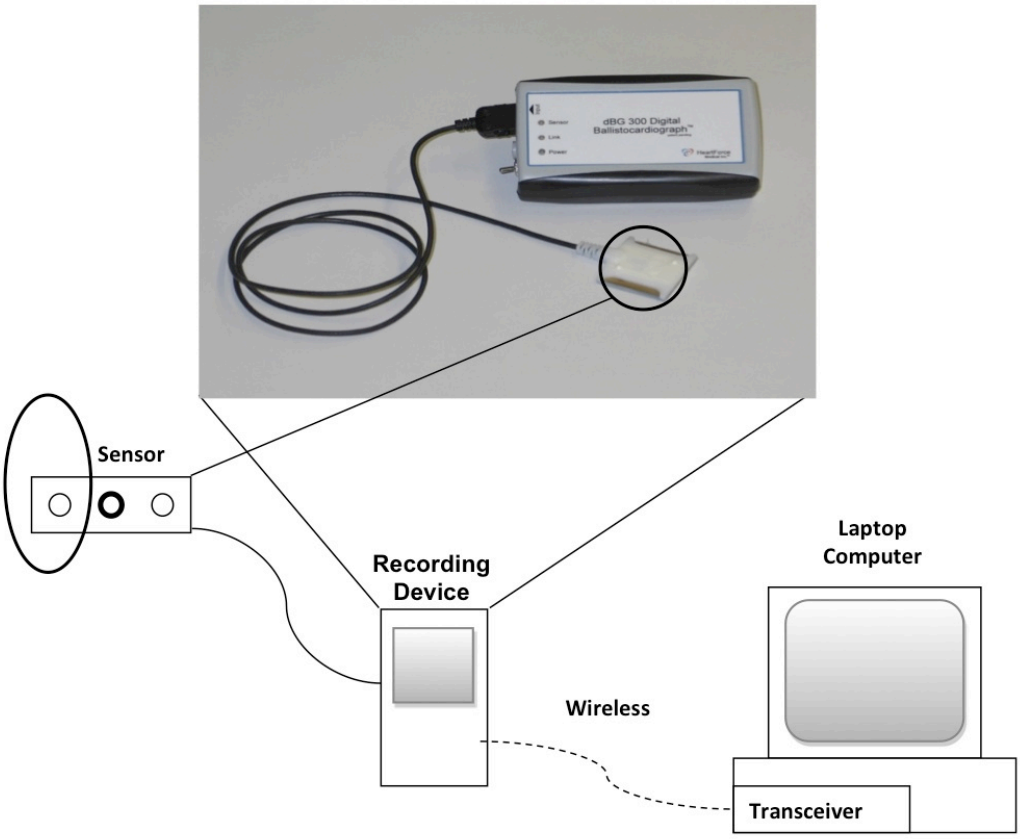

Figure 2. Schematic of a typical digital ballistocardiography setup.

computer. All recorded data is analysed off-line using the proprietary software, which allows for annotation and analysis of recorded cardiac cycle waveforms. BCG can also be designed to simultaneously capture the heart's electrical activity producing an ECG QRS complex (Bombardini, Gemignani, Bianchini, Venneri, Petersen, Pasanisi, Pratali, Alonso-Rodriguez, et al., 2008; Bombardini, Gemignani, Bianchini, Venneri, Petersen, Pasanisi, Pratali, Pianelli, et al., 2008). Figure 1 provides an illustration of a simultaneous ECG and BCG recording.

In addition to the timing events (measured in milliseconds) of the cardiac cycle, it is possible to record the acceleration amplitude (measured in milligravity, mG) that can be used as a surrogate for cardiac contractility (Neary, MacQuarrie, & Busse, 2010). In comparison to devices developed in the 20th century, extensive improvements have been made because of microprocessor technology. Presently, BCG technology can collect data from three anatomical planes (tri-axial) opposed to only using a single-axis accelerometer (see Figure 3). A tri-axial plane opposed to a single-axis accelerometer is more precise when it comes to annotating the ballistocardiogram. Three axes provide confirmation for where each cardiac cycle event occurs. An algorithm for automated analysis is still in the process of being developed. However, it is proposed that BCG will be a useful asset for clinicians and complement other diagnostic assessment tools (Bombardini, Gemignani, Bianchini, Venneri, Petersen, Pasanisi, Pratali, Alonso-Rodriguez, et al., 2008).

Devices such as echocardiograms and MRI are costly, time consuming, and require a knowledgeable technician to use. On the contrary, BCG is simple, extremely portable and does not require extensive training to operate. BCG is a potential tool to investigate cardiac function in a variety of settings. It is useful for assessing cardiac

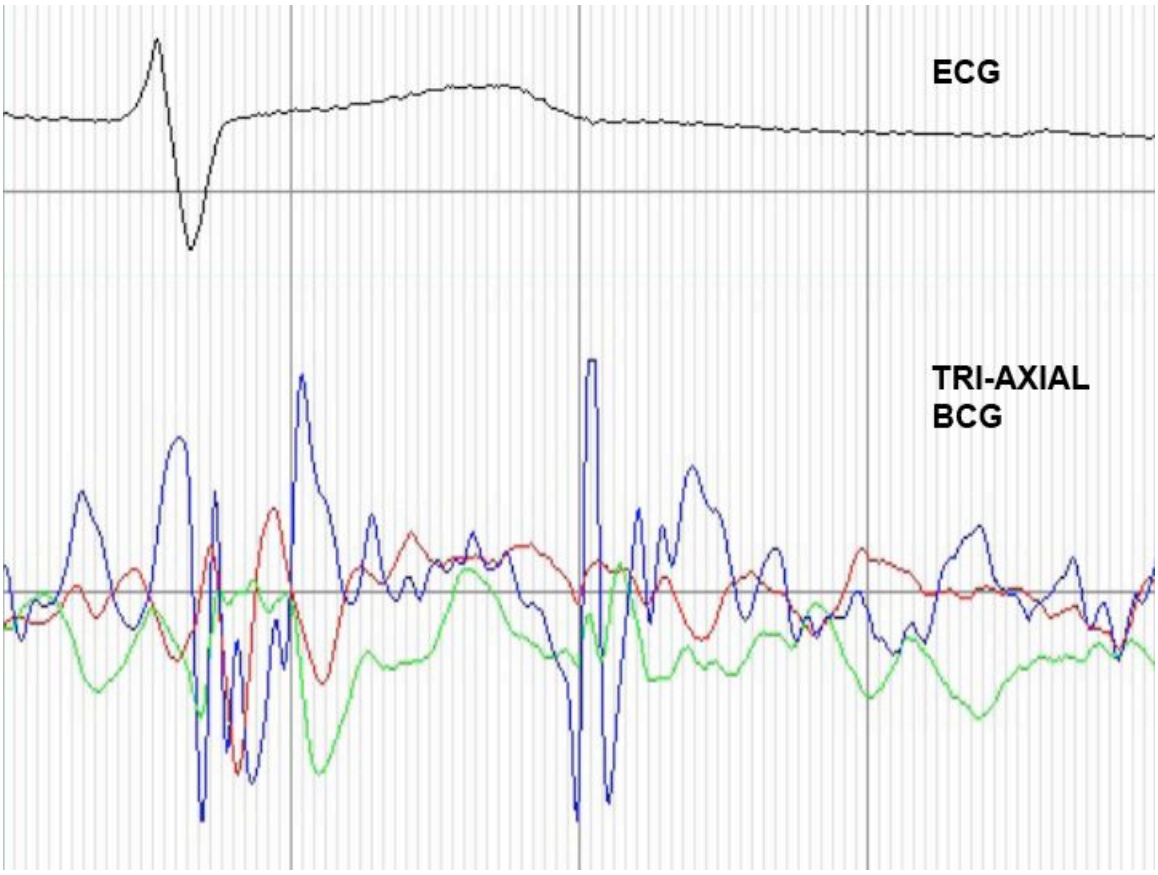

Figure 3. Illustration of a simultaneous electrocardiogram (ECG) and tri-axial ballistocardiogram (BCG) recording. Red, x-axis (head to foot); green, y-axis (shoulder to shoulder); blue, z-axis (anterior to posterior chest). Original in colour.

physiology in cardiology practice or clinical settings, as well as for investigation in sports and training settings. A number of unpublished studies presented in abstract form have been conducted to demonstrate the utility of using BCG to characterize cardiac performance (Neary, Len, Busse, MacQuarrie, & Goodman, 2009; Neary, Len, MacQuarrie, & Busse, 2008; Neary, Len, MacQuarrie, Jamnik, et al., 2008; Neary, MacQuarrie, & Busse, 2008; Neary, MacQuarrie, Jamnik, et al., 2009; Vogt, Neary, MacQuarrie, Len, & Busse, 2009).

Future Direction

Today, BCG has evolved into a digital technology that enables several different uses (Bombardini, Gemignani, Bianchini, Venneri, Petersen, Pasanisi, Pratali, Alonso-Rodriguez, et al., 2008; Bombardini, Gemignani, Bianchini, Venneri, Petersen, Pasanisi, Pratali, Pianelli, et al., 2008; Pinheiro et al., 2010; Vogt et al., 2009; Vogt, Neary, MacQuarrie, & Len, 2010). The BCG is primarily a technology that complements other procedures that examine cardiac performance (Gubner et al., 1953). In the past, there was no way to electronically or digitally analyze the BCG waveform, as it had not yet been discovered (McKay et al., 1999). Researchers lacked reliable comparison measures, such as angiography or echocardiography (Crow et al., 1994; McKay et al., 1999). To date, the timing and cardiac events observed using BCG have been digitally analyzed using proprietary software and compared to echocardiograms. It has been found that when comparing the labelling of the various cardiac events between both BCG and echocardiography, the analysis indicates that BCG provides a reliable measure of cardiac performance (Crow et al., 1994; MacQuarrie et al., 2011; Zanetti et al., 1991). As a result, it is evident that the technology surrounding BCG has significantly improved and

has increased its potential as an effective screening tool for cardiac performance in the future. Furthermore, BCG technology has been used recently to successfully conduct clinical research studying both healthy and diseased populations as well as for the evaluation of athletes (Bombardini, Gemignani, Bianchini, Venneri, Petersen, Pasanisi, Pratali, Alonso-Rodriguez, et al., 2008; Bombardini, Gemignani, Bianchini, Venneri, Petersen, Pasanisi, Pratali, Pianelli, et al., 2008; Neary, Len, et al., 2009; Neary, Len, MacQuarrie, & Busse, 2008; Neary, Len, MacQuarrie, Jamnik, et al., 2008; Neary, MacQuarrie, & Busse, 2009; Neary, MacQuarrie, et al., 2008; Neary, MacQuarrie, Jamnik, et al., 2009; Vogt et al., 2009; Vogt et al., 2010).

Conclusion

Recent developments in micro-technology have allowed BCG and SCG technology to re-surface and have provided researchers with the capacity to gain greater insights into cardiac function and performance. The potential applications for this technology not only include the medical, clinical and physiological settings, but also the athletic setting as a potential screening device for cardiac complications. As research utilizing BCG continues, future research will only aid in developing and refining the technology further.

CHAPTER II – EVALUATING CARDIAC CYCLE TIMING EVENTS IN MALE ICE HOCKEY PLAYERS USING BALLISTOCARDIOGRAPHY

ABSTRACT

Ballistocardiography (BCG), an emerging technology, measures ultra-low frequency vibrations of the heart and has the potential to screen for cardiac abnormalities. The purpose of the present study was to use BCG to record and compare the differences in the timing of cardiac cycle events in highly trained ice hockey players versus a recreationally active control group. Participants included 24 highly trained ice hockey players (aged 18.0 ± 1.3 years) and 20 recreationally active presumably healthy young adults (aged 22.5 ± 2.4 years) who were not training for any particular sport or event. Data collection occurred under quiet resting conditions for 30 seconds using BCG. Data were analyzed off-line using proprietary software designed to compute the timing and amplitude of cardiac cycle events. *t*-tests were performed to investigate the differences in timing of cardiac cycle events. Significance was set at $p < .05$. Timing differences were observed between the trained and untrained groups and significant events included isovolumic contraction (55.1 ± 8.6 vs. 52.8 ± 8.7 ms) and relaxation time (111.4 ± 16.6 vs. 100.4 ± 13.8 ms), pressure half-time (58.5 ± 11.2 vs. 54.6± 8.2 ms), early diastolic filling to atrial systole (498.9 ± 168.9 vs. 469.1 ± 142.8 ms), and systole (226.4 ± 22.9 vs. 230.2 ± 15.5 ms). Diastole was not different between groups. It was concluded that BCG is able to detect differences in cardiac timing events and can easily be used for mass testing of athletes and thus used as a potential screening device.

INTRODUCTION

Research using cardiovascular diagnostic devices (e.g., echocardiography) to assess the athletic heart has discovered that athletes experience adaptive improvements in cardiac function (e.g., increased stroke volume, cardiac output, and VO_2max) alongside changes to the timing of cardiac cycle events (Di Bello et al., 1996; Erol & Karakelleoglu, 2002; Fazel et al., 2009; Libonati, 1999; Libonati, Colby, Caldwell, Kasparian, & Glassberg, 1999; Nixon, Wright, Porter, Roy, & Arrowood, 1991; Warburton et al., 2002). Therefore, the importance of screening athletes' cardiac performance to detect normal versus abnormal cardiac adaptations needs to be emphasized.

Two of the most common methods used to assess physiological adaptations of the heart include echocardiography and electrocardiography (ECG). These tools currently play an important role in evaluating an athlete's heart during routine medical exams (Corrado et al., 2005). Echocardiography has been used previously to assess cardiac performance in athletes (Pelliccia & Maron, 1995) and a greater understanding of the cardiovascular adaptations that can occur with regular exercise training has been developed. Echocardiography quantifies heart structure variables such as thicknesses, internal dimensions and functions, as well as estimations of left ventricular mass. However, the use of echocardiography is not always possible due to expense, time and accessibility. Thus, its ease of use is limited particularly for mass-screening (Karamitsos et al., 2007). As a result, ECG has been suggested as a more cost-effective and practical method for mass screening of athletes (Corrado et al., 2009; Corrado et al., 2005). It is a simple and useful diagnostic technique that measures the electrical potential differences

between two points on the body (Green, 1987). Although it is a commonly used technique to measure cardiac performance, it is not able to detect all cardiac conditions (especially during the early stages of disease) (Lowenstein et al., 1962; Maron, 2009; Phibbs et al., 1967) and the logistics of using ECG to perform mass screening remains a concern (Le et al., 2010).

A reliable and cost-effective method that could be used to potentially evaluate an athlete's heart would have applications for medical and health personnel when assessing the cardiovascular function of athletes. Recently, ballistocardiography (BCG) technology has been used increasingly to assess cardiac function (Alametsa et al., 2009; Bombardini, Gemignani, Bianchini, Venneri, Petersen, Pasanisi, Pratali, Pianelli, et al., 2008; Ngai et al., 2009; Pinheiro et al., 2010) and has the potential to become an important cardiac assessment and screening tool for athletes. In the past, BCG was limited because there was no way to electronically or digitally analyze the BCG waveform. To date, advances in microprocessor and computer technology have improved BCG technology, thus a digitized signal representing the cardiac cycle is possible. Some BCG technology can collect data from three anatomical planes allowing for more precision when it comes to annotating the ballistocardiogram. Compared to a single-axis ballistocardiogram, three axes provide further confirmation for where each cardiac cycle event occurs. Presently, an algorithm for automated analysis is in the process of being developed.

Overall, BCG is a simple, non-invasive, time- and cost-efficient method which uses accelerometer technology to record the ultra-low frequency vibrations generated by the contraction of the heart (i.e., seismic activity). A BCG recording at rest is all that is needed to evaluate potential cardiac complications and the assessment can be completed

in a matter of minutes. It can provide an indication of the amplitude of waveforms as well as the timing and duration of events in the cardiac cycle when combined with a simultaneous ECG recording (Neary, MacQuarrie, Jamnik, et al., 2009). BCG has an advantage over other more traditional and commonly used diagnostic techniques (e.g., ECG and echocardiography). It can detect abnormalities during the early stages of disease as well as provide additional information about the duration of cardiac cycle timing events and amplitude (a surrogate for contractility changes) of cardiac events. BCG has been correlated and compared with echocardiography, a currently accepted and gold standard technology. When comparing the labeling of the various cardiac events between both BCG and echocardiography, results indicate that BCG provides a reliable measure of cardiac performance (Crow et al., 1994; Zanetti et al., 1991).

Using BCG, the purpose of this study was to examine the differences in cardiac timing characteristics of trained ice hockey players in relation to a recreationally active control group. It was hypothesized that BCG would be able to capture the differences in the timing of cardiac cycle events in highly trained male ice hockey players in comparison with a control group of recreationally active male volunteers.

METHODS

Participants

Highly trained male ice hockey players (n = 24; aged 18.0 ± 1.3 years; height 180.6 ± 3.5 cm; weight 83.4 ± 4.9 kg) and a recreationally active control group of young male adults (n = 20; aged 22.5 ± 2.4 years; height 178.1± 6.5 cm; weight 81.4 ± 10.5 kg) were recruited for this study. The highly trained group included male ice hockey players who were recruited, upon permission, from the Athol Murray College of Notre Dame in

Wilcox, Saskatchewan. Each hockey player practiced at a moderate to high intensity six times per week, on average. On game days, practice consisted of a light 30-minute skate in the morning. Additional workouts involving strength training were dependent on the game/practice schedule. Typically each player would lift weights twice a week, as well as attend 1 – 2 injury prevention workouts, consisting of foam rolling and flexibility exercises (30 – 45 minutes per session). Every 4 – 6 weeks the grip or stance of their weight lifting exercises would be modified by the team's strength and conditioning coach. For an overview of the trained participants' training program refer to Appendix A.

The recreationally active control group of presumably healthy active male college student volunteers was recruited on campus. Activity level varied amongst participants. A summary of the activity level for each participant is presented in Table 3. Prior to testing, written informed consent (Appendix B and C) was obtained after a thorough explanation of the procedures and objectives of the study, in accordance with ethical guidelines of the University of Regina Research Ethics Board (Appendix D).

Instrumentation

Using non-invasive BCG (dBG 300 Digital Ballistocardiograph, Heart Force Medical Inc., Vancouver, BC, Canada) the mechanical function of the heart was examined to assess the timing events of the cardiac cycle. The BCG uses a factory calibrated tri-axial accelerometer and proprietary software. Attachment of the accelerometer to the sternum one centimetre above the xiphoid process was achieved by using disposable, Q-Trace ECG electrodes placed on either side of the sensor (5400 Resting ECG Electrode, Tyco Healthcare Group LP, Mansfield, Massachusetts, USA).

Table 3

Frequency of Physical Activity of the Control Group

	Physical Activity Frequency		
Participant	**Strenuous**	**Moderate**	**Mild**
1	2	2	4
2	4	6	7
3	0	0	0
4	4	3	5
5	4	1	7
6	0	0	0
7	2	4	7
8	5	4	6
9	3	5	7
10	5	7	7
11	2	4	7
12	2	0	2
13	5	5	7
14	3	0	0
15	4	2	7
16	5	7	8
Mean ± SD	**3.1 ± 1.7**	**3.1 ± 2.5**	**5.1 ± 2.9**

Note. Physical activity frequency represents the number of times on average per week of strenuous (i.e., heart beats rapidly; running, jogging), moderate (i.e., not exhausting; fast walking, dancing), and mild exercise (i.e., minimal effort; yoga, housework) was performed. As in the Godin Leisure Physical Activity Questionnaire by Godin and Shephard (1985). Four of the participants did not provide their physical activity level and are not included in the table above.

This provided the BCG with the ability to collect a simultaneous (Lead II) electrocardiogram as illustrated in Figure 4.

Experimental Protocol

Participants were instructed to lay (supine) on a medical bed, with both arms relaxed by their sides for five minutes prior to assessment. During this time, the Q-Trace electrodes were attached to the sensor and the Bluetooth®-enabled software program was coupled to the BCG to allow for data collection. Prior to testing, the area of attachment was cleaned and prepared for data collection. Excessive body hair or perspiration that could have interfered with attachment of the sensor affecting the signal was removed. The sensor was attached one centimetre above the xiphoid process on the sternum using the adhesive Q-Trace electrodes placed on either side of the sensor. Adjustments were made until an optimal electrocardiogram and BCG signal was found. Participants were instructed to completely relax, breathe normally and not to move or talk throughout the procedure to ensure quiet resting conditions. Data collection occurred over a 30 second time frame.

Data Acquisition

BCG data was analyzed off-line using proprietary software designed to compute the timing and amplitude of the cardiac cycle events. The BCG annotations are derived based on the movement of the heart that occurs within each cardiac cycle. As the heart contracts and relaxes, the movement is sensed by the BCG and processed digitally by the software (Figure 4). Using nomenclature previously outlined by others, each cardiac cycle was annotated for timing and amplitude events by the same technician (Crow et al., 1994; Scarborough, 1955). For an overview of BCG annotations refer to Table 4.

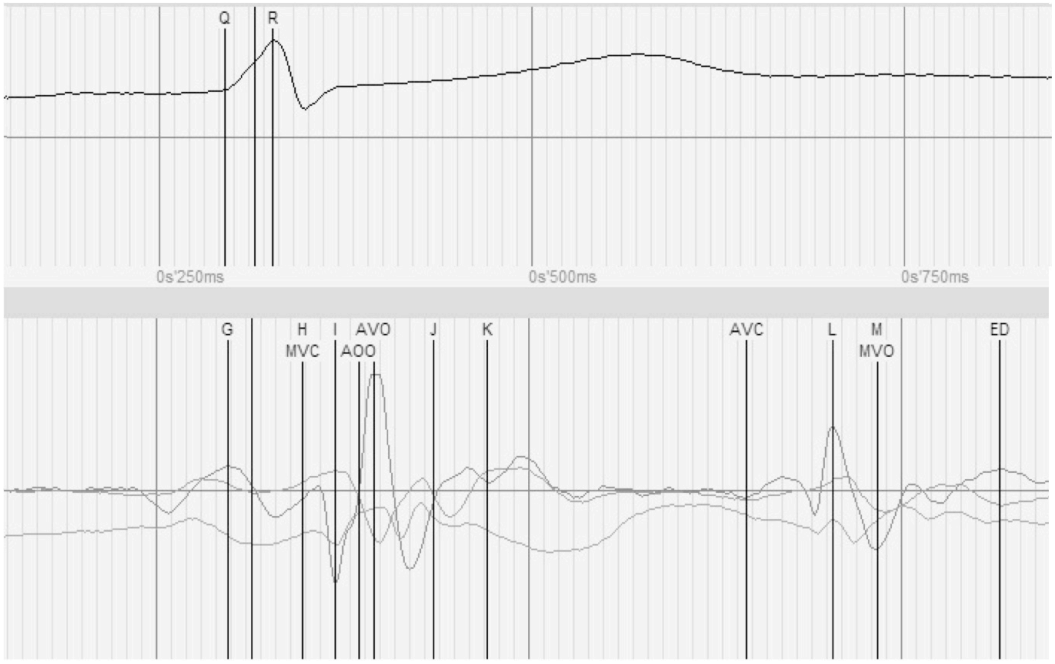

Figure 4. Digital ballistocardiogram (BCG) with simultaneous electrocardiogram (ECG) showing the different waveform annotations. G, atrial systole; H/MVC, mitral valve close; I, isovolumic contraction; AOO, onset of aortic valve open; AVO, aortic valve open; J, end of rapid ejection; AVC, aortic valve close; L, aortic recoil; M/MVO, mitral valve open; ED, early diastole. Annotations based on guidelines outlined by Crow et al. (1994).

Table 4

Ballistocardiograph Annotations

Annotation	Representation
Q	Ventricular depolarization
R	Ventricular depolarization
G/A	Atrial systole
H/MVC	Mitral valve close
I	Isovolumic contraction
AOO	Onset of aortic valve open
AVO	Aortic valve open
J	Rapid ejection end
K	N/A
AVC	Aortic valve close
L	Aortic recoil
MVO	Mitral valve open
ED	Early diastole
LD	Late diastole

Note: Annotations adapted from guidelines outlined by Crow et al. (1994).

For the purpose of this study, specified dependent variables for cardiac timing events were examined. Systolic and diastolic timing events of the cardiac cycle were examined from the resting BCG of each participant. Systole was defined as the segment of the cardiac cycle from the closing of the mitral valve to the closing of the aortic valve (Otto, 2004). Diastole was defined as the interval from the closing of the aortic valve to the closing of the mitral valve (Otto, 2004). Systolic and diastolic timing events included isovolumic contraction (IVCT) and relaxation time (IVRT), early diastolic filling to atrial systole (E to A), as well as pressure half-time (P½T). IVCT is the duration from the closing of the mitral valve to opening of the aortic valve. IVRT is from the closing of the aortic valve to the opening of the mitral valve. E to A is the time frame from early diastolic filling to atrial systole, and P½T is from the opening of the aortic valve to rapid ejection of blood from the left ventricle. IVCT, IVRT, E to A, and P½T are all parameters of overall systolic and diastolic function (Otto, 2004). As well, timing events of waveforms including Q-wave (ECG) to A, Q to MVC, Q to AVO, Q to J, Q to AVC, Q to MVO, Q to ED, and Q to LD were also examined.

Statistical Analysis

Independent samples *t*-test was used in examining differences in the dependent variables outlined above between groups. Training status (highly trained ice hockey players or recreationally active control group) was used as the independent variable in all analyses. Levene's test was utilized to analyse equality of variance and if violated, alternative *t*-values were used. Effect size was calculated to indicate the proportion of variance of the dependent variable that was explained by the independent variable. Effect sizes were categorized as small (≤ .01), medium (≥ .06), and large (≥ .138) (Cohen,

1988). Values are displayed as mean ± standard deviation (SD). Statistical significance was set at $p < .05$. All data were analyzed using a commercially available statistical software package (SPSS 17.0, SPSS Inc., Chicago, IL, USA).

RESULTS

All heartbeats, which included 621 beats for the ice hockey players and 562 beats for the control group, were annotated and included for analysis as each beat is unique and independent of each other. The average number of cardiac cycles annotated for each 30-second BCG recorded was 27 ± 4 beats per hockey player and 28 ± 4 beats for each control group participant. Resting heart rate was significantly different between the ice hockey players and those in the control group (60.5 ± 10.7 vs. 62.8 ± 9.2 beats·min^{-1}, $t(1174.7) = -3.915$, $p < .001$). Although heart rates were significantly different, the magnitude of the difference in the means was very small ($\eta^2 = .01$).

Significant differences were observed between groups for both systolic and diastolic parameters of the cardiac cycle (IVCT, P½T, IVRT, E to A, and systole) and are summarized in Table 5. Cardiac timing parameters including Q (ECG) to A, Q to MVC, Q to AVO, Q to J, Q to AVC, Q to MVO, Q to ED, and Q to LD were also significantly different. Diastole was the only cardiac timing event that was not significant. Cardiac timing values for each group are illustrated in Figures 5 and 6.

Table 5

Comparison of Cardiac Timing Parameters Between Groups

Measure	Mean ± SD (ms)		Normative Values (ms)	*p*	Partial Eta Squared
	Trained	Control			
Q to A	-14.9 ± 19.3	-10.9 ± 18	-	**.000**	.011
Q to MVC	31.6 ± 9	35.9 ± 10.7	-	**.000**	.044
Q to AVO	86.7 ± 8.8	88.7 ± 7.8	-	**.000**	.014
Q to J	145.2 ± 14.3	143.2 ± 10.8	-	**.007**	.006
Q to AVC	326.2 ± 25.8	318.8 ± 15.3	-	**.000**	.031
Q to MVO	424.4 ± 23	419.2 ± 15.2	-	**.000**	.018
Q to ED	510.5 ± 27	499.5 ± 19.1	-	**.000**	.053
Q to LD	1009.4 ± 188	968.3 ± 147.1	-	**.000**	.015
IVCT	55.1 ± 8.6	52.8 ± 8.7	40	**.000**	.017
P½T	58.5 ± 11.2	54.6 ± 8.2	70	**.000**	.039
IVRT	111.4 ± 16.6	100.4 ±13.8	80-100	**.000**	.114
E to A	498.9 ± 168.9	469.1 ± 142.8	-	**.001**	.009
Systole	226.4 ± 22.9	230.2 ± 15.5	250-290	**.001**	.009
Diastole	630.5 ± 172.6	608.0 ± 259.5	500	.079	-

Note. All established normative echocardiography values for cardiac timing events were derived from Otto (2004).

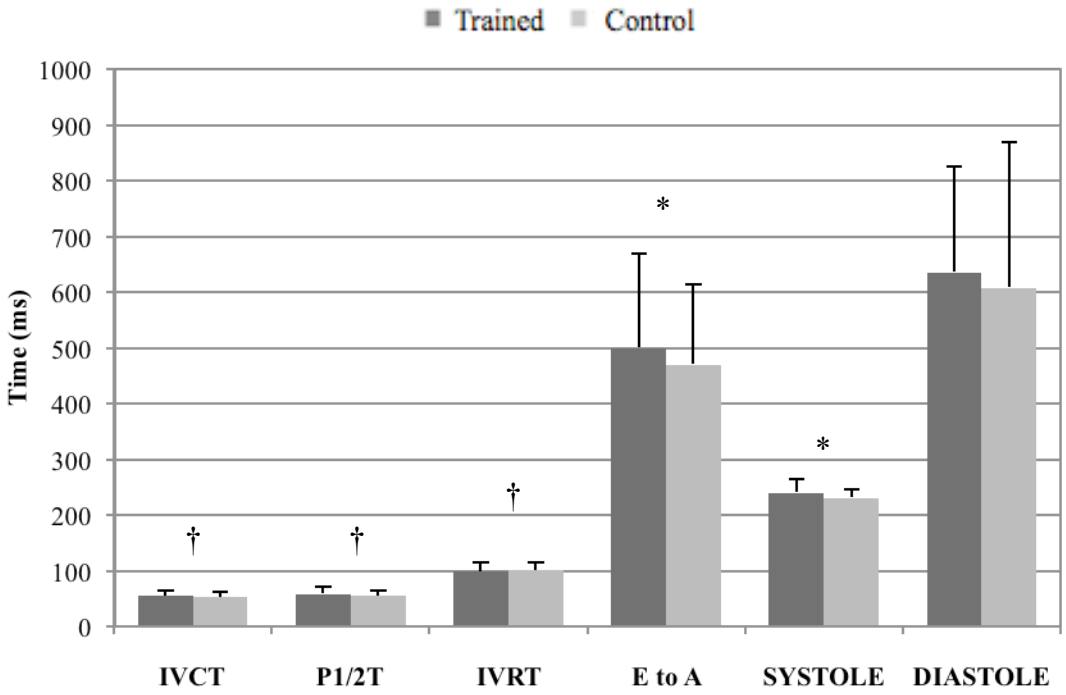

Figure 5. A visual representation of cardiac timing events in both the trained and control participants, measured using ballistocardiography. *Note.* * indicates value is significantly different at $p < 0.05$. † indicates value is significantly different at $p < .001$.

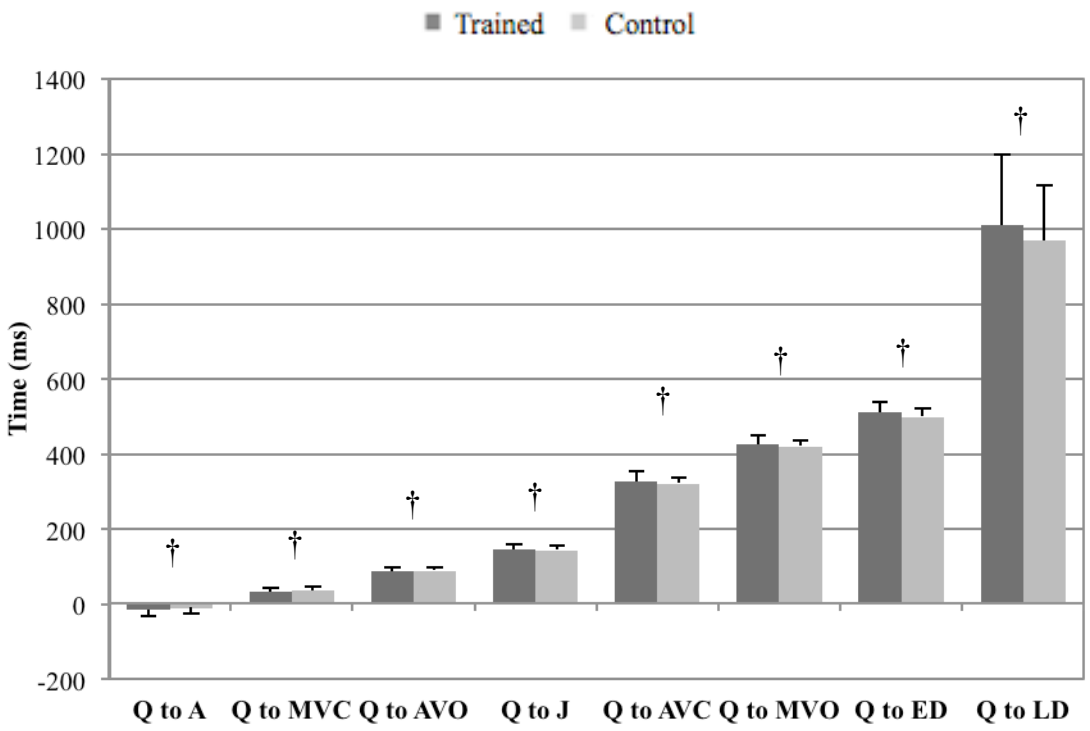

Figure 6. A visual representation of cardiac timing events (in relation to Q) in both the trained and control participants, measured using ballistocardiography. *Note.*† indicates value is significantly different at $p < .001$.

DISCUSSION

This study measured the timing of cardiac cycle events in highly trained ice hockey players and a recreationally active control group using BCG. Results showed that BCG can detect differences in cardiac performance in these groups. They were also in agreement with previous research suggesting that there are differences in the hearts of those who are highly trained, recreationally active and sedentary, specifically with regards to the duration of cardiac cycle events (Bossone et al., 2004; Di Bello et al., 1996; Fazel et al., 2009; Forman et al., 1992; Libonati, 1999; Libonati et al., 1999; Nixon et al., 1991; Petridis et al., 2004). The data of the current study illustrated significantly different durations of cardiac cycle events in highly trained ice hockey players in comparison to a recreationally active control group. When comparing timing values with established average echocardiography values for a normally active population (not training for any specific event or sport), the highly trained ice hockey players would be considered to display improved cardiac function (see Table 5) since the acceleration of the blood (e.g., timing events: diastole, IVRT, P½T, etc.) is the best predictor of cardiac function (Starr, 1965).

Significant differences were found between the highly trained ice hockey players and recreationally active control group for IVRT. The control group fell within the established average of 80 – 100 ms for IVRT (Otto, 2004), whereas the hockey players were above the average. This finding was surprising as typically IVRT is an inverse reflection of the compliance (i.e., stiffness) of the vascular system so one would assume athletes would have a shorter IVRT interval. Libonati et al. (1999), who investigated the

relationship between exercise capacity and resting cardiac function time intervals using non-invasive seismocardiography (similar to BCG), found a reduced IVRT interval with increased exercise capacity (80 ± 8 vs. 107 ± 8 ms; $p < 0.05$). Conversely, and in agreement with the current study, researchers have found that IVRT is prolonged in athletes in comparison to controls (Fardy, 1971; Finkelhor, Hanak, & Bahler, 1986; Levy, Cerqueira, Abrass, Schwartz, & Stratton, 1993; Underwood & Schwade, 1977), while others have found no change (Krzeminski, Niewiadomski, & Nazar, 1989).

E to A was also significantly different between groups. E to A does not have an established average value, as it is purely a function of heart rate. A slower heart rate will result in a longer E to A time. Therefore, the trend of longer E to A time observed in the hockey players versus the control group could be explained by a decreased heart rate. There are some studies that normalize cardiac cycle time intervals on the basis of heart rate to separate the effects of heart rate on cardiac timing intervals. However, because both the hockey players and control group fall within the generally accepted resting heart rate range, normalization was not necessary. Additionally, when heart rate is normalized, it can affect values that stay fairly constant (e.g., an average QRS complex is 40 ms in duration and if a fast heart rate is normalized this number will decrease disproportionately). Therefore, the normalization of heart rate would have only inflated the group differences.

The trained ice hockey players displayed a trend of higher average diastolic time as compared to the control group, although not statistically different between groups. A lower heart rate, as displayed in the trained ice hockey players, allows for more diastolic filling time because the heart does not have to work as hard to maintain the required

cardiac output due to other cardiac adaptations that can occur in response to exercise training (e.g., increased stroke volume). It is also possible that recorded diastolic timing values are greater than the average populations' as a result of structural remodeling leading to physiologic hypertrophy. Physiologic hypertrophy has the potential to enhance diastolic function through structural adaptations that increase left ventricular end-diastolic volumes (Libonati, 1999). Upon observation, approximately 60% of the trained participants demonstrated signs of hypertrophy (increased R- and T-wave amplitudes and multi-peaked R-waves) on their electrocardiograms (Thaler, 2010). These are all strong indicators of left ventricular hypertrophy, and when detected on the electrocardiogram there is a 90% chance that a thickened ventricle or a dilated chamber will be seen on an echocardiogram (Thaler, 2010).

The improvements in diastolic function likely contributed to those found in systolic performance. Structural remodeling can lead to greater diastolic filling mechanics which, in turn, improves systolic performance. As well, stronger contractile force in the hockey players may have also contributed, but this needs to be further explored in a prospective study. Normal reported values for systole duration range from 250 – 290 ms (Otto, 2004) and in this study, systolic timing values for both the trained ice hockey players and recreationally active young adults fell outside of this range. As well, the hockey players had a slightly shorter systolic period, in comparison to the control group. These results are similar to those reported by previous researchers as duration of systole was shortened with exercise training (Falsetti, Gisolfi, Lemon, Cohen, & Claxton, 1982), while others found no changes or prolongation of systole with greater exercise capacity (Falsetti et al., 1982; Fardy, 1971; Franks & Cureton, 1969; Krip,

Gledhill, Jamnik, & Warburton, 1997; Krzeminski et al., 1989; Lamont, 1980; Libonati et al., 1999; Underwood & Schwade, 1977). The shortened systolic period found in the current study may be better understood through a closer examination of specific systolic cardiac timing events, IVCT and P½T.

IVCT duration was above the reported normal average of 40 ms (Otto, 2004) for both the hockey players and recreationally active control group. The prolongation in the trained hockey players may have been the result of left ventricular hypertrophy, as it may delay contraction. Ventricular hypertrophy may cause asynchronous contraction of the ventricles, because of a muscle mass imbalance between the left and right ventricle. A study by Erol and Karakelleoglu (2002) compared IVCT values of athletes, with and without left ventricular hypertrophy, using echocardiography and it appeared that those with hypertrophy demonstrated prolonged IVCT (57.0 ± 24.4 vs. 61.1 ± 23.4 ms). These results are similar to those reported in our study.

The duration of P½T in both the trained ice hockey players and control group was less than the average normal value of 70 ms (Otto, 2004). A reduced P½T indicates that maximal acceleration was reached in a shorter time period – perhaps an effect of increased contractile force. This may also be related to the dynamics between heart rate, stroke volume, and cardiac output. In comparison to the hockey players, the control group had a shorter acceleration time. This observation allows for the speculation of a greater volume of blood (i.e., end diastolic volume) being expelled in the hockey players due to training. Because of an increased volume, ejecting the blood would have likely taken longer in the trained hockey players, in comparison to the control group. Consequently, the control group may have had a quicker acceleration time because of a decreased blood

volume. A recent study by Bossone, Vriz, Bodini, and Rubenfire (2004) supports this speculation. Upon examination of the hearts of well-trained university hockey players in comparison to a healthy normally active group, it was found that stroke volume at rest, although not statistically significant, displayed a trend of being higher in the hockey players than in the normally active control group (74.3 ± 15 vs. 59.1 ± 15 mL).

The results of this study are more difficult to generalize to a greater population than to specific groups of participants such as those in this study. The trained group consisted of highly trained ice hockey players who performed a combination of high dynamic and moderate static exercise. It is likely that if participants who perform a different competitive sport or combination of regular high intensity static and dynamic exercise (e.g., wrestlers, long-distance runners or volleyball players) were observed that the results would be altered. Based on these findings, it is very probable that the cardiac differences between the groups were a result of the high intensity exercise training performed by the ice hockey players.

Although the type and frequency of physical activity performed by the control group was not logged in great detail, we believe that the results presented here using a cross-sectional design are still a valid comparison for both groups. Comparisons between groups were made using independent samples *t*-tests using all recorded heartbeats for each participant, rather than calculating mean values across 30 seconds, despite the risk of increasing type I error. All beats were included to maintain the variability, uniqueness and individuality that is inclusive of the cardiac cycle. Future studies using a prospective design will add important information in this area. Echocardiography could also assist to

confirm BCG results, to strengthen its effectiveness in measuring cardiac timing events and detecting cardiovascular abnormalities.

Conclusion

The results of this study are novel in that to our knowledge, BCG has not been previously used to examine cardiac function in trained ice hockey players that use a combination of exercise training methods. These results confirm that BCG has the potential to provide insight into the athletic heart, and that significant differences were found between participants engaging in different levels of exercise training. Our findings of changes in diastolic (IVRT and E to A) and systolic (IVCT, P½T, and total systolic time) parameters in trained ice hockey players may allow for a better understanding of how the heart adapts to exercise training. Thus, future research using BCG will provide greater insight into the effects of exercise training on cardiac function, as prospective studies are needed to confirm these results. As well, it will allow clinicians and researchers the ability to observe the athletic heart in a simplified manner, which has the potential to help with the differentiation between physiological and pathological features.

CHAPTER III – ASSESSING CARDIAC CYLE MECHANICS IN MALE ICE HOCKEY PLAYERS USING BALLISTOCARDIOGRAPHY

ABSTRACT

Ballistocardiography (BCG), an emerging non-invasive technology, measures ultra-low frequency vibrations of the heart and has the potential to screen for cardiac abnormalities. The purpose of the present study was to determine if BCG can detect differences in waveform amplitudes of cardiac events (i.e., a surrogate of contractility force) in highly trained ice hockey players compared to a recreationally active control group. Participants included 24 trained hockey players (aged 18.0 ± 1.3 years) and 20 recreationally active healthy young adults (aged 22.5 ± 2.4 years) who were not training for any particular sport or event. Data collection occurred under quiet resting conditions for 30 seconds using BCG. Data were analyzed off-line using proprietary software designed to compute the timing and amplitude of cardiac cycle events. *t*-tests were performed to investigate the differences in waveform amplitude between groups. Significance was set at $p < .05$. The trained ice hockey players illustrated greater strength of all amplitude events measured when compared to the control group. Events with significantly different amplitudes included the I-wave (48.2 ± 13.3 vs. 28.9 ± 6.1 mG), aortic valve open (43.2 ± 16.8 vs. 27.5 ± 7.7 mG), rapid ejection (7.0 ± 5.5 vs. 3.9 ± 3.2 mG), mitral valve open (42.6 ± 10.6 vs. 21.7 ± 6.9 mG), and early (16.8 ± 6.4 vs. 9.0 ± 3.8 mG) and late (11.5 ± 6.4 vs. 6.7 ± 4.4) diastolic filling. It was concluded that BCG is an effective technology for detecting differences in cardiac performance variables and that ice hockey players experience greater contractility compared to recreationally active individuals.

INTRODUCTION

The maintenance of cardiac function is critical for athletic performance and can be achieved through careful monitoring of cardiac performance. Past research using cardiac screening tools, such as electrocardiography (ECG), echocardiography, and ballistocardiography (BCG) to measure cardiac performance of the athletic heart have found differences in the cardiac function of well-trained individuals (Bossone et al., 2004; Erol & Karakelleoglu, 2002; Fazel et al., 2009; George, Shave, Warburton, Scharhag, & Whyte, 2008; Libonati, 1999; Petridis et al., 2004; Warburton, Gledhill, Jamnik, Krip, & Card, 1999; Warburton et al., 2002), including increased contractility force of the heart (Holloszy, Skinner, Barry, & Cureton, 1964; Smith, Humphrey, Wohlford, & Flint, 1994; Stork, Mockel, Muller, Eichstadt, & Hochrein, 1992). Although the specific processes responsible for changes in cardiac contractility have not been clearly identified, there are several possibilities that can be considered. Possible adaptations leading to cardiovascular changes, and specifically improved myocardial contractility, include increased maximal cardiac output and stroke volume, reduced resting heart rate, and increased plasma volume (Fazel et al., 2009; Gledhill et al., 1994; Warburton et al., 2002).

It has been noted that cardiovascular screening tools can play an important role in evaluating an athlete's heart during routine medical exams (Corrado et al., 2005). Echocardiography, ECG, and cardiac MRI are some of the most common tools to assess and measure cardiac performance. Echocardiography has allowed clinicians and researchers to gain a better insight towards the understanding of the cardiovascular changes which occur from performing regular exercise training, and is commonly used in

some European countries to assess their athletes (Pelliccia & Maron, 1995). Echocardiography quantifies heart structure variables such as thicknesses, internal dimensions and functions, as well as estimations of left ventricular mass and blood flow characteristics. Cardiac MRI provides superior image quality in comparison to echocardiography and its use to evaluate the athletic heart is increasing (Prakken et al., 2009). Cardiac MRI allows for a complete and non-invasive evaluation of cardiac function, morphology and tissue characterization (Germans & van Rossum, 2008). It is able to provide information on both anatomical and physiological abnormalities of the cardiovascular system (Herfkens et al., 1983). Unfortunately, even with the vast amount of information provided, the practicality of performing mass screening using echocardiography and cardiac MRI is low as they are both costly and time consuming diagnostic techniques (Karamitsos et al., 2007). As a result, twelve-lead ECG has been proposed as a more cost-effective and practical method for screening the heart (Corrado et al., 2009; Corrado et al., 2005). ECG is a simple and useful diagnostic technique used to evaluate the electrical activity of the heart through the measurement of the potential difference between two points on the body (Green, 1987). However, mass testing with ECG encounters a similar problem as echocardiography and cardiac MRI. Although ECG is one of the most important and common tools used in cardiology today, the logistics of screening large numbers of individuals remains a concern (Le et al., 2010; Neary, Len, MacQuarrie, Jamnik, et al., 2008), and not all cardiac conditions are detected using ECG (Maron, 2009).

A reliable and cost-effective method which could potentially evaluate an athlete's heart would have applications for medical and health personnel when assessing the

cardiovascular function of athletes. Preliminary usage of such methods have proven useful at the annual National Hockey League scouting combine where all players are screened for cardiac abnormalities to detect disease responsible for sudden death in young athletes (i.e., hypertrophic cardiomyopathy, coronary artery anomalies, myocarditis, and atherosclerotic coronary artery disease) (Neary, Len, MacQuarrie, Jamnik, et al., 2008; Neary, MacQuarrie, Jamnik, et al., 2009). Sudden and unexpected deaths of athletes are tragic and can be prevented with proper screening techniques. However, simple and effective screening methods for detecting cardiac adaptations are lacking, especially within amateur sport settings (Harris, Sponsel, Hutter, & Maron, 2006).

Recently, with the advent of microprocessors and computers, the re-development of ballistocardiography (BCG) or seismocardiography (SCG) technology has the potential to become an important cardiac assessment and screening tool for athletes. Both SCG and BCG are non-invasive techniques used for recording cardiac vibrations caused by myocardial wall motion during systole and diastole (Alametsa et al., 2009; Crow et al., 1994). Through the placement of an ultra-low frequency accelerometer on the sternum of the chest the timing and amplitude of mechanical events of the cardiac cycle can be recorded (Crow et al., 1994; Salerno et al., 1991). Both BCG and SCG have been used to measure the strength of myocardial contraction by evaluating the amplitude of selected cardiac waveforms (Alametsa et al., 2008; Korzeniowska-Kubacka, Bilinska, & Piotrowicz, 2005; Starr, 1955).

Using BCG, the purpose of this study was to evaluate the differences in the strength of heart contractility in highly trained male ice hockey players in comparison to

a recreationally active control group. Past research using BCG to measure amplitude of cardiac waveforms found that exercise training resulted in greater amplitude of several cardiac events (Holloszy et al., 1964). It was hypothesized that trained ice hockey players would have higher cardiac forces and produce greater amplitudes in their BCG waveforms than the recreationally active control group.

METHODS

Participants

Highly trained male ice hockey players (n = 24; aged 18.0 ± 1.3 years; height 180.6 ± 3.5 cm; weight 83.4 ± 4.9 kg) and a recreationally active control group of young male adults (n = 20; aged 22.5 ± 2.4 years; height 178.1± 6.5 cm; weight 81.4 ± 10.5 kg) were recruited for this study. The highly trained group included male ice hockey players who were recruited, upon permission, from the Athol Murray College of Notre Dame in Wilcox, Saskatchewan. Each hockey player practiced at a moderate to high intensity six times per week, on average. On game days, practice consisted of a light 30-minute skate in the morning. Additional workouts involving strength training were dependent on the game/practice schedule. A typical week would include lifting weights twice a week and 1-2 injury prevention sessions, consisting of foam rolling and flexibility exercises (30 – 45 minutes per session). On game days they would also have to complete a light 30-minute practice in the morning. Every 4-6 weeks their weight lifting exercises would be modified by the supervising strength and conditioning coach. For an overview of the trained participants' training program refer to Appendix A.

The recreationally active control group was recruited at the University of Regina and was comprised of active male college students, who were not training for any

particular sport or event. Activity level varied amongst participants. Baseline activity level was determined by having each participant fill out a questionnaire indicating the number of times per week, on average, strenuous, moderate, and mild exercise was performed. Activity level for each untrained participant is summarized in Table 3. Prior to testing, written informed consent (Appendix B and C) was obtained after a thorough explanation of the procedures and objectives of the study, in accordance with ethical guidelines of the University of Regina Research Ethics Board (Appendix D).

Instrumentation

The mechanical function of the heart was monitored using non-invasive BCG (dBG 300 Digital Ballistocardiograph, Heart Force Medical Inc., Vancouver, BC, Canada). Using a factory calibrated tri-axial accelerometer, cardiac vibrations of the myocardial wall during systole and diastole were recorded. Attachment of the accelerometer to the sternum one centimetre above the xiphoid process was achieved by using disposable, Q-Trace ECG electrodes placed on either side of the sensor (5400 Resting ECG Electrode, Tyco Healthcare Group LP, Mansfield, Massachusetts, USA). This provided the BCG with the ability to collect a simultaneous (Lead II) electrocardiogram (see Figure 4).

Experimental Protocol

Participants were instructed to lay (supine) on a medical bed, with both arms relaxed by their sides for five minutes prior to assessment. During this time, the Q-Trace electrodes were attached to the sensor and the Bluetooth®-enabled software program was coupled to the BCG to allow for data collection. Prior to testing, the area of attachment was cleaned and prepared for data collection. Excessive body hair or perspiration that

could have interfered with attachment of the sensor affecting the signal was removed. The sensor was attached one centimetre above the xiphoid process on the sternum using the adhesive Q-Trace electrodes placed on either side of the sensor. Adjustments were made until an optimal electrocardiogram and BCG signal was found. Participants were instructed to completely relax, breathe normally and not to move or talk throughout the procedure to ensure quiet resting conditions. Data collection occurred over a 30 second time frame.

Data Acquisition

Data was analyzed off-line using proprietary software designed to compute the timing and amplitude of the cardiac cycle events. The BCG annotations are derived based on the movement of the heart that occurs within each cardiac cycle. As the heart contracts and relaxes the movement is sensed by the tri-axial accelerometer and processed digitally by the software (Figure 4). Each cardiac cycle was annotated for timing and amplitude events using nomenclature previously described by others (Crow et al., 1994; Scarborough, 1955). For an overview of BCG annotations refer to Table 4.

The following waveforms served as the dependent variables in the study and were analyzed for changes in amplitude, which was used as a surrogate of cardiac contractility (Neary et al., 2010). Using the nomenclature proposed by Crow et al. (1994), the A-wave, or atrial systole, is identified as a positive change in amplitude on the z-axis in the vicinity of the Q wave on the electrocardiogram rhythm strip and it is preceded by a U shaped wave. The A-wave is also referred to as the G and/or LD-wave. Typically, the G/LD-wave precedes the Q-wave of the electrocardiogram by approximately 30 ms. The G/LD-wave is annotated at the highest point of the wave. G and LD waves are

synonymous with each other as a G-wave is identical to LD of the preceding cardiac cycle. I-wave is described as the most negative point on the z-axis following mitral valve close (H/MVC). The I-wave is marked at the maximum negative point of the wave on the z-axis and is indicative of isovolumic contraction. Aortic valve opening (AVO) is identified as the first reversal following the H/MVC waveform on the z-axis. The AVO peak is marked at the highest positive point of the reversal following MVC on the z-axis. The end of the rapid ejection period (J) is identified as a positive upslope following the AVO waveform where the x- and z-axis cross. If the axes do not cross, J is annotated on the halfway point of the positive upslope to maintain consistency. Identified as the second negative wave following the closing of the aortic valve (AVC) on the z-axis, MVO signifies the opening of the mitral valve. The MVO annotation is marked at the most negative point of the wave on the z-axis. ED is the annotation marking the beginning of the diastolic period – or early diastole. It generally follows within 100 ms of MVO and is defined as the second positive peak following the MVO annotation (Crow et al., 1994).

Data Analysis

Independent samples *t*-test was used in evaluating differences in the dependent variables outlined above between groups. Training status (highly trained ice hockey players or recreationally active control group) was used as the independent variable in all analyses. Levene's test was utilized to analyse equality of variance and if violated, alternative *t*-values were used in compensation. Effect size was calculated to indicate the proportion of variance of the dependent variable that was explained by the independent variable. Effect sizes were categorized as small (≤ .01), medium (≥ .06), and large (≥ .138) (Cohen, 1988). Values shown are displayed as mean ± standard deviation (SD).

Statistical significance was set at $p < .05$. All data were analyzed using a commercially available statistical software package (SPSS 17.0, SPSS Inc., Chicago, IL, USA).

RESULTS

Using the cardiac events described above, all beats, which included 621 heartbeats for the ice hockey players and 562 heartbeats for the control group, were annotated and included for analysis as each beat is unique and independent of each other. The average number of cardiac cycles annotated for each BCG was 27 ± 4 beats per hockey player and 28 ± 4 beats for each control group participant. Resting heart rate was significantly different between the ice hockey players (60.5 ± 10.7 beats·min^{-1}) and those in the control group (62.8 ± 9.2 beats·min^{-1}) ($t(1174.7) = -3.915$, $p < 0.001$). Although heart rates were significantly different, the magnitude of the difference between the means was very small ($\eta^2 = .01$). The ice hockey players illustrated greater waveform amplitudes in comparison to the control group, as the strength of all amplitude events measured were significantly greater and included I-wave, AVO, J-wave, MVO, ED, and LD amplitude (Table 6). Figure 7 provides a graphical representation of the waveform amplitude differences between groups.

Table 6

Comparison of Amplitude in Cardiac Events Between Groups

Measure	Mean ± SD (mG)		*p*	Partial Eta Squared
	Trained	Control		
I	48.2 ± 13.3	28.9 ± 6.1	**.000**	.478
AVO	43.2 ± 16.8	27.5 ± 7.7	**.000**	.274
J	7.0 ± 5.5	3.9 ± 3.2	**.000**	.105
MVO	42.6 ± 10.6	21.7 ± 6.9	**.000**	.584
ED	16.8 ± 6.4	8.9 ± 3.8	**.000**	.365
LD	11.5 ± 6.4	6.7 ± 4.4	**.000**	.159

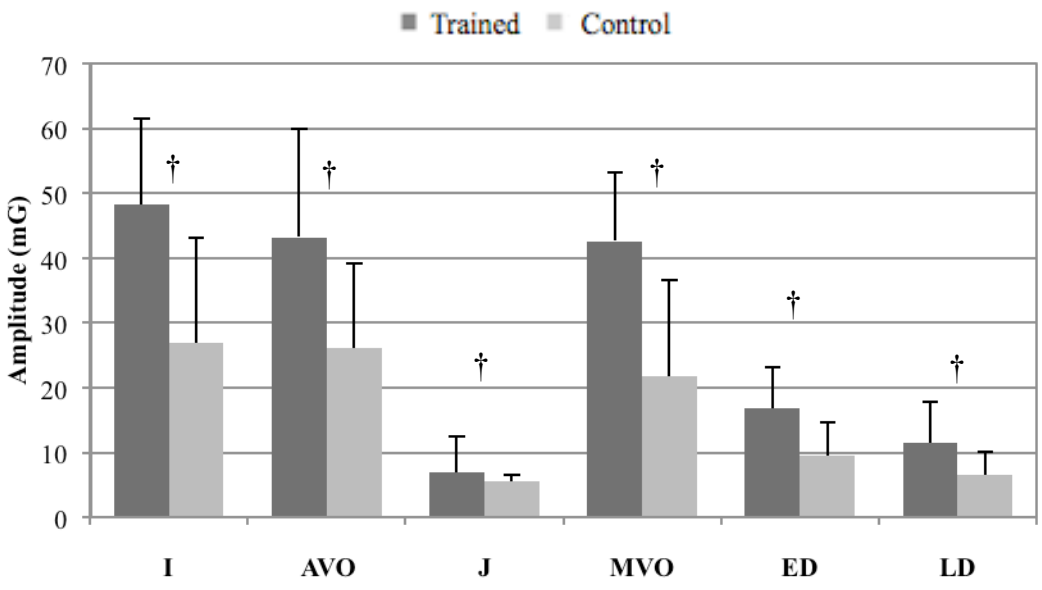

Figure 7. A visual representation of amplitude in cardiac events in both the trained and control participants, measured using ballistocardiography. *Note.* † indicates value is significantly different at $p < .001$.

DISCUSSION

This study measured the amplitude (mG) of selected cardiac cycle waveforms to assess the strength of cardiac contractility in highly trained ice hockey players using BCG technology to see if there is a difference in comparison to recreationally active controls. Results showed that: 1) the level of exercise training has an effect on the heart, 2) amplitude of cardiac cycle waveforms differs significantly with the level of exercise training performed, and 3) BCG is a method that can be used to accurately assess differences in heart contractility. The majority of research studies assessing cardiac function in athletes have used ECG, cardiac MRI, and echocardiography (Bossone et al., 2004; Corrado et al., 2009; Fazel et al., 2009). Although these methods are valid and reliable for measuring cardiac performance, unfortunately they are also costly, time consuming, and non-portable. BCG overcomes these obstacles as it is cost- and time-efficient (60 seconds to complete testing), easy to administer and can be used in athletic and field-like settings.

A unique and important finding of this study is that exercise training produces significantly different waveform amplitudes in diastolic events (Table 6). Potentially, these differences in diastolic parameters with training may be attributed to stronger contractile forces, as significant differences in systolic parameters were also evident.

Although there was an overall effect on cardiac performance, diastolic cardiac parameters changed more than systolic events in the trained hockey players compared to the control group. Factors contributing to this possibly include greater blood volume, left ventricular end diastolic pressure, diastolic filling time, myocardial compliance, and/or

increases in ventricular chamber size as well as hypertrophy of the heart (King et al., 2008; Moore & Palmer, 1999). Therefore, any differences in diastolic function observed in the trained ice hockey players likely occurred as a result of the established haemodynamic and structural adaptations which occur with repetitive hockey-specific training. These systematic adaptations may lead to changes in ventricular filling which can significantly affect stroke volume (i.e., increased end diastolic volume). For example, increases in blood volume which occur with exercise training can enhance diastolic filling capacity and subsequently end diastolic volume and stroke volume (Esch et al., 2007; Gledhill et al., 1994).

Diastolic cardiac events (I-wave, MVO, ED, and LD) were significantly different in the hockey players in comparison to the recreationally active control group. Through unpublished findings from our laboratory, it has been proposed that the I-wave signifies end diastolic movement and a measure of myocardial compliance. The ice hockey players had significantly different amplitude of the I-wave in comparison to the control group. This finding supports research that has examined the influence of exercise training on myocardial compliance (King et al., 2008; Moore & Palmer, 1999). A key factor of diastolic filling of the left ventricle is chamber stiffness (myocardial compliance is the inverse of myocardial stiffness) (King et al., 2008).Therefore, the higher amplitude observed in the trained participants may have been the result of changes to ventricular compliance, which would lead to improved venous flow and greater end diastolic volume. However, further research is warranted to confirm our hypothesis.

Differences in diastolic function were also seen in MVO and ED, as both reflect diastolic filling mechanics. The ice hockey players demonstrated changes in diastolic

filling versus the control group through higher amplitude in MVO and ED. Diastolic filling can change if the ventricle has greater compliance. For example, those suffering from heart failure would likely experience a decrease in the amplitude of MVO as a result of increased stiffness and/or decreased compliance in the left ventricle which limits diastolic filling. Royse et al. (2003) showed that those undergoing cardiac surgery demonstrated an increase in end-diastolic stiffness (King et al., 2008). Diastolic filling is limited because the ventricle becomes too stiff and therefore cannot accommodate large volumes of blood. It has been shown in normal healthy, as well as pathological hearts, exercise training can increase the compliance of the heart during diastole (Moore & Palmer, 1999). For example, myocardial compliance was measured using cardiac catheterisation in both elderly athletic and sedentary participants. It was concluded that lifelong exercise training could prevent myocardial stiffening that is typically caused by aging (King et al., 2008). As well, King (2008) found that there was a reduction in stiffness in healthy endurance rowers in comparison to controls.

In an unhealthy population, stiffer ventricles usually reflect incomplete relaxation with a greater percentage of filling occurring during the late diastolic phase (King et al., 2008). However, in the current study LD, a measure of late diastole, was significantly different between the ice hockey players and recreationally active control group. These findings suggest that the trained ice hockey players may have experienced more forceful atrial contraction, as G and LD waves are synonymous with each other.

Amplitude of AVO and J-wave (end of the rapid ejection period) was significantly increased in the trained ice hockey players. An improved myocardial compliance, as discussed earlier, is the main plausible explanation for the improved

systolic function with training. Theoretically, with greater compliance ventricle responsiveness to increased blood volume and preload would increase, resulting in higher stroke volume (Chummun, 2009).

Indicators of heart hypertrophy can be detected by elevated R- and T-wave amplitudes and multi-peaked R-waves on the electrocardiogram (Thaler, 2010). In this study, approximately 60% of the ice hockey players demonstrated signs of cardiac hypertrophy on their electrocardiograms. Therefore, it is possible that the hypertrophic changes occurring in the heart's muscle fibres with regular exercise training resulted in more forceful contractility, and is reflected as an increase in amplitude in ballistocardiographic events, supporting previous BCG research (Holloszy et al., 1964).

Although we did not directly measure cardiac contractility in this study, previously conducted research has examined changes in contractility using displacement cardiography (i.e., BCG and SCG) (Holloszy et al., 1964; Korzeniowska-Kubacka et al., 2005; Starr, 1955). Based on this research, we are confident that our waveform amplitude measures are a surrogate for contractility. However, future research in this area will confirm our premise. Independent *t*-tests were used to compare all recorded heartbeats between groups. All beats were included, rather than a 30 second average, despite the risk of increased type I error. Reasons for including all beats included maintaining the variability, uniqueness and individuality that is inclusive of the cardiac cycle. Furthermore, while there is some research available to show that BCG is correlated with echocardiography for particular cardiac cycle events (Crow et al., 1994) we did not have access to an echocardiograph for this particular study. Prospective research studies in this

area will help to confirm our data and strengthen the use of BCG as a technique to record cardiac events.

Conclusion

This study demonstrated that BCG is an effective technique in detecting differences in cardiac function and supports previously published literature using SCG (Libonati et al., 1999) and BCG (Holloszy et al., 1964). The findings indicated that trained ice hockey players in comparison to the control group had significantly different waveform amplitude in all measured diastolic and systolic cardiac events, supporting the notion that training athletes have stronger myocardial contractility resulting from habitual exercise training. Furthermore, these results suggest that BCG can be used as an assessment tool for mass athletic testing.

CHAPTER IV – THESIS CONCLUSION

Major Findings

This thesis examined cardiac performance in highly trained ice hockey players utilizing a simple-to-use, non-invasive technology called BCG. **Chapter 1** provides a thorough outline of more traditional and commonly used cardiovascular diagnostic devices, as well as an in-depth review of the history and technological advances in the field of BCG. BCG technology has advanced considerably over the years and is presently an ideal means to evaluate cardiac function of large groups such as athletic teams. It is time-efficient, simple to use as well as cost-efficient. Future research using BCG will help to determine its efficacy to quantify the effects of exercise training on cardiac performance. Both **Chapter 2 and 3** provided evidence to support that regular exercise training leads to differences in the heart.

Chapter 2 evaluated the timing of cardiac events and found that there were changes to the timing of both systolic and diastolic cardiac events. Even though diastolic filling time was not significantly different the trained ice hockey players had a trend of higher average diastolic time in comparison to the normal and recreationally active heart. Diastolic parameters IVRT and E to A were both found to be significantly different between groups. The longer IVRT in the ice hockey players suggests more efficient filling, supporting previous research (Fardy, 1971; Franks & Cureton, 1969; Underwood & Schwade, 1977). Changes to diastolic filling may have occurred as a result of haemodynamic and structural adaptations in response to regular exercise training (Gledhill et al., 1994). Improvements in diastolic function support enhanced systolic performance observed in this study.

The results in **Chapter 2** revealed that the ice hockey players had a slightly shorter systolic period in comparison to the control group. This may be better understood by examining systolic cardiac timing parameters including IVCT and P½T, which were both significantly different between the trained ice hockey players and the recreationally active heart. Findings for IVCT indicated that acceleration time of the ventricle was longer in the ice hockey players than in the control group or reported norms in the literature (Otto, 2004). However, the control group, in comparison to the trained hockey players, had a shorter acceleration time. This may be the result of haemodynamic changes as the trained ice hockey players may have had a greater volume of blood to expel as a result it would have taken longer to expel the blood in the hockey players in comparison to the recreationally active control group.

The results from **Chapter 3** showed that the amplitude of all selected cardiac event waveforms, both during systole and diastole, were significantly different when comparing the trained ice hockey players to the control group. However, exercise training seemed to have a greater effect on diastolic events in comparison to systolic. Factors such as increased blood volume, left ventricular end diastolic pressure, diastolic filling time, myocardial compliance, and/or increased ventricular chamber size as well as hypertrophy of the heart may have contributed to the differences in amplitude of cardiac events.

The diastolic parameters I-wave, ED, and LD were significantly different in the trained ice hockey players in comparison to the recreationally active control group. The greater amplitude found in the I-wave suggest that the hockey players had greater myocardial compliance, thus leading to changes in diastolic filling of the left ventricle (ED). LD or late diastolic filling is synonymous with atrial contraction. Therefore, the

findings of LD indicated that the ice hockey players were experiencing more forceful atrial contraction in comparison to the control group. Systolic parameters AVO and the J-wave were also significantly different in the ice hockey players. Improvements observed in AVO and the J-wave may also have been because of improved myocardial compliance, which leads to a greater stretch of the ventricle, thus increasing preload and ultimately stroke volume. Changes to the myocardium, similar to skeletal muscle adaptations (e.g., fibres hypertrophy, myofibrils and capillaries increase, and thickening of the sarcolemma) may also contribute to the stronger contractile force observed in the hearts of trained ice hockey players, which was reflected as an increase in amplitude in BCG events.

Future Implications

The findings of this thesis project add to the knowledge available in that regular exercise training leads to differences in cardiac function or performance. Additionally, this thesis provides support for the use of BCG as a screening technology, particularly for mass screening of large groups of athletes. The importance of screening athletes is of great concern as serious complications can arise if cardiac anomalies are not detected prior to participation in competition or training. BCG has been found to be simple, time-efficient as well as cost-effective in comparison to more traditional and commonly used diagnostic devices such as echocardiography or ECG. BCG has the potential to detect changes in not only the athletic heart, but also the diseased and normal healthy heart. Therefore, the results of this thesis will hopefully not only assist in providing support to screen athletes using BCG, but the clinical and general population as well.

Future research using BCG should examine diverse populations to provide evidence to demonstrate that it is a useful technology to detect changes in cardiovascular function. This will allow researchers to continue to contribute knowledge and information about the effects of certain cardiovascular states (i.e., diseased, athletic heart etc.) on cardiovascular function or performance, as well as provide support for the use of BCG as a cardiac screening technology. Overall, BCG has the potential to contribute to not only cardiac research, but also to become a screening technology in clinical and athletic settings as well as for the general population.

REFERENCES

Alametsa, J., Varri, A., Viik, J., Hyttinen, J., & Palomaki, A. (2009). Ballistocardiogaphic studies with acceleration and electromechanical film sensors. *Medical Engineering and Physics, 31*(9), 1154-1165.

Alametsa, J., Viik, J., Alakare, J., Varri, A., & Palomaki, A. (2008). Ballistocardiography in sitting and horizontal positions. *Physiological Measurement, 29*(9), 1071-1087.

Alfidi, R. J., Haaga, J. R., El-Yousef, S. J., Bryan, P. J., Fletcher, B. D., LiPuma, J. P., et al. (1982). Preliminary experimental results in humans and animals with a superconducting, whole-body, nuclear magnetic resonance scanner. *Radiology, 143*(1), 175-181.

Anavekar, N. S., & Oh, J. K. (2009). Doppler echocardiography: A contemporary review. *Journal of Cardiology, 54*(3), 347-358.

Baker, B. M. (1968). Ballistocardiography: Predictor of coronary heart disease. *Circulation, 37*(1), 1-3.

Bayevski, R. M., Egerov, A. D., & Kasarjan, L. A. (1964). Seismocardiography. *Kardiologiia, 18*, 87-89.

Berne, R. M., & Levy, M. N. (1986). *Cardiovascular physiology*. Toronto: The C. V. Mosby Company.

Bigi, R., & De Chiara, B. (2005). Prognostic value of noninvasive stressing modalities in patients with chest pain and normal coronary angiogram. *Herz, 30*(1), 61-66. doi: 10.1007/s00059-005-2640-6

Bombardini, T., Gemignani, V., Bianchini, E., Venneri, L., Petersen, C., Pasanisi, E., et al. (2008). Diastolic time - Frequency relation in the stress echo lab: Filling timing and flow at different heart rates. *Cardiovascular Ultrasound, 6*, 15. doi: 10.1186/1476-7120-6-15

Bombardini, T., Gemignani, V., Bianchini, E., Venneri, L., Petersen, C., Pasanisi, E., et al. (2008). Arterial pressure changes monitoring with a new precordial noninvasive sensor. *Cardiovascular Ultrasound, 6*, 41. doi: 10.1186/1476-7120-6-41

Bossone, E., Vriz, O., Bodini, B. D., & Rubenfire, M. (2004). Cardiovascular response to exercise in elite ice hockey players. *Canadian Journal of Cardiology, 20*(9), 893-897.

Brandes, A., & Bethge, K. P. (2008). Long term electrocardiography (Holter monitoring). *Herzschrittmacherther Elektrophysiol, 19*(3), 107-129.

Caso, P., D'Andrea, A., Caso, I., Severino, S., Calabro, P., Allocca, F., et al. (2006). The athlete's heart and hypertrophic cardiomyopathy: Two conditions which may be misdiagnosed and coexistent. Which parameters should be analysed to distinguish one disease from the other? *Journal of Cardiovascular Medicine, 7*(4), 257-266. doi: 10.2459/01.JCM.0000219318.12504.bb

Charlton, G. A., & Crawford, M. H. (1997). Physiologic consequences of training. *Cardiology Clinics, 15*(3), 345-354.

Chummun, H. (2009). Understanding changes in cardiovascular pathophysiology. *British Journal of Nursing, 18*(6), 359-364.

Clark, A. N., & Beller, G. A. (2005). The present role of nuclear cardiology in clinical practice. *Quarterly Journal of Nuclear Medicine and Molecular Imaging, 49*(1), 43-58.

Clausen, J. P. (1977). Effect of physical training on cardiovascular adjustments to exercise in man. *Physiological Reviews, 57*(4), 779-815.

Cohen, J. (1988). *Statistical power analysis for the behavioural sciences* (2nd ed.). Hillsdale, NJ: Lawrence Erlbaum Associates.

Corrado, D., Biffi, A., Basso, C., Pelliccia, A., & Thiene, G. (2009). 12-lead ECG in the athlete: Physiological versus pathological abnormalities. *British Journal of Sports Medicine, 43*(9), 669-676. doi: 10.1136/bjsm.2008.054759

Corrado, D., Pelliccia, A., Bjornstad, H. H., Vanhees, L., Biffi, A., Borjesson, M., et al. (2005). Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: Proposal for a common European protocol. *European Heart Journal, 26*(5), 516-524.

Crow, R. S., Hannan, P., Jacobs, D., Hedquist, L., & Salerno, D. M. (1994). Relationship between seismocardiogram and echocardiogram for events in the cardiac cycle. *American Journal of Noninvasive Cardiology, 8*, 39-46.

Cunningham, D. A., Telford, P., & Swart, G. T. (1976). The cardiopulmonary capacities of young hockey players: Age 10. *Medicine & Science in Sports & Exercise, 8*(1), 23-25.

Desjardins, G., & Cahalan, M. (2009). The impact of routine trans-oesophageal echocardiography (TOE) in cardiac surgery. *Best Practice and Research Clinical Anaesthesiology, 23*(3), 263-271.

Di Bello, V., Santoro, G., Talarico, L., Di Muro, C., Caputo, M. T., Giorgi, D., et al. (1996). Left ventricular function during exercise in athletes and in sedentary men. *Medicine & Science in Sports & Exercise, 28*(2), 190-196.

Dock, W., & Taubman, F. (1949). Some technics for recording the ballistocardiogram directly from the body. *American Journal of Medicine, 7*(6), 751-755, illust.

Dock, W., & Zoneraich, S. (1967). A diastolic murmur arising in a stenosed coronary artery. *American Journal of Medicine, 42*(4), 617-619.

Donald, K. W., Lind, S. R., McNichol, G. W., Humphreys, P. W., Taylor, S. H., & Stauton, H. P. (1967). Cardiovascular responses to sustained (static) contractions. *Circulation Research, S1*, 15-32.

Eblen-Zajjur, A. (2003). A simple ballistocardiographic system for a medical cardiovascular physiology course. *Advances in Physiology Education, 27*(1-4), 224-229.

Erol, M. K., & Karakelleoglu, S. (2002). Assessment of right heart function in the athlete's heart. *Heart Vessels, 16*(5), 175-180. doi: 10.1007/s003800200018

Esch, B. T., Bredin, S. S., Haykowsky, M. J., Scott, J. M., & Warburton, D. E. (2007). The potential role of the pericardium on diastolic filling in endurance-trained athletes under conditions of physiological stress. *Applied Physiology, Nutrition & Metabolism, 32*(2), 311-317. doi: 10.1139/h06-086

Fagard, R. H. (1997). Impact of different sports and training on cardiac structure and function. *Cardiology Clinics, 15*(3), 397-412.

Falsetti, H., Gisolfi, C., Lemon, D., Cohen, J., & Claxton, B. (1982). Noninvasive evaluation of left ventricular function in trained bicyclists. *The Journal of Sports Medicine and Physical Fitness, 22*(2), 199-206.

Fardy, P. S. (1971). The influence of physical activity on selected cardiac cycle time components. *The Journal of Sports Medicine and Physical Fitness, 11*(4), 227-233.

Fazel, P., Roberts, B. J., Brooks, J., & Graybum, P. A. (2009). Echocardiographic findings in professional hockey players. *Baylor University Medical Center Proceedings, 22*(3), 218-220.

Finkelhor, R. S., Hanak, L. J., & Bahler, R. C. (1986). Left ventricular filling in endurance-trained subjects. *Journal of the American College of Cardiology, 8*(2), 289-293.

Forman, D. E., Manning, W. J., Hauser, R., Gervino, E. V., Evans, W. J., & Wei, J. Y. (1992). Enhanced left ventricular diastolic filling associated with long-term endurance training. *Journal of Gerontology, 47*(2), M56-58.

Franks, B. D., & Cureton, T. K., Jr. (1969). Effects of training on time components of the left ventricle. *The Journal of Sports Medicine and Physical Fitness, 9*(2), 80-88.

Freund, P. R., Gobbs, S. F., & Rowell, L. B. (1979). Cardiovascular responses to muscle ischemia in man, dependency on muscle mass. *Journal of Applied Physiology, 45*, 762-767.

George, K., Shave, R., Warburton, D., Scharhag, J., & Whyte, G. (2008). Exercise and the heart: Can you have too much of a good thing? *Medicine & Science in Sports & Exercise, 40*(8), 1390-1392. doi: 10.1249/MSS.0b013e318172ceec

Germans, T., & van Rossum, A. C. (2008). The use of cardiac magnetic resonance imaging to determine the aetiology of left ventricular disease and cardiomyopathy. *Heart, 94*(4), 510-518. doi: 10.1136/hrt.2007.122770

Gledhill, N., Cox, D., & Jamnik, R. (1994). Endurance athletes' stroke volume does not plateau: Major advantage is diastolic function. *Medicine & Science in Sports & Exercise, 26*(9), 1116-1121.

Godin, G., & Shephard, R. J. (1985). A simple method to assess exercise behavior in the community. *Canadian Journal of Applied Sport Science, 10*(3), 141-146.

Gordon, J. W. (1877). Certain molar movements of the human body produced by the circulation of the blood. *Journal of Anatomy, 11*, 533-536.

Green, J. F. (1987). *Fundamental cardiovascular and pulmonary physiology*. Philadelphia, PA: Lea & Febiger.

Grubb, B. P., Temesy-Armos, P., Hahn, H., & Elliott, L. (1991). Utility of upright tilt-table testing in the evaluation and management of syncope of unknown origin. *American Journal of Medicine, 90*(1), 6-10.

Gubner, R. S., Rodstein, M., & Ungerleider, H. E. (1953). Ballistocardiography: An appraisal of technic, physiologic principles, and clinical value. *Circulation, 7*(2), 268-286.

Harris, K. M., Sponsel, A., Hutter, A. M., Jr., & Maron, B. J. (2006). Brief communication: Cardiovascular screening practices of major North American professional sports teams. *Annals of Internal Medicine, 145*(7), 507-511. doi: 145/7/507 [pii]

Heller, G. V., Calnon, D., & Dorbala, S. (2009). Recent advances in cardiac PET and PET/CT myocardial perfusion imaging. *Journal of Nuclear Cardiology, 16*(6), 962-969.

Herfkens, R. J., Higgins, C. B., Hricak, H., Lipton, M. J., Crooks, L. E., Lanzer, P., et al. (1983). Nuclear magnetic resonance imaging of the cardiovascular system: Normal and pathologic findings. *Radiology, 147*(3), 749-759.

Higgins, C. B. (2009). Early use of contrast in cardiac magnetic resonance. *JACC Cardiovascular Imaging, 2*(2), 241-244. doi: 10.1016/j.jcmg.2008.07.017

Holloszy, J. O., Skinner, J. S., Barry, A. J., & Cureton, T. K. (1964). Effect of physical conditioning on cardiovascular function. A ballistocardiographic study. *The American Journal of Cardiology, 14*, 761-770.

Holly, R. G., Shaffrath, J. D., & Amsterdam, E. A. (1998). Electrocardiographic alterations associated with the hearts of athletes. *Sports Medicine, 25*(3), 139-148.

Inan, O. T., Etemadi, M., Paloma, A., Giovangrandi, L., & Kovacs, G. T. (2009). Non-invasive cardiac output trending during exercise recovery on a bathroom-scale-based ballistocardiograph. *Physiological Measurement, 30*(3), 261-274.

Karamitsos, T. D., Arnold, J. R., Neubauer, S., & Petersen, S. E. (2007). Redefining cardiomyopathies: The role of cardiovascular magnetic resonance imaging. *European Heart Journal, 28*(24), 3094-3095. doi: 10.1093/eurheartj/ehm496

Kervancioglu, P., & Hatipoglu, E. S. (2007). Echocardiographic evaluation of left ventricular morphology and function in young male football players and runners. *Cardiology Journal, 14*(1), 37-43.

King, G. J., Murphy, R. T., Almuntaser, I., Bennett, K., Ho, E., & Brown, A. S. (2008). Alterations in myocardial stiffness in elite athletes assessed by a new Doppler index. *Heart, 94*(10), 1323-1325. doi: 10.1136/hrt.2008.142083

Korzeniowska-Kubacka, I., Bilinska, M., & Piotrowicz, R. (2005). Usefulness of seismocardiography for the diagnosis of ischemia in patients with coronary artery disease. *Annals of Noninvasive Electrocardiology, 10*(3), 281-287. doi: 10.1111/j.1542-474X.2005.00547.x

Krip, B., Gledhill, N., Jamnik, V., & Warburton, D. (1997). Effect of alterations in blood volume on cardiac function during maximal exercise. *Medicine & Science in Sports & Exercise, 29*(11), 1469-1476.

Krzeminski, K., Niewiadomski, W., & Nazar, K. (1989). Dynamics of changes in the cardiovascular response to submaximal exercise during low-intensity endurance training with particular reference to the systolic time intervals. *European Journal of Applied Physiology and Occupational Physiology, 59*(5), 377-384.

Lamont, L. S. (1980). Effects of training on echocardiographic dimensions and systolic time intervals in women swimmers. *The Journal of Sports Medicine and Physical Fitness, 20*(4), 397-404.

Le, V. V., Wheeler, M. T., Mandic, S., Dewey, F., Fonda, H., Perez, M., et al. (2010). Addition of the electrocardiogram to the preparticipation examination of college athletes. *Clinical Journal of Sport Medicine, 20*(2), 98-105.

Levy, W. C., Cerqueira, M. D., Abrass, I. B., Schwartz, R. S., & Stratton, J. R. (1993). Endurance exercise training augments diastolic filling at rest and during exercise in healthy young and older men. *Circulation, 88*(1), 116-126.

Libonati, J. R. (1999). Myocardial diastolic function and exercise. *Medicine & Science in Sports & Exercise, 31*(12), 1741-1747.

Libonati, J. R., Colby, A. M., Caldwell, T. M., Kasparian, R., & Glassberg, H. L. (1999). Systolic and diastolic cardiac function time intervals and exercise capacity in women. *Medicine & Science in Sports & Exercise, 31*(2), 258-263.

Lind, A. R., Taylor, S. H., Humphreys, P. W., Kennelly, B. M., & Donald, K. W. (1964). The circulatory effects of sustained voluntary muscle contraction. *Clinical Science, 27*, 229-244.

Lowenstein, A. S., Arbeit, S. R., & Rubin, I. L. (1962). Cardiac involvement in progressive muscular dystrophy: An electrocardiographic and ballistocardiograph study. *The American Journal of Cardiology, 9*(4), 528-533.

MacQuarrie, D. M., Gebhardt, V. A., & Neary, J. P. (2011). Comparison of seismocardiography to echocardiography for measuring cardiac cycle events *Book of Abstracts of the 16th Annual Congress of the European College of Sport Science*. Liverpool: ECSS.

Maron, B. J. (1997). Risk profiles and cardiovascular preparticipation screening of competitive athletes. *Cardiology Clinics, 15*(3), 473-483.

Maron, B. J. (2005). Distinguishing hypertrophic cardiomyopathy from athlete's heart: A clinical problem of increasing magnitude and significance. *Heart, 91*(11), 1380-1382. doi: 10.1136/hrt.2005.060962

Maron, B. J. (2009). Distinguishing hypertrophic cardiomyopathy from athlete's heart physiological remodelling: Clinical significance, diagnostic strategies and implications for preparticipation screening. *British Journal of Sports Medicine, 43*(9), 649-656. doi: 10.1136/bjsm.2008.054726

Maron, B. J., & Pelliccia, A. (2006). The heart of trained athletes: Cardiac remodeling and the risks of sports, including sudden death. *Circulation, 114*(15), 1633-1644.

McKay, W. P., Gregson, P. H., McKay, B. W., & Militzer, J. (1999). Sternal acceleration ballistocardiography and arterial pressure wave analysis to determine stroke volume. *Clinical and Investigative Medicine, 22*(1), 4-14.

McMullen, J. R., & Jennings, G. L. (2007). Differences between pathological and physiological cardiac hypertrophy: Novel therapeutic strategies to treat heart failure. *Clinical and Experimental Pharmacology and Physiology, 34*(4), 255-262. doi: 10.1111/j.1440-1681.2007.04585.x

Moore, R. L., & Palmer, B. M. (1999). Exercise training and cellular adaptations of normal and diseased hearts. *Exercise and Sport Sciences Review, 27*, 285-315.

Neary, J. P., Len, T. K., Busse, E. F., MacQuarrie, D. S., & Goodman, D. (2009). Using digital ballistocardiography to detect autonomic nervous system changes in the concussed athlete. *British Journal of Sports Medicine, 43 (Supplement)*, i93.

Neary, J. P., Len, T. K., MacQuarrie, D. M., & Busse, E. F. G. (2008). Cardiac changes during maximal exercise in varsity athletes using digital ballistocardiography. *Proceedings of the Physiological Society 11*, C93.

Neary, J. P., Len, T. K., MacQuarrie, D. M., Jamnik, V., Gledhill, N., & Busse, E. F. G. (2008). Cardiac cycle characteristics of elite prospect hockey players: A digital ballistocardiograph (dBG) study. *Applied Physiology, Nutrition & Metabolism, 33*(S1), S70.

Neary, J. P., MacQuarrie, D. M., & Busse, E. F. (2009). Cardiac cycle timing events are reliably measured day-to-day using digital ballistocardiography. *Proceedings of the Physiological Society 15*, PC202.

Neary, J. P., MacQuarrie, D. M., & Busse, E. F. (2010). Cardiac contractility and performance can be reliably measured day-to-day using digital ballistocardiography. *Medicine & Science in Sports & Exercise, 42*(S1), 538. doi: 10.1249/01.MSS.0000385322.24044.0c

Neary, J. P., MacQuarrie, D. M., & Busse, E. F. G. (2008). Impaired cardiac cycle timing events post-marathon as detected using digital ballistocardiography. *Physiologist, 51*(6), 33.

Neary, J. P., MacQuarrie, D. M., Jamnik, V., Gledhill, N., Gledhill, S., & Busse, E. F. G. (2009). Hypertrophic cardiomyopathy in an elite hockey player: Using digital ballistocardiography to confirm echocardiography and MRI. *Applied Physiology, Nutrition & Metabolism, 34*(S1), S67.

Ngai, B., Tavakolian, K., Akhbardeh, A., Blaber, A. P., Kaminska, B., & Noordergraaf, A. (2009). Comparative analysis of seismocardiogram waves with the ultra-low frequency ballistocardiogram. *Proceedings of the 31st IEEE Engineering in Medicine and Biology Conference*, 2851-2854.

Nixon, J. V., Wright, A. R., Porter, T. R., Roy, V., & Arrowood, J. A. (1991). Effects of exercise on left ventricular diastolic performance in trained athletes. *The American Journal of Cardiology, 68*(9), 945-949.

Noordergraaf, A. (1961). Further studies on a theory of the ballistocardiogram. *Circulation, 23*, 413-425.

Otto, C. M. (2004). *Textbook of clinical echocardiography*. Philadelphia: Elsevier Saunders.

Pelliccia, A., & Maron, B. J. (1995). Preparticipation cardiovascular evaluation of the competitive athlete: Perspectives from the 30-year Italian experience. *American Journal of Medicine, 75*(12), 827-829.

Pennell, D. J. (2010). Cardiovascular magnetic resonance. *Circulation, 121*, 692-705.

Petridis, L., Kneffel, Z., Kispeter, Z., Horvath, P., Sido, Z., & Pavlik, G. (2004). Echocardiographic characteristics in adolescent junior male athletes of different sport events. *Acta Physiologica Hungarica, 91*(2), 99-109. doi: 10.1556/APhysiol.91.2004.2.2

Phibbs, B., Lowe, C. R., & Holmes, R. W. (1967). The ultra low frequency force ballistocardiograph in acute cardiomyopathy. *Circulation, 36*(1), 92-100.

Pinheiro, E., Postolache, O., & Girao, P. (2010). Theory and developments in an unobtrusive cardiovascular system representation: ballistocardiography. *The Open Biomedical Engineering Journal, 4*, 201-216. doi: 10.2174/1874120701004010201

Plowman, S. A., & Smith, D. L. (2003). *Exercise physiology for health, fitness, and performance*. Toronto: Benjamin Cummings.

Poliac, M. O., Zanetti, J. M., & Salerno, D. M. (1991). Performance measurements of seismocardiogram interpretation using neural networks. *Computers in Cardiology, 18*, 573-576.

Powers, S. K., & Howley, E. T. (2007). *Exercise physiology: Theory and application to fitness and performance*. Toronto: McGraw-Hill.

Prakken, N. H., Velthuis, B. K., Cramer, M. J., & Mosterd, A. (2009). Advances in cardiac imaging: the role of magnetic resonance imaging and computed tomography in identifying athletes at risk. *British Journal of Sports Medicine, 43*(9), 677-684. doi: 10.1136/bjsm.2008.054767

Rimbaud, S., Garnier, A., & Ventura-Clapier, R. (2009). Mitochondrial biogenesis in cardiac pathophysiology. *Pharmacological Reports, 61*(1), 131-138.

Rosenblatt, W. H. (1957). Ballistocardiography: Concepts of its applicability in the office practice of cardiology. *Diseases of the Chest, 32*(4), 400-412.

Rost, R. (1982). The athlete's heart. *European Heart Journal, 3*(Supplement A), 193-198.

Rost, R. (1997). The athlete's heart. Historical perspectives: Solved and unsolved problems. *Cardiology Clinics, 15*(3), 493-512.

Rost, R., & Hollmann, W. (1983). Athlete's heart--a review of its historical assessment and new aspects. *International Journal of Sports Medicine, 4*(3), 147-165. doi: 10.1055/s-2008-1026028

Rost, R., & Hollmann, W. (1992). Cardiac problems in endurance sports. In R. J. Shepard & P. O. Astrand (Eds.), *The encyclopaedia of sports medicine: Endurance in sport*. Boston: Human Kinetics

Rowell, L. B. (1986). Human circulation: Regulation during physical stress. New York: Oxford University Press.

Rowell, L. B. (1993). Human cardiovascular control. New York: Oxford University Press.

Royse, C. F., Royse, A. G., Wong, C. T., & Soeding, P. F. (2003). The effect of pericardial restraint, atrial pacing, and increased heart rate on left ventricular systolic and diastolic function in patients undergoing cardiac surgery. *Anesthesia and Analgesia, 96*(5), 1274-1279.

Saladin, K. S. (2007). *Anatomy & physiology: The unity of form and function*. New York: McGraw-Hill.

Salerno, D. M., Zanetti, J. M., Green, L. A., Mooney, M. R., Madison, J. D., & Van Tassel, R. A. (1991). Seismocardiographic changes associated with obstruction of coronary blood flow during balloon angioplasty. *The American Journal of Cardiology, 68*(2), 201-207.

Scarborough, W. R. (1955). Ballistocardiogram in the diagnosis of coronary atherosclerosis. *Minnesota Medicine, 38(12)*(12), 880-887.

Scarborough, W. R., Mason, R. E., Davis, F. W., Jr., Singewald, M. L., Baker, B. M., Jr., & Lore, S. A. (1952). A ballistocardiographic and electrocardiographic study of 328 patients with coronary artery disease: Comparison with results from similar study of apparently normal persons. *American Heart Journal, 44*(5), 645-670.

Seals, D. R., Washburn, R. A., & Hanson, P. G. (1985). Increased cardiovascular response to static contraction of larger muscle groups. *Journal of Applied Physiology, 54*(2), 434-437.

Sharma, S., Maron, B. J., Whyte, G., Firoozi, S., Elliott, P. M., & McKenna, W. J. (2002). Physiologic limits of left ventricular hypertrophy in elite junior athletes: Relevance to differential diagnosis of athlete's heart and hypertrophic cardiomyopathy. *Journal of the American College of Cardiology, 40*(8), 1431-1436.

Sjogaard, G., Savard, G., & Juel, C. (1988). Muscle blood flow during isometric activity and its relation to muscle fatigue. *European Journal of Applied Physiology and Occupational Physiology, 57*(3), 327-335.

Smith, D. L., Misner, J. E., Bloomfield, D. K., & Essandoh, L. K. (1993). Cardiovascular responses to sustained maximal isometric contractions of the finger flexors. *European Journal of Applied Physiology and Occupational Physiology, 67*(1), 48-52.

Smith, S. A., Humphrey, R. H., Wohlford, J. C., & Flint, D. L. (1994). Myocardial adaptation and weight fluctuation in college wrestlers. *International Journal of Sports Medicine, 15*(2), 70-73. doi: 10.1055/s-2007-1021022

Starr, I. (1955). Normal standards for amplitude of ballistocardiograms calibrated by force. *Circulation, 11*(6), 914-926.

Starr, I. (1965). Progress towards a physiological cardiology: A second essay on the ballistocardiogram. *Annals of Internal Medicine, 63*(6), 1079-1105.

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. *International Journal of Sports Medicine, 13*(8), 600-604. doi: 10.1055/s-2007-1024573

Talbot, S. A. (1958). Biophysical aspects of ballistocardiography. *The American Journal of Cardiology, 2*(4), 395-403.

Talbot, S. A., & Harrison, W. K., Jr. (1955). Dynamic comparison of current ballistocardiographic methods. I. Artifacts in the dynamically simple ballistocardiographic methods. *Circulation, 12*(4), 577-587.

Thaler, M. S. (2010). *The only EKG book you'll never need* (6th ed.). Philadelpha, PA Lippincott Williams & Wilkins.

Tuttle, W. W., & Horvath, S. M. (1957). Comparison of effects of static and dynamic work on blood pressure and heart rate. *Journal of Applied Physiology, 10*(2), 294-296.

Underwood, R. H., & Schwade, J. L. (1977). Noninvasive analysis of cardiac function of elite distance runners: Echocardiography, vectorcardiography, and cardiac intervals. *Annals of the New York Academy of Sciences, 301*, 297-309.

Vogt, E. S. M., Neary, J. P., MacQuarrie, D. M., Len, T. K., & Busse, E. F. (2009). Detecting effects of obesity and diabetes mellitus on cardiac performance using digital ballistocardiography. *Applied Physiology, Nutrition & Metabolism, 34*(S1), S99.

Vogt, E. S. M., Neary, J. P., MacQuarrie, D. S., & Len, T. K. (2010). Detecting effects of physical training on cardiac performance using noninvasive ballistocardiography. *Applied Physiology, Nutrition & Metabolism, 35*(S1), S107-108.

Warburton, D. E., Gledhill, N., Jamnik, V. K., Krip, B., & Card, N. (1999). Induced hypervolemia, cardiac function, VO_2max, and performance of elite cyclists. *Medicine & Science in Sports & Exercise, 31*(6), 800-808.

Warburton, D. E., Haykowsky, M. J., Quinney, H. A., Blackmore, D., Teo, K. K., & Humen, D. P. (2002). Myocardial response to incremental exercise in endurance-trained athletes: Influence of heart rate, contractility and the Frank-Starling effect. *Experimental Physiology, 87*(5), 613-622.

Wijeysundera, D. N., Beattie, W. S., Austin, P. C., Hux, J. E., & Laupacis, A. (2010). Non-invasive cardiac stress testing before elective major non-cardiac surgery: Population based cohort study. *British Medical Journal, 340*, 5526.

Zanetti, J. M., Poliac, M. O., & Crow, R. S. (1991). Seismocardiography: Waveform identification and noise analysis. *Computers in Cardiology, 18*, 49-52.

APPENDIX A – NOTRE DAME HOUNDS TRAINING PROGRAM

Fillers
4 Point Hip Mobility
Fire Hydrant
Half Kneeling Wall Hip Flexor Mobilization
Ankle Mobilization
Bent Over T-Spine Rotation
Quadruped Extension-Rotation
Kneeling Rockback Mobilization
Knee Punches
Y,T,W
Lying Knee to Knee Stretch
Internal Rotation

Movement Preparation Exercises
Hip Crossover - 6/side
Scorpion - 4/side
Hand Walk - 5
Inverted Hamstring - 4/leg
Forward Lunge/Forearm to Instep- 4/leg
Backward Lunge with Twist- 6/leg
Drop Lunge- 5/side
Lateral Lunge- 5/side
Sumo Squat-to-Stand- 8

Note. All information regarding the Notre Dame Junior A Hockey Program was provided by Brad Posehn B.S.c. Kin, CEP, who is employed at the Athol Murray College of Notre Dame in Wilcox, SK.

Load Definition

Heavy: Need a spotter to complete the final 1 or 2 repetitions.

Medium: Able to complete all repetitions with perfect technique, but would not be able to complete 2 more.

Day 1 - Speed/Strength Workout

Hip Mobility Sequence
Anterior Posterior Step-Overs - 5 reps
Lateral Step-Overs - 5 reps
Lateral Duck-Unders - 5 reps
Lateral Under-Overs - 4 reps/side
Lateral Duck-Under to Warrior Lunge - 4 reps/side
Band Stomps - 10 reps/leg

Exercises	Sets x Reps	Load	Tempo	Rest
Dynamic Warm-up (movement preparation)				
Hang Clean	4 x 5	75%	Explosive	3 minutes
Box Jumps	4 x 5	Bodyweight	Explosive	
BB Bench-Press (feet in air)	4 x 5	Heavy	Fast	
Wide Grip Pull-ups	4 x 6	Heavy	Fast	
Held Squat Face Pulls on One Leg	4 x 8	Medium	Fast	2 minutes
Bulgarian Split Squats	4 x 5/leg	Heavy	Fast	
Natural Glute-Ham Raises	4 x 5	Bodyweight	Fast	2 minutes
Cool Down				
10 minute bike HR < 140 bpm				

Day 2 Strength/Speed Workout				
Exercises	**Sets x Reps**	**Load**	**Tempo**	**Rest**
Dynamic Warm-up (movement preparation)				
Power Clean (work up to your 2 rep max)	4 x 2	90-95%	Explosive	
Single Leg Hops	4 x 4/leg	Body Weight	Explosive	3 minutes
Push Press	4 x 4	Medium	Explosive	
Chin-ups	4 x 6	Medium	Fast	
One Arm/Leg Bent Over Row	3 x 6/side	Light	Fast	2 minutes
Squat Jumps	3 x 4	20%	Explosive	
Good Mornings	3 x 10	Light	Fast	2 minutes
Cool Down				
10 minutes on bike HR < 140 bpm				

APPENDIX B – INFORMED CONSENT FORM (NOTRE DAME)

Consent for Subjects to Participate in this Research Project

Title of Project: ***Using digital ballistocardiography to measure cardiac events during exercise.***

Principal Investigators:

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The university and those conducting this project subscribe to the ethical conduct of research, and at all times to protect the interests, comfort, and safety of subjects. This form and the information it contains are given to you for your protection and full understanding of the procedures being conducted in this research. Your signature on this form will signify that you have received a document that describes the procedures, possible risks and benefits of this research, and that you have received an adequate opportunity to consider the information in this document, and that you voluntarily agree to participate in this project.

Purpose: This project is aimed at understanding the differences between the athletic, diseased, and normal heart. The digital ballistocardiography (dBG) device is an accelerometer and has the ability to record the movement and forces generated by the heart during each heart beat. Therefore, with this device we are able to record the timing of the events associated with the opening and closing of the heart valves, which will allow us to make comparisons between different groups of subjects that participate in this research to gain a greater insight on how the heart adapts to different types of exercise training.

Procedures: This testing will be performed in a laboratory setting that will ensure your privacy. This testing will last approximately one-half hour in duration. The test session will require that you complete an exercise test. The test that you perform is dependent on what group you are in.

1. Aerobic trained group – you will perform a maximal treadmill or cycle ergometer (VO_2max) test lasting approximately 10-14 minutes. This test will not be any more physical than what you will normally experience during your sport.
2. Strength trained group – you will perform a 10 repetition maximum (10RM) bench press with a barbell.
3. Chronic disease group – you will perform a 6 minute (self-paced) walk test. This test is well accepted in the literature for testing subjects with chronic disease.
4. Sedentary control group – you will perform a maximal treadmill or cycle ergometer (VO_2max) test lasting approximately 10-14 minutes.

These tests have been used in thousands of research studies on all groups of subjects, and Dr. Neary and Dr. Candow have performed hundreds of these tests in the past.

Potential Risks and Discomforts: The exercise protocol is designed to elevate your heart rate to a level that you are capable of performing during the volitional exercise test. If you feel, at any time or for any reason, that you need to stop exercise, you may do so without penalty. The dBG device is an accelerometer which is non-invasive and there are no known risks associated with this device and pose no known or foreseeable risks to you.

Possible Benefits to Subject and/or Society: This research study may provide you with an opportunity to gain some knowledge and education about the health of your heart. Your participation in this study may also assist us, the researchers, in determining how the heart adapts to exercise training.

Confidentially: Any information that is obtained during this study will be kept confidential to the full extent permitted by law. The researchers will guarantee complete confidentiality with all aspects of this study. Subjects will be given a unique identification number and any information provided will be marked with this ID number. Only group means will be reported in any published documents and thus ensuring subject anonymity. All materials will be locked in the office of Dr. Patrick Neary, with access available to Dr. Darren Candow and graduate student Trevor Len. In the event that that the researchers find evidence that the subject may have cardiac complications (for the cardiac group, beyond what is currently known about their condition) the information will be passed on to your family doctor and/or team's medical physician for your safety.

Participants and Withdrawal: If you feel, at any time, that you would like to withdraw from the research study, you may do so freely and without consequence. You also have the option to remove your data from the research study at any time.

Feedback: You will have full access to your individual test results, upon request, once the data has been analyzed.

Questions: If, at any time, you have questions about this study, feel free to ask any of the investigators. If you wish to speak to someone not associated with this project you may contact the Research Ethics Committee at the University of Regina, Phone: (306) 585-4775; Email: research.ethics@uregina.ca

Participation is voluntary and you may withdraw at any time without penalty. Please complete the section on the following page. Thank you for your participation!

CONSENT FOR SUBJECTS TO PARTICIPATE IN THIS RESEARCH PROJECT

Title of Project*:* ***Using digital ballistocardiography to measure cardiac events during exercise.***

- I understand that my participation in this study is voluntary and that I may withdraw my participation in this experiment at any time, without any consequences.

- I am aware that I will be expected to disclose any previous medical information for my safety.

- I have been informed that all information collected from me will be treated confidentially, and will be locked in the desk of the principal investigator, Dr. Patrick Neary, with access available to Dr. Darren Candow and graduate student Trevor Len.

- I have been assured that I may contact the principle investigator, Dr. Patrick Neary, at patrick.neary@uregina.ca or (306-585-4844) at any time if I have questions or would like more information about this research study. I may obtain a copy of my results, upon completion of the study, by contacting any of the above mentioned researchers.

- I understand that I may register any concerns I might have about this experiment with Research Ethics Committee, University of Regina, Phone: (306) 585-4775; Email: research.ethics@uregina.ca.

- I understand the contents of this form, and I agree to participate in this research study during the period of May 2008 to May 2009.

- I have received a copy of the information sheet and this informed consent form for my records.

NAME (Please print legibly): _____

ADDRESS: _____

SIGNATURE: _____ **PARENT/GUARDIAN:** _____

DATE: _____ **WITNESS:** _____

FAMILY DOCTOR: _____ **PHONE:** _____

APPENDIX C – INFORMED CONSENT FORM (CONTROL)

Informed Consent Form

Title of the study: Effect of resistance-exercise and creatine application strategies on muscle function and exercise performance.

Names of Researchers:
Darren G. Candow, Ph.D., CEP, Assistant Professor, Faculty of Kinesiology and Health Studies, Ph. (306) 585- 4906, E-mail: Darren.Candow@uregina.ca

Patrick Neary, Ph.D., CEP, Professor, Faculty of Kinesiology and Health Studies, E-mail: Patrick.Neary@uregina.ca

Kristie Mueller, Honors Student, Faculty of Kinesiology and Health Studies, E-mail: muellerk@uregina.ca

Purpose of the study: To determine if creatine supplementation during short-term equal-volume resistance exercise training 2 days per week is as effective as 3 days per week on muscle mass, strength and exercise performance. Creatine is a nitrogen-containing compound naturally produced in the body and/or consumed in the diet from red meat and seafood and derived from reactions involving the amino acids glycine, arginine, and methionine. Creatine supplementation, in combination with resistance-exercise, has been shown to result in significant muscle mass leading to greater muscle strength.

Possible benefits of the study: You may increase muscle mass, strength and exercise performance. These benefits are not guaranteed.

Procedures: You will initially be given a questionnaire (the physical activity readiness questionnaire) which assesses whether you are at a health risk from participating in exercise training. If there are possible health risks, we will require you to have permission from your family physician to participate in the study.

You will be randomized (by chance) into one of four groups: Group 1 will receive 0.15g/kg body mass of creatine during 2 days per week of exercise, Group 2 will receive 0.10 g/kg body mass creatine during 3 days per week of exercise, Group 3 will receive placebo (rice flour; 0.15g/kg body mass during 2 days per week of exercise, and Group 4 will receive placebo (0.10g/kg body mass during 3 days per week of exercise). The creatine and placebo capsules will be identical in energy content, taste, texture and appearance. Neither you nor the researchers will know which group you are in until the end of the study.

You will be required to come into the laboratory on three occasions. During the first visit to the laboratory, you will perform a familiarization trial of the bench press and leg press. This will take approximately 30 minutes. No supplementation will occur at this time. Prior to and following the study, you will come back to the laboratory and have your body composition, cardiovascular function, and strength tested in order. This pre- and post-testing will take approximately 1.5 hours. Following the baseline testing session, you will be given your supplement to be consumed only on exercise training days. Each day's workout session will take approximately 1.5 hours and you will be shown the proper technique and routine for each exercise. Each days supplement will be consumed in two equal doses each training day (half before and half after exercise). After the 6-week period, you will return to the laboratory for post-testing.

All groups will participate in 6 weeks of strength training. Training will occur 2 or 3 days per week, depending on random assignment, and each training session will last for 1-1.5 hours and will require you to perform 9 different exercises designed to train all your major muscle groups.

Your body composition (lean tissue and fat mass) will be measured twice: prior to the start of the study and at the end of training (6 weeks). Body composition will be assessed by a technique called "air displacement plythesmography". This measurement requires that you sit still in a chamber. Your body density will be determined by the amount of air displaced from the chamber. Your body density is then used to estimate your lean tissue and fat mass. This measurement takes about 10 minutes.

Your muscle thickness will be measured twice: prior to the start of the study and at the end of training (6 weeks). Muscle thickness will be measured using ultrasound by placing a gel over your skin and applying a probe to your skin surface. Muscle thickness will be measured at the front and back of your right upper arm and upper leg. This procedure will take 20-30 minutes.

Your cardiovascular function will be assessed prior to and following the study (6 weeks) using digital ballistocardiography (dBG). The dBG device is an accelerometer which is placed on the chest and records movement and measures the forces generated by the contraction and relaxation of the heart. This procedure will take 5 minutes.

Your muscular strength will be measured for two different exercises (bench press, leg press) prior to and following the study (6 weeks). This procedure will take 30 minutes.

You will be required to collect urine for 24 hours at two time points: prior to the start of the study and at the end of training (6 weeks). Prior to this urine collection, you will have to consume a meat-free diet for 3 days. The purpose of

the urine collection is to measure a marker of muscle protein breakdown. Meat consumption affects the level of this marker; therefore, three days without meat is required.

You will be required to record all the food you eat in a food diary, for three days, at the start and end of the study.

Foreseeable risks, side effects or discomfort:
The exercise training and strength testing may result in muscle pulls or strains. You will be given a proper warm-up prior to exercising and this will minimize this risk. You may feel claustrophobic inside the body composition chamber, but there is a window in the chamber, through which you can look. This will minimize this risk.

Creatine supplementation, on occasion, has been shown to be associated with minimal side effects, especially with the low dose given in this study. There have been anecdotal reports of increased muscle cramping or muscle pulls during high-does creatine supplementation, but when this is compared to subjects receiving placebo, there is no differences in rates of occurrence of muscle cramping or pulls. Creatine supplementation has been shown, on two occasions, to worsen kidney function in individuals who already had kidney disease. If you have any problems with kidney function you should not participate in this study. There may be unknown or unforeseen risks associated with the training or creatine supplementation.

Alternatives to this study:
You do not have to participate in this study to increase your muscle mass and strength. You can perform alternative exercises (i.e. free-body exercises such as push-ups or chin-ups instead of the exercise program in this study). You could also increase your creatine consumption from your diet by consuming more red meat and seafood.

Confidentiality: All data collected will be kept confidential in a locked storage cabinet and in password-protected computer files only the researchers can access. Your name will not be attached to any information, nor mentioned in any study report, nor be made available to anyone except the research team. It is the intention of the research team to publish results of this research in a scientific journal and to present the findings at related conferences and workshops, but your identity will not be revealed. Results from this study will not be used for commercial purposes.

Voluntary Participation: If you decide to participate you must sign and return this consent form. Before making the decision to participate, please review this form carefully and discuss it with family, friends, and/or your physician.

Voluntary Withdrawal: Your participation in this research is entirely voluntary. You may withdraw from this study at any time. If you decide to enter the study and to withdraw at any time in the future, there will be no penalty or loss of benefits to which you are otherwise entitled. If you choose to enter the study and then decide to withdraw at a later time, all data collected about you during the enrolment in the study will be retained for analysis.

Ethics Approval: This project was approved by the Research Ethics Board, University of Regina. If you have any questions or concerns about your rights or treatment as a research participant, you may contact Dr. Darren Candow at (306) 585-4906 or the Chair of the University of Regina Research Ethics Board at 585-4775 or by e-mail at research.ethics@uregina.ca.

Your results may serve as a personal indication of aerobic fitness and/or changes in performance due to caffeine supplementation. If you would like your results mailed to you, please provide a complete mailing address:

_____
_____
_____

Consent statement

Having read the above, I agree to participate in this study and content to the above. Moreover, I agree not to disclose any information that could be linked to any specific individual. I will also not disclose any identifying information about other members of the testing group. I acknowledge that I have received a copy of this form.

_____ _____
Signature of participant Date

_____ _____
Signature of researcher Date

_____ _____
Signature of witness Date

APPENDIX D – ETHICS APPROVAL

UNIVERSITY OF REGINA

OFFICE OF RESEARCH SERVICES

MEMORANDUM

DATE: May 4, 2010

TO: Dr. J. Patrick Neary
Faculty of Kinesiology and Health Studies

FROM: Dr. Bruce Plouffe
Chair, Research Ethics Board

Re: **Using Ballistocardiography to Measure Cardiac Performance in Elite Ice Hockey Players (File # 75R0910)**

Please be advised that the University of Regina Research Ethics Board has reviewed your proposal and found it to be:

☑ 1. APPROVED AS SUBMITTED. Only applicants with this designation have ethical approval to proceed with their research as described in their applications. For research lasting more than one year (Section 1F). **ETHICAL APPROVAL MUST BE RENEWED BY SUBMITTING A BRIEF STATUS REPORT EVERY TWELVE MONTHS.** Approval will be revoked unless a satisfactory status report is received. Any substantive changes in methodology or instrumentation must also be approved prior to their implementation.

☐ 2. ACCEPTABLE SUBJECT TO MINOR CHANGES AND PRECAUTIONS (SEE ATTACHED). Changes must be submitted to the REB and approved prior to beginning research. Please submit a supplementary memo addressing the concerns to the Chair of the REB.** Do not submit a new application. Once changes are deemed acceptable, ethical approval will be granted.

☐ 3. ACCEPTABLE SUBJECT TO CHANGES AND PRECAUTIONS (SEE ATTACHED). Changes must be submitted to the REB and approved prior to beginning research. Please submit a supplementary memo addressing the concerns to the Chair of the REB.** Do not submit a new application. Once changes are deemed acceptable, ethical approval will be granted.

☐ 4. UNACCEPTABLE AS SUBMITTED. The proposal requires substantial additions or redesign. Please contact the Chair of the REB for advice on how the project proposal might be revised.

D. Bruce Plouffe
Dr. Bruce Plouffe

** supplementary memo should be forwarded to the Chair of the Research Ethics Board at the Office of Research Services (Research and Innovation Centre, Room 109) or by e-mail to research.ethics@uregina.ca

Phone: (306) 585-4775
Fax: (306) 585-4893
www.uregina.ca/research

VITA

Candidate's Full Name: Emelie Sara Maria Vogt
emelie.vogt@uregina.ca

University Attended:

University of Regina
Faculty of Kinesiology and Health Studies
2009 – 2011
Master of Science (Kinesiology and Health Studies)

University of Regina
Faculty of Kinesiology and Health Studies
2005 – 2009
Bachelor of Kinesiology (Fitness and Lifestyle)

Publications:

Vogt, E. S. M., Len, T. K., MacQuarrie, D. S., Candow, D. G., Neary, J. P. (submitted for publication). Evaluating cardiac cycle timing events in male ice hockey players using ballistocardiography.

Vogt, E. S. M., Len, T. K., MacQuarrie, D. S., Candow, D. G., Neary, J. P. (submitted for publication). Assessing cardiac cycle mechanics in male ice hockey players using ballistocardiography.

Vogt, E. S. M., MacQuarrie, D. S., Neary, J. P. (submitted for publication). Non-invasive screening techniques to monitor cardiac function.

Abstracts:

Vogt, E. S. M., Neary, J. P., MacQuarrie, D. S., Len, T. K. (2010). Detecting effects of physical training on cardiac performance using non-invasive ballistocardiography. *Applied Physiology, Nutrition & Metabolism, 35*(S1), S107.

Len, T. K., Neary, J. P., MacQuarrie, D. S., **Vogt, E. S. M.**, Goodman, D. G., Busse, E. F. G. (2009). Detecting changes in autonomic function following sport concussion in adolescent male athletes using digital ballistocardiography. *Applied Physiology, Nutrition & Metabolism, 34*(S1), S55-56.

Vogt, E. S. M., Neary, J. P., MacQuarrie D. S., Len, T. K., Busse, E. F. G. (2009). Detecting effects of obesity and

diabetes mellitus on cardiac performance using digital ballistocardiography. *Applied Physiology, Nutrition & Metabolism, 34* (S1), S99.

Presentations:

Vogt, E. S. M., Neary, J. P., MacQuarrie, D. S., Len, T. K. (2010). Detecting effects of physical training on cardiac performance using non-invasive ballistocardiography. Presented at the CSEP Conference, Toronto, ON.

Vogt, E. S. M., Neary, J. P., MacQuarrie, D. S., Len, T. K., Busse, E. F. G. (2009). Detecting effects of obesity and diabetes mellitus on cardiac performance using digital ballistocardiography. Presented at the CSEP Conference, Vancouver, BC.