EFFECTS OF EXERCISE TRAINING ON HEART FAILURE MEASURED USING SEISMOCARDIOGRAPHY

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Jonathan Eric Sibernagel, candidate for the degree of Master of Science in Kinesiology & Health Studies, has presented a thesis titled, *Effects of Exercise Training on Heart Failure Measured Using Seismocardiography*, in an oral examination held on November 25, 2019. The following committee members have found the thesis acceptable in form and content, and that the candidate demonstrated satisfactory knowledge of the subject material.

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Abstract

Heart failure has become a growing health concern across most of the Western world and will soon become a global health focus. The nature of the disease presents with a very high mortality rate. In Canada alone, the average mortality rate is approximately 50% within the first five years of diagnosis [1]. Exercise has now become a common treatment modality for many chronic disease conditions including heart failure. As heart failure advances, the myocardium goes through a remodeling phase that alters the contractility of the heart and its pumping efficiency. Exercise is known to lead to a positive remodeling of the myocardium in healthy populations, but the degree to which exercise reverses pathological remodeling in individuals with heart failure remains to be determined.

The purpose of this study was to analyze the Systolic Timing Intervals (STI) of the myocardium, specifically left ventricular ejection time (LVET) and the pre-ejection period (PEP) using seismocardiography (SCG), as well as to investigate the functional health changes measured using the six-minute walk test (6MWT). Participants exercised three times per week for a period of 12 weeks involving a combination of aerobic and resistance type activities. The SCG screening and 6MWT were performed at the commencement, and at the 12 week point of their exercise program. Eleven individuals participated in this study with varying etiologies for heart failure (ischemic n=6 and non-ischemic n=5). Among the individuals with ischemic heart failure, significant improvement were observed in 6MWT distance, (477.0±127.0m to 539.3±113.9m t(5)=-3.01, p=0.030), but no significant improvements were noted in indices of myocardial function. However, in the non-ischemic group, significant changes were noted in indices
of myocardial function including LVET (449.6±36.0ms to 438.4±30.5ms t(149)=4.28, p=<0.001), and PEP (128.0±23.5ms to 119.9±18.5ms t(149)=6.87, p=<0.001), but no statistically significant changes were observed in 6MWT distance (p=0.056). This study showed that SCG can be used to record the mechanical function of the heart in individuals with heart failure, that exercise training can produce positive mechanical changes to the heart for individuals with non-ischemic heart failure, and exercise capacity can increase in for individuals with ischemic based heart failure.
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To David Mac Quarrie, thank you for helping me with the nuances of the seismocardiography. You helped me put theory into practice and navigate the many roads of cardiac physiology.
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List of Abbreviations

HF – Heart Failure
MI – Myocardial Infarction
CVD – Cardiovascular Disease
QOL – Quality of Life
LV – Left Ventricle
ADL – Activities of Daily Living
NYHA – New York Heart Association
Q – Cardiac Output
SV – Stroke Volume
HR – Heart Rate
EF – Ejection Fraction
EDV – End Diastolic Volume
HRR – Heart Rate Reserve
LVH – Left Ventricular Hypertrophy
VO2 – Oxygen Uptake
RM – Repetition Maximum
ACE – Angiotensin Converting Enzyme
ECHO – Echocardiography
MRI – Magnetic Resonance Imaging
ECG – Electrocardiography
SCG – Seismocardiogram
BCG – Ballistocardiogram
STI – Systolic Timing Intervals
PEP – Pre Ejection Period

LVET – Left Ventricular Ejection Time

AVO – Aortic Valve Open

6MWT – 6 Minute Walk Test

BMI – Body Mass Index

AVC – Aortic Valve Close

RPE – Rating of Perceived Exertion

MCID – Minimal Clinical Important Difference
Effects of Exercise Training on Heart Failure Measured using Seismocardiography

CHAPTER 1: Introduction

Heart failure (HF) is the pathophysiological state in which the heart is unable to pump blood at a rate commensurate with the requirements of the metabolizing tissue [2-4]. Heart failure represents a growing health concern due to extreme disability that the disease places on the individual. Individuals with HF often reduce their level of activity and adopt sedentary lifestyles and as a result of the symptoms associated with the condition, this leads to further deconditioning of the cardiovascular and musculoskeletal systems [5]. By promoting physical activity, it is possible to break this vicious cycle [6].

The disease process that leads up to the development of HF can be quite complex. Typically HF can develop one of two ways; development of a cardiomyopathy or from a myocardial infarction (MI). Cardiomyopathy presents in three distinct forms; hypertrophic, restrictive, and dilated. Each etiology of cardiomyopathy impacts the myocardium in a different manner but the end result leaves the heart unable to fulfill the body’s metabolic demands (discussed below). As an individual develops a cardiomyopathy, if a treatment regime is not put in place, there can be a shift from cardiomyopathy to permanent HF. Cardiomyopathy can either be acquired through lifestyle or be congenital in nature; either way the outcomes can lead to an increase in morbidity and ultimately mortality.

1.1 Prevalence of heart failure in North America

Heart failure represents a growing health care concern for North America and most developed and developing countries. The Heart and Stroke Association of Canada estimated that in 2016, 600,000 people were living with HF [7] and the numbers are on
the rise (Figure 1). The increase in HF can be attributed to a number of factors; first of all, HF is a disease of age where the greatest incidence occurs in the 7th decade of life [2].

The recent advances in the health care systems, across many developed nations, have led to an increase in life expectancy across all genders. However, the debate persists as to whether the added life expectancy produces an increase in healthy years or an increase in years of disease. This added life expectancy and the onset of a large Canadian and American population entering the 7th decade of life will lead to more people being susceptible to HF. For example, the population of the individuals aged 65 and older in the United States is expected to grow from 35 million in 2000 to 70.3 million by 2030 [7]. With an average HF prevalence of 2.2% among the American population, and increasing from 0.7% in persons aged 45 through 54 years to 8.4% for those aged 75 years or older [8], this leads to a potential increase in cases of HF across North America in the years to come (Figure 2).

Another theory as to the possible increase in incidence of HF is due to the advances in therapies for cardiovascular disease (CVD). The ability for individuals to better manage CVD risk factors (e.g., hypercholesterolemia, high blood pressure and type2 diabetes) have lengthened survival of patients, which creates a lengthened time under disease, and therefore, exposure of patients to cardiovascular risk factors [6]. Additionally, the advances in medical care for the treatment and diagnosis of MIs may also be contributing to the growing incidence of HF. More people are now able to survive more MIs than previously [5], which leaves a greater number of individuals with damaged hearts and more susceptibility to the development of HF. As a result, the incidence and prevalence of HF is increasing [9].
Figure 1. Congestive heart failure prevalence is expected to double by the year 2030 due to the large "baby boomer" aging population [1].
Figure 2. Number of hospitalizations for CHF (actual and projected) in Canada 1980-2025 [2].
The increasing prevalence of HF leads to not only the increase of disability among the population, but places a large economic burden on the health care system. Due to the complexity and severity of HF, the disease process often leads to frequent hospitalizations [3], reduced quality of life (QOL) and premature death [10], producing a 5-year mortality rate of HF of close to 50% [1]. Unfortunately, the treatment of HF costs money. The American Heart Association estimated that in 2010, HF cost the United States $39.2 billion in direct health care costs, medications, and lost productivity [9].

1.2 Aetiology of heart failure

As mentioned previously, HF can develop by way of MI or cardiomyopathy. Cardiomyopathy is a disease of the myocardium and presents in three distinct forms: hypertrophic, restrictive and dilated.

Hypertrophic cardiomyopathy is primarily present as a hereditary disease where a disordered growth of the left ventricle (LV), and sometimes the right ventricle, leads to inefficient pumping of blood. The septum of the heart usually becomes enlarged, which then reduces the blood flow to the aorta. This compromises the heart’s ability to relax and accept more blood, which will then in turn limit the amount of blood available to be ejected on the next heartbeat; also known as a reduction in preload.

Restrictive cardiomyopathy is the least common of the cardiomyopathies. This form of cardiomyopathy occurs when the heart becomes stiff due to disease or scar tissue growth, and thus the heart is unable to accept the increase in blood (reduction in preload), and the blood backs up through the rest of the body (increase in afterload) [11].

Dilated cardiomyopathy is the most common of the three types of cardiomyopathy and can occur either two ways: as a result of CVD, or of idiopathic
(unknown) origin. Dilated cardiomyopathy occurs when the LV becomes enlarged and weak leading to a thinning of the LV wall. The thinning of the LV wall alters the heart’s pumping mechanics via change of the force/contraction relationship and also due to difference in end systolic/end diastolic relationships. When discussing dilated cardiomyopathy of an idiopathic origin, a reduction of energy stores available to the myocardium usually is the root cause of the pathology. As the body requires more resources and the heart is unable to supply the resources due to an increased end-systolic volume, a process of remodeling begins. The LV dilates to add volume in an attempt to supply more blood to the body, which in turn alters the force of contraction. This leads to a vicious cycle of myocardial remodeling, ultimately leading to HF.

Ischemic heart disease is the most common cause of HF in industrialized societies [1]. Ischemic heart disease may lead to the development of HF as a direct insult to the myocardium, due to the inhibition of sufficient oxygenated blood flow to heart tissue. The lack of oxygen, in myocardial injury, often causes cell death which can lead to a loss of contractility. The longer the myocardium is without oxygen, the more cell damage will occur. The greater amount of myocardial damage, the greater is the loss of contractility. As a loss of contractility occurs, there is an increase in end-systolic volume which will ultimately lead to reduced diastolic filling and eventually leads to LV dilation [12]. The resulting adaptations to the heart initiates remodeling the heart chambers and therefore decreases the heart’s efficiency for pumping blood; this often leads to the inability of the heart to fulfil the metabolic demands of daily living.
1.3 Symptoms and classification of heart failure

Some of the hallmark symptoms associated with HF include exercise intolerance, early fatigue, shortness of breath, and occasionally peripheral edema [2, 5, 13, 14]. Recently, there has been some insight into the cause of some symptoms associated with HF. It appears that the cause of exercise intolerance and early fatigue, which occurs in people with HF, is a result of an issue with the periphery and not due totally to cardiovascular dysfunction [5, 15]. One contributing factor for exercise intolerance and early fatigue is due to the early onset of lactate acid in the muscles. For individuals with HF, lactate accumulates at a relatively low work rate, when compared to healthy individuals, which contributes to hyperventilation and early fatigue [16, 17]. Exercise training helps improve oxygen consumption, and delays the onset of the lactate accumulation in skeletal muscle in patients with stable HF [9]. These benefits then help the individual improve their exercise tolerance by delaying early fatigue and therefore increasing their functional capacity. The discovery of the importance of muscular and vascular abnormalities, especially at the level of the peripheral vessels and the muscles, has been a major breakthrough in the understanding of the physiology of the limitation of exercise response in chronic HF [18]. However, it is agreed that cardiovascular function, albeit not the main contributing factor, does contribute to some of the associated symptoms.

The ongoing debilitating symptoms such as, decreased exercise capacity, shortness of breath and early fatigue, that people experience as a result of HF often reduce the ability to perform activities of daily living (ADL) and, therefore negatively affect their independence and quality of life (QOL) [9]. This reduction in the ability to
perform ADL may lead to further health problems both psychological and physiologically. A systemic review has shown that exercise training has a significant impact on QOL [10, 18] and issues associated with depression [19]. As people become more tolerant of dyspnea and experience less fatigue, they become more comfortable participating in ADL [19].

The severity of disease can be graded depending on effects of physical exertion and the New York Heart Association (NYHA) has developed a grading scale to determine the severity of the disease based on physical limitations associated with HF (Table 1). The grading scale takes into account the degree of functional impairments and assigns a severity class based on the physical limitations. However, since the symptoms reported are mostly subjective in nature, the NYHA class may vary depending upon the symptoms experienced. Since symptoms may vary daily, it is possible to not only have an individual in a single NYHA class, but individuals may be grouped into a combination of classes that best describe their physical limitations [20]. Most of the evidence of the benefits of exercise training in patients with HF is from patients with stable HF in NYHA functional classes II or III (Table 1); exercise training is now generally accepted to be safe and encouraged in these patients [2, 9, 21].

1.4 Pathophysiology of heart failure

The reduction in cardiovascular function is closely related to a poor cardiac output for people with HF. Cardiac output (Q) is defined as the product of stroke volume (SV) and heart rate (HR) which represents the volume of blood ejected by the heart in one minute, measured in litres per minute.

\[ Q \ (L \cdot \text{min}^{-1}) = (SV \ [ml] \times HR \ [bpm]) \]
<table>
<thead>
<tr>
<th>NYHA Class</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No symptoms and no limitation in ordinary physical activity, e.g. shortness of breath when walking, climbing stairs etc.</td>
</tr>
<tr>
<td>II</td>
<td>Mild symptoms (mild shortness of breath and/or angina) and slight limitation during ordinary activity.</td>
</tr>
<tr>
<td>III</td>
<td>Marked limitation in activity due to symptoms, even during less-than-ordinary activity, e.g. walking short distances (20-100 m). Comfortable only at rest.</td>
</tr>
<tr>
<td>IV</td>
<td>Severe limitations. Experiences symptoms even while at rest. Mostly bedbound patients.</td>
</tr>
</tbody>
</table>

Adapted from The Criteria Committee of the New York Heart Association. *Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Great Vessels.* [1].
When a healthy person exercises they are able to increase their Q to fulfill the extra demand placed on the heart, whereas individuals with HF are not able to increase their Q in accordance with the extra load that activity places on the heart. The reason for this may be twofold. First of all, SV cannot increase during exercise due to the dilation and/or impairment in contractility of the LV. Since the LV is dilated at rest, the chamber is unable to accept significantly more blood as activity increases, therefore increasing the physical limitation. Also, due to the reduction of contractility in the LV, the portion of blood that is ejected with each beat is also greatly reduced. To analyze the ratio of blood ejected with each heartbeat, the ejection fraction (EF) of the LV is calculated. The ejection fraction is calculated by taking the SV (mL) and dividing it by the amount of blood left in the LV after the heartbeat (End Diastolic Volume EDV)

\[
EF(\%) = \frac{SV(ml)}{EDV(ml)}
\]  

(2)

A normal EF is reported to be ≥ 50%, whereas mild dysfunction is noted with an EF of 41-49%, moderate dysfunction 35-40% and severe dysfunction is reported when an EF is below 35% [19]. A lower EF indicates that there is a reduction in the amount of blood ejected during systole, leaving a greater amount of blood in the ventricle during diastole and therefore limiting the amount of blood that can enter the heart during the diastolic period; this is known as systolic dysfunction. This then limits the amount of blood that can be circulated when physical demands increase and therefore causing fatigue to settle in as the body cannot fulfill its energy needs. In healthy individuals, the LV is able to stretch as the requirements for oxygen rich blood increases. This will allow
the LV to accept more blood during diastole and in turn apply more force during systole; this is known as the Frank-Starling Mechanism [22]. In the case of HF, the LV is already stretched at rest, due to the re-modelling of the heart, so the question of usefulness of the Frank-Starling Mechanism has often come into question. Warburton et al. suggested that individuals with systolic dysfunction may (in part) be able to make use of the Frank-Starling Mechanism to aid SV during activity [4].

The second contributing factor for the reduction in Q is heart rate. In the Q equation, HR is the variable that is prone to the greatest change. A healthy individual may have a resting HR of 70 beats per min (BPM), and as exercise increases, the Q can potentially increase 2-3 fold as a result of the increasing HR. However, since SV does not change appreciably for individuals with HF when they exercise, the increase in Q reflects the ability to increase the HR with exercise [23]. For individuals with HF, HR is increased at rest and decreased at peak exercise leading to a reduction of the Heart Rate Reserve (HRR) [15].

\[
\text{HRR} = (\text{Max HR} - \text{Resting HR}) \times \text{target exercise intensity} + \text{Resting HR}
\]  

(3)

It has been observed that peak HR may be reduced by as much as 20% in people with HF when compared to matched healthy controls [4]. This reduction in maximal HR limits the HRR in people with HF and is a major contributing factor for reduced exercise tolerance [5]. The benefits of exercise training on HR response have been well documented over the years. Exercise training leads to a decrease in HR at rest which will ultimately have a direct impact on the HRR and HR recovery [18].
The heart goes through a dramatic remodeling process during the development of HF. The natural shape of the heart is elliptical and as the disease progresses continues, the LV begins to dilate to try to attenuate the loss of contractility resulting in an observable left ventricular hypertrophy (LVH). The resulting remodeling of the heart alters the elliptical shape to more of a spherical shape. This change in the shape of the heart may inhibit the ability of the heart to make use of the Frank-Starling Mechanism and as the LV becomes more dilated, the possibility of pericardial constraint comes into play [5]. Pericardial constraint is the physical limitation of the pericardium not allowing the LV to dilate any further. If the LV is at the upper end of dilation there may be no way for the LV to allow a greater EDV during activity.

The added volume of the LV is associated with neurohumoral changes as well. As the body attempts to reverse the effect of reduced Q and the resultant reduction of organ perfusion, the sympathetic and renin-angiotensin-aldosterone system is then activated (by release of catecholamines, renin, vasopressin, atrial natriuretics) in an attempt to increase myocardial contractility. The renin-angiotensin-aldosterone system is primarily responsible for maintenance of peripheral vascular resistance and blood pressure. As sympathetic tone increases, renin is released from the kidney into the bloodstream. The added increase of renin into the bloodstream has a direct impact on circulating angiotensin to produce an influx of angiotensin I. Angiotensin Converting Enzyme (ACE), located primarily in the lungs, converts circulating angiotensin I to angiotensin II which increases vasoconstriction [24]. However, persistent neurohumoral excitation actually results in a deterioration of myocardial function with inflammatory response, end-organ damage and muscle derangement, which leads to worsened exercise
capacity [5, 9]. Since the myocardium does not have the ability to replace dead cells, the remaining myocytes have to increase the work done causing an overload and therefore creating a hypertrophic response and promoting further cell death creating a vicious cycle of remodeling [25].

1.5 Exercise and its effects on heart failure

Exercise training was previously contraindicated for people with HF. Prior to the mid 1980’s, physical strain and exercise was not recommended due to the belief that the physical strain would cause further decline in cardiac function [5, 13, 17, 19, 21]. Within the past ten to twenty years, research has shown that exercise is safe, effective, and beneficial for people with HF and is recommended in most professional guidelines as a necessary intervention for patients with stable HF [5, 10, 16, 18, 26-28]. Exercise has been shown to produce no negative effects nor significant deterioration in central hemodynamics but in fact [2, 29], exercise training has shown to improve both central hemodynamics and peripheral muscle function leading to improvements in exercise capacity, functional status and overall QOL in people with HF [5]. The improvement in muscular function allows individuals to perform exercise at a greater sub-maximal level, therefore requiring a lower voluntary muscle contraction [30], and with the net return of decreased energy demands, an increase exercise efficiency for the same sub-maximal exercise level occurs.

When discussing the effects of exercise intolerance on individuals with HF, it is important to take note that the major limitation for not participating in ADL is not the fact the individual has a reduced peak aerobic activity, but rather limitations in sustained sub-maximal effort [30]. Since people with HF seldom work at peak exercise, an
improvement in sub-maximal aerobic capacity may potentially lead to a greater benefit in QOL [21].

Exercise is a valuable tool in managing the negative remodeling process associated with HF. Since participating in regular exercise has shown not to worsen LV function, it has been thought to confer a mild reverse remodeling effect of the LV [7]. The heart’s normal response to cardiovascular exercise is for the myocardium to increase in mass as a direct result of the added load required to perform required activities and therefore producing a non-pathological LV hypertrophy [31]. The LV hypertrophy that is gained as a result of exercise is physiologically quite different than LV hypertrophy that is produced as a result of a diseased state of heart. The non-pathological hypertrophy is associated with the cardiac myocytes gaining length opposed to the pathological state where mass is added by the myocytes gaining girth. The added length of the myocardium helps increase the Frank-Starling Mechanism and therefore increases the contractility of the LV. Also, the added hypertrophy also leads to an increase in end-diastolic volume and improvement in EF [9, 13]. This increase in EDV allows more blood to be accepted into the LV and allows more blood to be ejected during systole.

However, the benefit of exercise training on LV function in HF still remains controversial. Studies have shown that individuals with HF, who exercise, can produce an anti-remodeling effect to the heart [13]. A meta-analysis by Haykowski et al. showed that regular exercise does not worsen LV function and actually produces a mild remodeling effect on the LV leading to an increase of 2.6% in EF [10, 32]. Other randomized studies showed that there was no effect or a small beneficial effect on left ventricular remodeling, with a slight but significant increase in EF [18]; whereas Tabet et
al. states that short training programs (3-5 sessions per week at 4-8 weeks) to have no or limited effect on LV remodeling [26]. The consensus remains that exercise may benefit LV function, but to what extent still remains unknown.

1.5.1 Aerobic training in heart failure

Oxygen uptake (VO$_2$) is the key variable when discussing exercise intolerance and fatigue for people with HF. Where VO$_2$ is calculated based upon the Fick Equation [33].

$$\text{VO}_2 = Q \times a\text{VO}_2\text{diff}$$ (4)

Typically, in a clinical setting, EF is the main prognostic factor when discussing disease severity. However, Selig et al. suggest that VO$_2$ is a more of a prognostic factor than EF [14]. Typically people with HF have a VO$_2$ that is less than 50% of age matched norms [5, 6], and is not uncommon to have a VO$_2$ of 12-15 mL·kg$^{-1}$·min$^{-1}$ range. A VO$_2$ of <10 mL·kg$^{-1}$·min$^{-1}$ identifies individuals that fall into a high risk category and individuals with a VO$_2$ >18 mL·kg$^{-1}$·min$^{-1}$ identifies low risk individuals and moderate risk falls between the two ranges [2]. These low oxygen consumption rates produce an interesting situation for individuals with HF. The minimum threshold for independent living for men and women is reported to be 18 and 15 mL·kg$^{-1}$·min$^{-1}$ respectively [5], leaving the possibility for individuals with HF to lead a lifestyle dependent on others. Several investigators have reported that people who achieve a VO$_2$ >14 mL·kg$^{-1}$·min$^{-1}$ appear to have a similar prognosis similar to those individuals who receive heart transplants (mortality rate of 10% at 1 year) [29]. Also, studies have shown that an
increase in VO₂ of 2 mL·kg⁻¹·min⁻¹, to a level greater than 12 mL·kg⁻¹·min⁻¹, within a 6±5 months of exercise training, leads to a 2 year survival rate of 100% [29]. The expected increase in VO₂ varies dramatically among studies performed. A typical range of improvement after exercise training is found to be in the range of 1.0-3.5 mL·kg⁻¹·min⁻¹ [13]. Although the improvement range may be at a relatively low level, keep in mind the very low functional capacity that individuals with HF start with. Other studies have reported an improvement in oxygen consumption as a percent improvement in VO₂ rather than relative ranges. Ranges vary quite substantially depending upon the starting oxygen consumption of the participants in the study and type of training methods. For example, results from the CHANGE (Chronic Heart failure ANd Graded Exercise) study lead to an overall increase of 10% improvement in VO₂ using a steady state training methodology [21]. The positional statement on Exercise Training in HF from Australia, states that an expected improvement in peak VO₂ should be in the order of 15% [14], but no training methodology was mentioned. Exercise has also been shown, in some small randomized and clinical trials, to improve exercise capacity by 15-25% in class II or III NYHA [2]. However, Crimi et al. [9] indicate that aerobic interval training to be superior to steady-state endurance training by increasing VO₂ 46% vs. 14% respectively [9, 14]. Simply put, exercise will lead to a relative increase of 10-30% in VO₂ depending upon intensity, duration [30] and NYHA class. The context of improvement must be fully understood to verify previous study results.

Although exercise is recommended for people with HF, the optimal dose and frequency remain to be determined [6, 9]. Previously, steady state exercise was the modality of choice, but recently, research has determined that superior gains in oxygen
consumption are derived from higher intensity aerobic interval training [9]. The cardiovascular effect of aerobic interval training (up to 95% peak heart rate) was found to be superior to that of moderate continuous training (70% peak heart rate) in elderly patients, who exercised three times per week for 12 weeks [9]. This produced a greater improvement in EF than a continuous or a steady-state program [9, 18] and led to a reverse remodeling of the LV [9, 13], improved aerobic capacity, endothelial function, and QOL [18]. These results were confirmed through the Canadian Cardiovascular Society and they recommend performing short bouts of high-intensity interval training (15-30 seconds on and off) since ADL for individuals with HF are often performed in short bouts of activity opposed to activity of longer duration [34].

Regardless of frequency, dose, and intensity, duration does matter in the development of an aerobic program for people with HF. Just recently a novel study by Höllriegel et al. investigated the long-term effects of exercise training on exercise capacity and LV remodeling. They observed that oxygen uptake and anti-remodeling properties improved most dramatically from baseline to the 3 month mark, continued to improve from the 3 to 6 month mark, albeit at a slower rate, and remained consistent from the 6 month to 12 month mark [35].

Regardless of the intensity and modality used for exercise training, occasionally individuals are not able to improve their functional capacity. This lack of significant improvement, with short term exercise training, leads to a poor prognosis for people with HF. These people who did not respond to exercise were found to have a lower EF and a lower HRR [26].
There are many suggested exercise prescriptions for individuals with HF. Steven J. Keteyian, a well-respected author and clinical exercise physiologist, has promoted the development of exercise routines for HF patients. His team suggests that an exercise prescription needs to be individualized, occur 3-5 sessions/week at 30-60 min·session\(^{-1}\) with an intensity of 50-60% HRR, and progressively increase to 70-80% of HRR [10].

1.5.2 Resistance training in heart failure

Views have differed regarding the effectiveness of resistance training as a treatment modality for HF. A growing body of literature suggests that resistance training prevents the decline in skeletal muscle mass, and function, that is associated with aging [9, 34], and has been found to cause no adverse changes in LV function and central hemodynamics [14, 28]. In people with HF, several cellular changes occur within the skeletal muscle that is associated with exercise intolerance. These include a shift towards type II fibres (fast twitch) [9, 18, 23, 31] and a reduction in the percentage of type I fibres (slow twitch) [5, 18] leading to an increase in muscle fatigability [9, 23, 31]. Regular and vigorous resistance training results in a shift from fatigue-prone type II fibres to fatigue-resistant type I fibres in patients with CVD who suffer from skeletal muscle myopathy [9]. However, recently literature has indicated that for older adults resistance training should be done in an explosive and quick manner to preserve the type II muscle fibres. The preservation of type II muscle fibres and the development of power has been found to be more relevant for many ADLs than strength measures alone [36]. Since the loss of functionality is often related to the loss of type II muscle fibres in older adults, the mode of resistance training to optimally benefit people with HF has yet to be determined.
Tabet et al. recommend that one set of 8-10 different exercises, that train major muscle groups, be performed 2-3 days per week for 10-15 repetitions at a low relative resistance (less than 50% of 1 repetition maximum (RM)) [18]. Tabet et al. may have been conservative in their recommendations of always performing resistance training at less than 50% of 1 RM in people with HF. Beckers et al. reports that a study performed by McKelvie et al. found that there was no evidence of significant deterioration of LV function during resistance exercises performed at intensities of 60-70% of 1 RM [30] and higher intensity levels of exercise training (70% of peak VO₂) seems to be necessary to obtain a significant re-shift to type I fibre, and significant decrease in type II fibres [18]. Although empirical evidence is mounting regarding the benefits of strength training in HF, there still appears to be some reservation in using in this methodology. De Maeyer et al. have indicated that “sustained maximal isometric exercise (e.g., weightlifting) is contraindicated in these patients, because of the excessive rise in blood pressure and the lowering of stroke volume” [33]. Finally, the Canadian Cardiovascular Society Heart Failure Management Guidelines of 2013, suggested an arbitrary use of light weights (5-10lbs), for 10-20 reps performed 2-3x/week [34]. Obviously taking a “one-size fits all” approach to strength training will not elicit the types of improvements that should be expected. There appear many different beliefs as to which training methodology will elicit the best results for strength training in individuals with HF.

Resistance training is usually paired with a combination of aerobic training during a complete workout. The benefits of endurance training on remodeling of the LV and improvements in functional capacity have previously been established, but this benefit has not been confirmed with combined resistance and endurance training [28, 30].
Recently there have been a number of studies performed investigating the effects of resistance training and a combination resistance and aerobic training on heart failure. Jewiss, Ostman and Smart conducted a meta-analysis of these new studies to understand their derived benefit. They found that there was no difference in VO$_2$ between a combined training methodology and aerobic only training. However, they found that when comparing combined training against a control group, and resistance training against a control group, that resistance training alone provided a superior training edge. They also found very little benefit amongst the two training modalities when investigating improvements in EF and resting systolic blood pressure measurements [37]. Contradictory evidence of LV remodeling was found by Beckers et al. who compared an aerobic training group and a combined training group and found that there was small comparable improvements in EF in both groups [30]. Although there is a growing pool of evidence for the benefits of resistance training in the HF population, some researchers are still resistant to promote the benefits of this training modality [38]. Unlike aerobic activity, resistance training requires more advanced instruction on proper technique and initial load development. This may be one reason as to why it is a less commonly prescribed method of activity for research studies. As further research is performed in the years to come, a definite picture as to the benefits resistance training will provide for the individuals participating in the training will become clear.

1.6 Medications and their effect on heart failure

Medications are used to manage HF by primarily helping deter and/or hinder the remodeling process of the myocardium, and to help manage the signs/symptoms associated with HF. The key treatment categories include ACE inhibitors, β-adrenergic
receptor blockers, diuretics, and possibly digoxin [2]. It is important to note that when discussing remodeling principles of the LV, advances in pharmaceutical medications have a positive effect on LV remodeling that may alter results during exercise training studies. To attenuate the possible effects medications have on the myocardium, usual requirements are to have stable medications prior to enrolment in research studies, have participants using the same pharmaceutical categories, and to make note of any medication change during the study. Pharmaceutical management of HF has become routine in the past years that treatment has now become standardized and most participants will be using a majority of the recommended pharmaceutical classes. Therefore, any benefits that are derived from the study can be contributed to the methodology used during the study opposed to benefits derived from pharmaceutical treatment.

Each recommended pharmaceutical class benefits the myocardium in different aspects. ACE inhibitors reduce myocardial remodeling by suppressing the renin-angiotensin-aldosterone system associated with HF and also [39] decrease afterload of the heart, therefore, increasing SV and consequently increasing Q [40]. β blockers also positively alter ventricular remodeling by reducing LVH, but also decrease Q by reducing sympathetic activity of the myocardium and ultimately HR. Heart rates can be reduced by as much 30bpm at rest and therefore reducing the HRR (see Initial Program Development in Procedures). Diuretics do not directly alter the remodeling of the myocardium but also reduce Q by reducing blood volume and arterial pressure [40].
1.7 Safety of exercise in heart failure

According to the American Heart Association’s Statement on Exercise and Acute Cardiovascular Events, cardiac rehabilitation centers reported 1 cardiac arrest for every 116,906 hours of exercise, 1 MI every 219,970 hours of exercise, 1 fatality every 752,365 hours of exercise and 1 major complication every 81,670 hours of exercise [41]. Although the potential for complications exists during activity, the incidence remains low due to the prior medical screening and the ability of professionals to handle emergencies as they arise. This compares to one non-fatal cardiac event every 1,124,200 hours of recreational exercise and one fatal event every 887,526 hours of recreational exercise for healthy people [41]. Since this was a very important area to look at, Smart and Marwick conducted a review of 30, single-center trials and reported there were no exercise-related deaths that occurred during more than 60,000 hours of exercise training among HF participants [13, 18]. Similarly, the HF-ACTION trial (which was the largest study exploring the benefits of HF and exercise) concluded that regular aerobic-type exercise is, indeed, safe in patients with chronic, stable HF [10].

CHAPTER 2: Assessing Cardiac Function

2.1 Methods used to assess cardiac function

There are a number of diagnostic tools that are used to assess the structure and function of the heart. These include: Echocardiography (ECHO), magnetic resonance imaging (MRI), to a lesser extent electrocardiography (ECG) and seismocardiography (SCG). While ECHO, MRI, ECG are well established and provide both structural and functional information about the heart, including, flow, volume, and pressure changes,
they can be very costly and time-consuming. SCG, a technology that was actively researched in the 1960’s (as cited in [42]), is regaining interest in the research and medical communities today [43, 44].

2.2 History of seismocardiography (SCG)

Seismocardiography is a non-invasive technique used to analyze the mechanical function of the heart and strength of the myocardial contraction [45] by recording vibrations that the heart produces. This principle has been observed by having an individual stand on a spring operated scale and observe that the body, and the display of the scale move rhythmically with each beat of the heart [46]. J.W. Gordon first discovered this in 1877, when he first recorded the ballistic forces that the human heart creates by having participants lay on a horizontal wooden frame that hung from ropes and was attached to mechanical recorder to trace vibrations caused by the beating heart [47]. In 1938, Dr. Isaac Starr used the same principles that J.W. Gordon discovered and had individuals lay on a rigid platform attached to heavy springs to analyze cardiac vibrations. He then advanced the technique further by classifying the waveforms and coining the term ballistocardiogram (BCG) [47, 48]. The technology failed to catch on with practitioners due to the heavy and cumbersome equipment that was not user friendly. With advances in technology, there once again has been interest expressed in ballistocardiography. In the 1960’s Baevski was inspired by seismologists studying vibrations underground and started measuring chest wall vibrations on Russians in their aerospace program during space flight [42]. In the late 1980’s, a team composed of an earthquake seismologist and a cardiologist, teamed up to advance the technology once again and introduce chest wall vibrations into the field of medicine. John Zanetti and
David Salerno applied the advances in accelerometer technology to record forces that heart produces and used the term SCG to describe their methods [49]. The modern advances differ from the old technology by using sensitive accelerometers and advanced microprocessors to identify cardiac vibrations along with the use of the ECG to help with the timing of the cardiac cycle [50].

The early studies using BCG were aimed at measuring cardiac movements and their correlation with the Q, while studies using SCG were aimed at recording and measuring the whole spectrum of cardiac vibrations. Therefore, BCG will refer to the low-frequency component of the SCG reflecting recoil movements, while SCG will indicate whole spectrum of cardiac vibrations [49].

The SCG is based upon Newton’s third law of motion that states “for every action there is an equal and opposite reaction” [46]. As the heart goes through its normal cardiac cycle, the vibrations caused by components of the heart can be recorded by the recording devices. These movements reflect the recoil forces that result from contraction of the heart muscle, valve closures and cardiac systole [49]. Since the forces generated by the heart are transmitted through the body tissues and skeleton before reaching the recording system, they may be subject to distortion and modification. This leaves the possibility that the larger the person is, the greater the possibility of distortion exists [46].

2.3 The seismocardiogram

There have been other terms used to describe the study of cardiac vibrations. They include apex-cardiography, kineto-cardiography, cardio-kymocardiography, BCG and SCG; with the latter two being the most common [49, 51, 52]. The early clinicians using ballistocardiography used a lettering system to describe the wave formations that
occur as a result of cardiac vibrations. The lettering system described by Starr and colleagues [53] used a system starting from the letter H and ending with the letter M. The lettering system named each successive wave whether positive or negative and assigned the corresponding letter. The work done by Salerno et al. [52, 54] took the principles developed by Starr et al. [55] and used ECHO to validate the points previously described. ECHO showed that a majority of the points on the SCG corresponded to specific mechanical events of the cardiac cycle (Table 2) (Figure 3). However, others could not validate certain aspects on the SCG that Salerno claimed, but the errors were later attributed to the differences in ECHO technology from the late 1980’s to present [56]. Just as important as mechanical timing of the cardiac cycle is the amplitude of the SCG waves. Direct relationships exist between myocardial contractility, the peak acceleration of blood from the LV, and the amplitude of the early systolic waves of the SCG [48]. In a strong heart the forces are explosive, where the largest deflections come early in systole. The forces arising from acceleration of blood at the onset of systole greatly exceeds those of deceleration. In weak hearts the reverse may be true. When the heart is weak, there is no initial jerk and the wave rises more slowly to a later peak [55]. Amplitude of the SCG also comes into play as the positioning of the body changes. As the body moves from a horizontal to a more vertical position, the effect of gravity places a greater demand on the circulatory system by influencing the hydrostatic pressure in the veins of the body. This change of pressure emits larger amplitude waves on the SCG [45].
<table>
<thead>
<tr>
<th>SCG point</th>
<th>Mechanical Descriptions</th>
<th>Annotation Placement</th>
</tr>
</thead>
<tbody>
<tr>
<td>MC</td>
<td>Mitral Valve Closure</td>
<td>Beginning of the sharp down slope following QRS onset</td>
</tr>
<tr>
<td>IM</td>
<td>Isovolumetric Movement</td>
<td>Bottom of down slope following MC</td>
</tr>
<tr>
<td>AO</td>
<td>Aortic valve opening</td>
<td>Peak of the next up sloping segment following IM</td>
</tr>
<tr>
<td>IC</td>
<td>Isotonic Contraction</td>
<td>Bottom of down slope following AO</td>
</tr>
<tr>
<td>RE</td>
<td>Rapid Ejection</td>
<td>Peak of rounded positive wave following IC</td>
</tr>
<tr>
<td>AC</td>
<td>Aortic Valve Closure</td>
<td>Sharp negative slope change near the end of the T wave on ECG</td>
</tr>
<tr>
<td>MO</td>
<td>Mitral Valve opening</td>
<td>Bottom of 2nd down slope following AC</td>
</tr>
<tr>
<td>RF</td>
<td>Peak of Diastolic filling</td>
<td>2nd rounded peak following MO</td>
</tr>
<tr>
<td>AS</td>
<td>Atrial Systole</td>
<td>2nd positive peak occurring after P wave on ECG. Identified by the mechanical contraction of the atria</td>
</tr>
</tbody>
</table>

Note. SCG = Seismocardiogram, BCG = Ballistocardiogram

Modified from Crow et al. [50]
Figure 3. Sample seismocardiogram in relation to the ECG waveform [3].
The SCG can then be used to determine matrices of myocardial performance. When investigating myocardial performance, the Systolic Timing Intervals (STI) are often studied. The two measures of systolic timing that are often measured are the: the pre-ejection period (PEP) and the left ventricular ejection time (LVET). The PEP is the time from onset of ventricular depolarization (onset of the QRS of the ECG) to the aortic valve opening. The LVET is the period from aortic valve opening (end of the PEP) to closure of the aortic valve [57], and total systole is the combination of the PEP and the LVET. The PEP is looked at as the timing component of systole (the time required for the LV to develop pressure to close the mitral valve) and the LVET is the mechanical component [57].

2.4 Reproducibility of the seismocardiogram

Reproducibility of the SCG is essential for determining accurate waveforms of the cardiac cycle. As with the main principles of the SCG measuring the vibrations of the myocardium, mechanical properties of breathing may also come into play. McKay et al. tested the reproducibility of the SCG by randomly analyzing tracings at different respiratory cycles at the same phase of respiration. They concluded that different respiratory cycles did not produce significantly different waves [58]. Also, when McKay. et al. analyzed samples of SCG tracings, point by point, from randomly selected samples, they also concluded that the SCG was sufficiently reproducible with a R value of 0.94 [58].

2.5 Disease process and the seismocardiogram

During the presence of cardiovascular disease, there are remarkable changes that occur to the SCG. Since ischemic CVD tends to damage the myocardium by way of
myocardial cell death and scar tissue formation, a change in cardiac mechanics can be expected. As discussed earlier, cell death in the myocardium changes the myocardial function which will cause a remodeling of the LV and alter the cardiac mechanics. These changes can be observed on the SCG. To first understand what is abnormal, the normal SCG mechanical timing must be defined. Zanetti and Salerno used a group of 10 healthy subjects to determine mean systolic time intervals for the cardiac cycle. They averaged all heart beats to a reference point of 60 b·min\(^{-1}\) and found the average total systole to be 411±15ms, the PEP to be 105±3ms and the left ventricular ejection time LVET to be 304±13ms [54].

In the presence of disease a few things happen to the SCG. As discussed earlier, the weaker heart produces smaller amplitude to the SCG wave and also alters the mechanics of the myocardium. Since ECHO is often considered the gold standard for measuring cardiac mechanics, a great deal of time has been invested into determining corresponding points to the SCG. Studies have shown that whether ECHO is used, or the SCG is used, subjects with cardiomyopathy consistently had longer cardiac intervals when compared to normal [50]. Furthermore, Bombardini et al. showed, using ECHO, that people with HF are categorized by a prolongation of LV systole, and an abnormal shortening of diastole [59]. Crow et al. also analyzed the systolic components of the cardiac cycle and determined that the presence of disease had more of an influence specifically during the aortic valve opening (AO) and rapid ejection (RE) points on the SCG when compared to ECHO points [50]. Crow et al. hypothesized that the occurrence of this is due to the dys-synchrony between the mechanical movement of the myocardium and the actual flow associated with contraction. With the accumulation of information of
how the disease process affects certain aspects of the mechanical properties of the myocardium, it is likely that the SCG will be able to determine these points pre- and post-exercise and determine if there is a change in mechanical function in the myocardium.

CHAPTER 3: General Purpose

Research has well established that people with HF have altered cardiac mechanics due to the negative remodeling that occurs as a result of a MI or cardiomyopathy. These altered mechanics ultimately produce a reduction in the pumping efficiency of the beating heart. Exercise has proven to be useful for all populations in the improvement of cardiac mechanics and functional capacity. However, a consensus as to the degree to which exercise will improve cardiac mechanics and functional capacity in people with HF remains yet to be seen. The general purpose of this thesis is to determine whether the SCG can be used to detect the altered cardiac mechanics in people with HF, to determine if a combined aerobic and resistance training program will produce significant benefits in the timing aspects of the cardiac cycle in people with HF and to determine whether a combined aerobic and resistance training program will improve exercise intolerance when measured using the 6MWT.

3.1 Hypothesis

H$_1$: It is hypothesized that the SCG will be able to differentiate changes in cardiac mechanics, specifically left ventricular ejection time and the pre-ejection period, after exercise training in HF patients.
H2: It is hypothesized that walking distance, measured using the six-minute walk test, will improve as a result of the combined exercise program and therefore, exercise intolerance will improve.

CHAPTER 4: Methods

4.1 Participants

Ethical approval was obtained from the University of Regina’s Ethics Review Board (REB#109R1112) and written and informed consent was gathered from each participant prior to enrollment in the study (Appendix A, B, C).

Participants were purposefully selected from a physician referred client base attending the Dr. Paul Schwann Applied Health and Research Centre (DPSC) Cardiac Rehabilitation Program at the University of Regina. All participants were deemed medically able to participate in the exercise program as determined by the participant’s family practitioner or cardiologist. Participants for the research study were required to have a period of medical stability for a minimum period of three months to meet criteria for entrance in the research study. Medical stability will be defined as no medication change or adverse events/hospitalizations due to nature or course of the disease [60]. Participants were also required to be assessed as either NYHA class II or III with etiology of ischemic heart disease/dilated cardiomyopathy. A sample size of 28 participants (14 control and 14 interventional) between 50 and 75 years of age were sought to fulfill the requirements of the study.

Participants were excluded from participation in the study if there was a medication change within three months of entering the exercise program, and were
removed as a participant from the study if any medication changes occurred during the study. Participants were excluded from the study if any complications or adverse events occurred within three months of entering the study or at any point during the study. Other factors that excluded participants from the study included chronic obstructive pulmonary disease, documented exercise-induced ischemia or exercise-induced ventricular tachycardia, NYHA class IV symptoms, uncontrolled hypertension and orthopedic, peripheral vascular or neurological disease limiting the ability to exercise [21].

4.2 Equipment

The Seismocardiograph was used to record the mechanical function of the heart. This is comprised of a tri-axial accelerometer and a lead II ECG to record both mechanical and electrical activity of the heart. Additional equipment used was, sphygmomanometer and stethoscope for blood pressure readings, stopwatch and a 60m ovalar walking track for the six-Minute Walk Test (6MWT) and portable pulse oximeter for precise heart rate measurements and recording of oxygen saturation. For the purpose of the exercise program, a variety of exercise equipment was available for the participants in the study. The available exercise equipment consisted of Monarch stationary cycles, Lifecycle recumbent cycles, Monarch arm ergometers, Biodex and/or NuStep semi-recumbent elliptical trainers, a Keiser weight circuit consisting of 10 exercises and a 200m walking track.

4.3 Dependent Variable Measurements

Seismocardiograph. Seismocardiograph measurements were collected for a period of 60 seconds under quiet resting conditions with participants lying supine with
arms resting to their sides. The sensor of the seismocardiograph was placed on the sternum 1 cm above the xyphoid process [44]; excess body hair was removed (if required) prior to attachment of the electrode to ensure no interference with signal acquisition.

Analysis of wave forms and amplitudes were performed off-line, after the collection period, using designed software to annotate the waveforms. Two systolic timing indices were calculated: the LVET (aortic valve open (AVO) to aortic valve close (AVC)) and the PEP (onset of Q wave of ECG to AVO) (Figure 4 and 5). The AVO point was determined to be the largest vertical point on the Z axis past the “R” wave of the ECG [42, 61, 62] and the AVC point was determined to the largest first vertical point on the Z axis past the “T” wave of the ECG [61-63]. Both variables were indexed (reference equations determined by Weissler et al. (equations 5-8) as noted in [64]), as to remove the variability of HR and the potential effect it may have on the systolic timing index.

\[
\text{LVETI (ms) (male)} = 1.7 \times \text{HR (bpm)} + \text{LVET (ms)} \quad (5)
\]
\[
\text{LVETI (ms) (female)} = 1.6 \times \text{HR (bpm)} + \text{LVET (ms)} \quad (6)
\]
\[
\text{PEPI (ms) (male)} = 0.4 \times \text{HR (bpm)} + \text{PEP (ms)} \quad (7)
\]
\[
\text{PEP (ms) (female)} = 0.4 \times \text{HR (bpm)} + \text{PEP (ms)} \quad (8)
\]

**Pre-and Post-Exercise Blood Pressure.** Pre-exercise blood pressure was recorded using sphygmomanometer and stethoscope by a Registered Nurse, after sitting quietly for one minute. Post-exercise blood pressure was recorded promptly after
Figure 4. Normal tri-axial SCG tracing including the ECG waveform
Figure 5. Sample tri-axial SCG tracing of an individual with HF
completion of the 6MWT and recorded to the nearest 2mmHg. The nurse that initially recorded resting blood pressure also recorded the post-exercise blood pressure to ensure inter-tester reliability.

**Pre-and Post-Exercise Heart Rate.** Pre-exercise HRs were recorded during the rest period prior to taking pre-exercise blood pressure. A portable pulse oximeter (Nellcor N-20, Minneapolis, MN) was used to ensure accuracy of the pre-exercise HR. Upon completion of the 6MWT, post-exercise heart rates were recorded immediately using the same portable pulse oximeter and recorded to the nearest one b·min⁻¹.

**Oxygen Saturation** was recorded prior to and upon completion of the 6MWT via portable pulse oximetry (Nellcor N-20, Minneapolis, MN).

**Oxygen Uptake** was calculated using a regression formula, based upon results from 6MWT, developed by Cahalin et al. (Equation 7) [65]. The regression formula was developed using patients with symptomatic HF with dilated and ischemic cardiomyopathies. Results are recorded to the nearest 0.1 mL·kg⁻¹·min⁻¹.

\[
VO2\text{peak} \ (\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) = 0.02 \cdot distance(\text{m}) - age(\text{yrs}) - 0.07 \cdot weight(\text{kg}) + 0.03 \cdot height(\text{cm}) + 0.26 \cdot RPP \cdot (10 - 3) + 2.45
\] (9)

**Anthropometric Variables** were assessed at the start and at the 12 week point of the exercise program. Variables that were included for data analysis were: height, body mass, body mass index (BMI), waist girth and hip girth. Height was recorded to the nearest 0.1 cm using a stadiometer, weight was recorded to the nearest 0.1kg using a digital scale (Tanita WB-110A, Toyko, Japan), waist girth was measured at the level of
the navel after exhalation and hip girth was measured with feet together at the greatest protrusion of buttocks.

4.4 Procedures

**Recruitment of participants.** Participants were recruited on a volunteer basis from a pool of attendees at the DPSC Cardiac Rehabilitation Program. Each participant was approached by an employee of the DPSC cardiac rehabilitation team regarding the possibility of participating in the research study. An information sheet outlining the research study’s goals, and requirements, was given to each potential participant (Appendix B). If more information was required regarding the study, questions were then directed to the principal investigator. All potential participants were approached regarding potential participation in the study during the initial program development or during their first or second follow-up session of their exercise program. The subjects were required to have a specific medical history to enroll in the study (i.e., absence of chronic obstructive pulmonary disorder, musculoskeletal limitations, NYHA class IV symptoms, history of complicated heart rhythms, and uncontrolled hypertension). Once consent was obtained to enter the study, a SCG tracing was recorded at the earliest convenience.

**Initial Program Development.** Each participant, when entering the Cardiac Rehabilitation Program at the DPSC, goes through an extensive interview process which involves a comprehensive medical history. If further information was required regarding their medical history, consent was obtained to gather further medical information and test results from the Regina Qu’Appelle Health Region. All participants attending the rehabilitation program were physician referred and deemed physically able to perform
exercise for the purpose of rehabilitation. Informed consent was obtained from all participants for attendance to the DPSC rehabilitation program (Appendix D).

Prior to the development of the exercise program, a routine 12-lead ECG was gathered from each participant. Exercise programs were then developed under medical supervision and were individually tailored for the individual’s current physical and functional state. Standard prescription for aerobic exercise intensity is difficult for individuals in this population because of the prescription of medications. Standard procedure for prescribing HR for aerobic activity involves using the HRR method which takes into account maximal heart rate, resting heart rate and intensity ranges. The recommended aerobic intensity for individuals with HF is a training range of 40%-70% of HRR (See Equation 3)[66].

Individuals who are prescribed β-blockers experience reduced resting and maximal HR therefore drastically altering their HRR and exercise prescription. For individuals who are taking β blockers, aerobic intensity was prescribed based on the 6-20 Borg scale of perceived exertion (Table 3) with the goal to achieve a Rating of Perceived Exertion (RPE) in the range of 11(“light”) – 13 (“somewhat hard”) [33].

Resistance training intensity was set for the individual to achieve failure after completion of the 10th repetition of the first set. Participants were instructed to gradually build on repetitions until reaching a plateau of 15 repetitions, while maintaining one set of repetitions. After the participant maintained consistency of 15 repetitions, a 10% increase was added to the resistance machines and the individual started over at 10 repetitions. Sample exercise prescription is included in Appendix E.
<table>
<thead>
<tr>
<th>Exertion</th>
<th>RPE</th>
</tr>
</thead>
<tbody>
<tr>
<td>No exertion at all</td>
<td>6</td>
</tr>
<tr>
<td>Extremely light</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>8</td>
</tr>
<tr>
<td>Very light</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>10</td>
</tr>
<tr>
<td>Light</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>12</td>
</tr>
<tr>
<td>Somewhat hard</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>14</td>
</tr>
<tr>
<td>Hard</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>16</td>
</tr>
<tr>
<td>Very hard</td>
<td>17</td>
</tr>
<tr>
<td></td>
<td>18</td>
</tr>
<tr>
<td>Extremely hard</td>
<td>19</td>
</tr>
<tr>
<td>Maximal exertion</td>
<td>20</td>
</tr>
</tbody>
</table>

**Exercise Sessions.** When the participants arrived for their next exercise session, they were asked to gather some baseline measurements and perform a 6MWT on a 60m oval walking track. Resting blood pressure was taken prior to the 6MWT by a Registered Nurse and a resting HR along with oxygen saturation was recorded by using a portable pulse oximeter to ensure the accuracy of the HR. Participants were then instructed to:

“**Walk as quickly as you can, following the 60m oval track for a period of six minutes. If you need a break during the six minutes, feel free to take one. If you experience any adverse symptoms during the test, stop immediately and inform the investigator and/or the nurse.**”

Upon completion of the 6MWT, post-exercise HR and oxygen saturation were recorded using the portable pulse oximeter and a post blood pressure was taken immediately (within 10-15sec) and recorded by the Registered Nurse. Distance was recorded to the nearest meter along with any symptoms experienced during the 6MWT. A RPE (Table 3) was also recorded. Initial weight was recorded to the nearest 0.1kg and height was recorded to the nearest 0.1cm.

Participants were then requested to attend their exercise program three times per week, for a period of 12 weeks. During the participant’s exercise sessions, HRs were measured via radial artery palpation and recorded in the participant’s exercise journal. The HR was then used to determine the physical load of each exercise and to determine if the participant was exercising within their prescribed exercise intensity range. Upon
completion of the 12 week exercise program, the participant underwent another SCG recording and a 6MWT (as described previously in **Procedures**). Body mass (kg) was also recorded at this point. Adjustments to the participant’s exercise program were performed at the 6 week point, following the progressive overload model, to facilitate further functional changes. Again, changes were made in accordance to the participant’s current functional state. All exercise program updates and 6MWT administration was performed by employees of the DPSC Cardiac Rehabilitation team. Seismocardiogram acquisition was performed by the principal investigator, with the recording being performed at the same time of day as the initial screening. Attendance rates were calculated by determining the number of sessions completed of the available sessions in a 12 week period.

Upon completion of each 6MWT, VO\textsubscript{2} was determined by using data obtained during the 6MWT and applying it to Equation 9.

All SCG readings were annotated off-line and indexed to ensure the systolic timing intervals were compared at a similar rate. Previous studies have shown that HR has a direct influence on cardiac timing intervals by either speeding up or slowing down certain aspects of timing [68, 69]. By normalizing HR this ensures intervals are compared on similar levels.

Anthropometric variables were analyzed on two separate occasions; at the beginning of the exercise program and upon completion of the 12 weeks of exercise programming.
4.5 Data analysis

**Statistical analysis.** Each beat of the 60 sec SCG was annotated and analyzed, means and standard deviations were calculated for each of the systolic timing intervals of the SCG. A paired samples t-test was used to determine improvements in 6MWT distances, peak VO\(_2\), and timing intervals of the SCG, from the beginning to the end of the exercise training program. After determining significant results, a Cohen’s d post hoc analysis was performed to determine the effect size pre-and post-12 weeks of training for 6MWT distance, pre-and post-6MWT heart rates, pre-and post-systolic and diastolic blood pressure, LVET and the PEP A paired samples t-test was also used to compare Anthropometric Variables (Body Mass Index (BMI), waist girth and hip girth) that were recorded at the start of the exercise program and at the 12 week point. When looking at participant demographics amongst ischemic and non-ischemic groups, a one-way ANOVA was used to test for significance between groups. Statistical significance was set at \( p \) value of \( \leq 0.05 \). All data were analyzed using a statistical software package (SPSS 18.0; IBM, Chicago, IL)

**Data storage.** All data were stored under double lock and key or password encrypted computer. All data will be stored for the minimum period of seven years. All paper records will be disposed of accordingly and all data records electronically will be erased and deleted. Participants were assigned an individualized identification number and are only referred to as that number for the duration of the study.

**CHAPTER 5: Results**

Twenty individuals consented to participate in the study. Of those 20 individuals, one participant was removed from the study due to changes in medication
regime, 8 were removed for failing to comply with the exercise program, resulting in a participant pool of 11 (Male=9). Significance was measured at $p \leq 0.05$ and all results are reported as mean ± standard deviation (SD).

Table 4 summarizes the participant demographics upon entry to the research study. The outcomes of the study lead to some significant improvements when looking at the variables involving the systolic timing intervals (STI) and the 6MWT followed by some minor changes in anthropometric variables. For this study, two specific timing intervals of the cardiac cycle were assessed, the Pre Ejection Period (PEP) and the Left Ventricular Ejection Time (LVET). The LVET resulted in a 6.2ms decrease in ejection time (451.4±29.6ms to 445.2±26.8ms $t(329)=4.22$, $p=<0.001$, effect size 0.22), and the PEP decreased by 3.6ms (126.8±25.0ms to 123.2±25.7ms $t(329)=4.75$, $p=<0.001$ effect size 0.14) (Table 7).

The results of the 6MWT also showed significant results. The walking distance improved from 480.0±94.8 m to 538.4±92.6 m, $t(10)=-4.21$, $p=0.002$, for an overall improvement of 58.4 m or 12.2%, and when applying these results to predict $\text{VO}_2\text{peak}$ (Equation 3) the absolute improvement was 1.2 mL·kg$^{-1}$·min$^{-1}$, $t(9)=-2.61$, $p=0.028$ (Table 6).

When looking at the anthropometric variables over 12 weeks of the exercise program, there was no significant change to weight, BMI, waist girth and hip girth (Table 5). Additionally, when the anthropometric variables are broken down into the distinct etiologies for HF, there still remains no statistical significance amongst the variables from start to completion of the program (Table 9). Initially this may seem as a failure for the participants to not have any improvement in weight or girth measurements during
their 12 weeks of exercise training, but the volume and intensity of the program has to be taken into consideration when discussing this. These participants are not able to exercise at a volume or intensity high or long enough to warrant significant caloric expenditure; their focus was to gain functionality during the exercise program.

The participants were also divided into two distinct groups; ischemic heart failure (n=6) and non-ischemic heart failure (n=5). There were no significant differences between the groups when comparing the participant demographics (Table 8). The systolic timing intervals still produced significant results, however, only in the non-ischemic group, LVET 449.6±36.0 ms to 438.4±30.5 ms t(149)=4.28 p=≤0.001 with a small to medium effect size of 0.34 and PEP 128.0±23.5 ms to 119.9±18.5 ms t(149)=6.87 p=≤0.001 with a small to medium effect size of 0.38, and saw no discernible changes to the systolic timing intervals in the ischemic group (Table 11). Interestingly, when it came to the 6MWT the opposite occurred; the ischemic group produced significant improvement in walking distance, 477.0±127.0 m to 539.3±113.9 m t(5)=3.01 p=0.030 with a medium effect size of 0.51, whereas the non-ischemic group produced no significant changes in 6MWT distance (Table 10).
Table 1. Participant Demographics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>n</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male (%)</td>
<td>9</td>
<td>81.8</td>
</tr>
<tr>
<td>Female (%)</td>
<td>2</td>
<td>18.2</td>
</tr>
<tr>
<td>Age (years)</td>
<td>11</td>
<td>54.5±6.8</td>
</tr>
<tr>
<td>Diagnosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemic (%)</td>
<td>6</td>
<td>54.5</td>
</tr>
<tr>
<td>Non-Ischemic (%)</td>
<td>5</td>
<td>45.4</td>
</tr>
<tr>
<td>Time from diagnosis to program entrance (months)</td>
<td>11</td>
<td>16.8±21.1</td>
</tr>
<tr>
<td>Sessions used (%)</td>
<td>11</td>
<td>80.1±10.6</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>9</td>
<td>26.6±7.5</td>
</tr>
<tr>
<td>Co-morbidities</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pacemaker (%)</td>
<td>5</td>
<td>45.5</td>
</tr>
<tr>
<td>Valve replacement (%)</td>
<td>3</td>
<td>27.3</td>
</tr>
<tr>
<td>NIDDM (%)</td>
<td>4</td>
<td>36.4</td>
</tr>
<tr>
<td>Medications</td>
<td></td>
<td></td>
</tr>
<tr>
<td>β blocker (%)</td>
<td>10</td>
<td>90.0</td>
</tr>
<tr>
<td>ACE inhibitors (%)</td>
<td>9</td>
<td>81.8</td>
</tr>
<tr>
<td>Diuretic (%)</td>
<td>9</td>
<td>81.8</td>
</tr>
<tr>
<td>Digoxin (%)</td>
<td>3</td>
<td>27.3</td>
</tr>
<tr>
<td>Warfarin (%)</td>
<td>7</td>
<td>63.6</td>
</tr>
<tr>
<td>ASA (%)</td>
<td>7</td>
<td>63.6</td>
</tr>
</tbody>
</table>

Note: NIDDM = Non-Insulin Dependent Diabetes Mellitus (Type 2); ACE = Angiotensin Converting Enzyme; ASA = Acetylsalicylic Acid.
Table 2. Anthropometric variables pre and post 12 weeks of exercise training. (n=10). Values are mean ± SD.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Initial</th>
<th>12 Weeks</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI (kg/m²)</td>
<td>29.30±6.1</td>
<td>29.80±6.1</td>
<td>t(9)= -1.87, p=0.095</td>
</tr>
<tr>
<td>Body Mass (kg)</td>
<td>86.1±22.2</td>
<td>87.5±22.2</td>
<td>t(9)= -1.93, p=0.086</td>
</tr>
<tr>
<td>Waist Girth (cm)</td>
<td>105.00±18.3</td>
<td>104.4±17.9</td>
<td>t(9)=0.32, p=0.755</td>
</tr>
<tr>
<td>Hip Girth (cm)</td>
<td>104.2±13.1</td>
<td>103.7±12.1</td>
<td>t(9)=0.31, p=0.766</td>
</tr>
</tbody>
</table>

Note: BMI = Body Mass Index
**Table 3. Results of the 6-Minute Walk Test pre and post 12 weeks of exercise training. (n=11). Values are mean ± SD.**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Initial</th>
<th>12 Weeks</th>
<th>Sig.</th>
<th>Effect Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre walk test HR (bpm)</td>
<td>68.6±9.6</td>
<td>68.1±12.5</td>
<td>t(10)=0.13, p=0.897</td>
<td>-</td>
</tr>
<tr>
<td>Post walk test HR (bpm)</td>
<td>86.2±8.6</td>
<td>92.6±14.9</td>
<td>t(10)=2.13, p=0.060</td>
<td>-</td>
</tr>
<tr>
<td>Pre walk test SBP (mmHg)</td>
<td>109.4±13.2</td>
<td>111.1±15.1</td>
<td>t(10)=0.39, p=0.704</td>
<td>-</td>
</tr>
<tr>
<td>Pre walk test DBP (mmHg)</td>
<td>67.8±9.7</td>
<td>69.0±8.6</td>
<td>t(10)=0.41, p=0.692</td>
<td>-</td>
</tr>
<tr>
<td>Post walk test SBP (mmHg)</td>
<td>122.7±21.9</td>
<td>127.6±20.4</td>
<td>t(10)=0.54, p=0.602</td>
<td>-</td>
</tr>
<tr>
<td>Post walk test DBP (mmHg)</td>
<td>68.9±11.5</td>
<td>74.1±10.0</td>
<td>t(10)=2.40, p=0.037</td>
<td>0.48</td>
</tr>
<tr>
<td>Pre walk test SaO2 (%)</td>
<td>96.5±1.5</td>
<td>96.9±1.6</td>
<td>t(10)=0.81, p=0.437</td>
<td>-</td>
</tr>
<tr>
<td>Post walk test SaO2 (%)</td>
<td>95.6±1.9</td>
<td>95.9±3.00</td>
<td>t(10)=0.30, p=0.774</td>
<td>-</td>
</tr>
<tr>
<td>Walk test RPE</td>
<td>12.2±1.5</td>
<td>13.0±1.1</td>
<td>t(9)=1.63, p=0.137</td>
<td>-</td>
</tr>
<tr>
<td>Walk test distance (m)</td>
<td>480.0±94.8</td>
<td>538.4±92.6</td>
<td>t(10)=4.21, p=0.002</td>
<td>0.62</td>
</tr>
<tr>
<td>Predicted VO2 (mL·kg⁻¹·min⁻¹)</td>
<td>13.5±3.4</td>
<td>14.7±3.2</td>
<td>t(9)=2.61, p=0.028</td>
<td>0.36</td>
</tr>
</tbody>
</table>

*Note: HR = Heart Rate, SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, RPE = Rating of Perceived Exertion, SaO2 = Oxygen Saturation*
Table 4. The Systolic Timing Intervals pre and post 12 weeks of exercise training. Values are mean ± SD. (n=11)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Initial</th>
<th>12 Weeks</th>
<th>Sig.</th>
<th>Effect Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (bpm)</td>
<td>66.6±10.7</td>
<td>66.9±10.0</td>
<td>t(329)= -0.63, p=0.530</td>
<td>-</td>
</tr>
<tr>
<td>LVET index (ms)</td>
<td>451.4±29.6</td>
<td>445.2±26.8</td>
<td>t(329)= 4.22, p=&lt;0.001</td>
<td>0.22</td>
</tr>
<tr>
<td>PEP index (ms)</td>
<td>126.8±25.0</td>
<td>123.2±25.7</td>
<td>t(329)= 4.75, p=&lt;0.001</td>
<td>0.14</td>
</tr>
<tr>
<td>PEP index/LVET index</td>
<td>0.28±0.06</td>
<td>0.28±0.07</td>
<td>t(329)= 1.75, p=0.082</td>
<td>-</td>
</tr>
</tbody>
</table>

Note: HR = Heart Rate, LVET = Left Ventricular Ejection Time, PEP = Pre-Ejection Period
### Table 5. Participant Demographics for the two distinct aetiologies of heart failure.

*Values are mean ± SD.*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Ischemic (n=6)</th>
<th>Non-Ischemic (n=5)</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male (%)</td>
<td>83.3</td>
<td>80</td>
<td>F(1,9) = 0.017, p = 0.900</td>
</tr>
<tr>
<td>Female (%)</td>
<td>16.7</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>55.7±6.3</td>
<td>53.0±7.9</td>
<td>F(1,9) = 0.392, p = 0.570</td>
</tr>
<tr>
<td>Time from diagnosis to program entrance (months)</td>
<td>27.0±24.6</td>
<td>4.6±3.4</td>
<td>F(1,9) = 4.010, p = 0.076</td>
</tr>
<tr>
<td>Sessions used (%)</td>
<td>83.3±10.3</td>
<td>76.2±10.8</td>
<td>F(1,9) = 0.017, p = 0.294</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>26.2±9.5</td>
<td>27.0±5.4</td>
<td>F(1,9) = 0.022, p = 0.886</td>
</tr>
<tr>
<td>Co-morbidities</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pacemaker (%)</td>
<td>66.7</td>
<td>20</td>
<td>F(1,7) = 2.506, p = 0.148</td>
</tr>
<tr>
<td>Valve replacement (%)</td>
<td>16.7</td>
<td>40</td>
<td>F(1,9) = 0.657, p = 0.438</td>
</tr>
<tr>
<td>NIDDM (%)</td>
<td>33.3</td>
<td>40</td>
<td>F(1,9) = 0.043, p = 0.840</td>
</tr>
<tr>
<td>Medications</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>β blocker (%)</td>
<td>83.3</td>
<td>100</td>
<td>F(1,9) = 0.818, p = 0.389</td>
</tr>
<tr>
<td>ACE inhibitors (%)</td>
<td>83.3</td>
<td>40</td>
<td>F(1,9) = 0.017, p = 0.900</td>
</tr>
<tr>
<td>Diuretic (%)</td>
<td>100</td>
<td>60</td>
<td>F(1,9) = 3.273, p = 0.104</td>
</tr>
<tr>
<td>Digoxin (%)</td>
<td>33.3</td>
<td>20</td>
<td>F(1,9) = 0.205, p = 0.662</td>
</tr>
<tr>
<td>Warfarin (%)</td>
<td>50</td>
<td>80</td>
<td>F(1,9) = 0.960, p = 0.353</td>
</tr>
<tr>
<td>ASA (%)</td>
<td>83.3</td>
<td>40</td>
<td>F(1,9) = 2.267, p = 0.166</td>
</tr>
</tbody>
</table>

*Note: NIDDM = Non-Insulin Dependent Diabetes Mellitus (Type 2); ACE = Angiotensin Converting Enzyme; ASA = Acetylsalicylic Acid.*
Table 6. *Anthropometric variables for the 2 aetiologies of heart failure. Values are mean ± SD.*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Ischemic (n=6)</th>
<th>Non-Ischemic (n=4)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial</td>
<td>12 Weeks</td>
</tr>
<tr>
<td><strong>BMI (kg/m^2)</strong></td>
<td>28.5±4.1</td>
<td>28.6±4.24</td>
</tr>
<tr>
<td><strong>Weight (kg)</strong></td>
<td>84.1±13.4</td>
<td>84.5±13.3</td>
</tr>
<tr>
<td><strong>Waist Girth (cm)</strong></td>
<td>102.6±14.7</td>
<td>101.3±12.3</td>
</tr>
<tr>
<td><strong>Hip Girth (cm)</strong></td>
<td>102.3±9.0</td>
<td>102.7±10.3</td>
</tr>
</tbody>
</table>

*Note: BMI = Body Mass Index*
Table 7. Results of the 6-Minute Walk Test pre and post 12 weeks of exercise training for the two aetiologies of heart failure.
Values are mean ± SD.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Initial Ischemic (n=6)</th>
<th>12 Weeks Ischemic</th>
<th>Sig.</th>
<th>Effect Size</th>
<th>Initial Non-Ischemic (n=5)</th>
<th>12 Weeks Non-Ischemic</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre walk test HR (bpm)</td>
<td>65.2±7.2</td>
<td>62.3±8.0</td>
<td>t(5)=1.29 p=0.254</td>
<td>-</td>
<td>72.8±11.3</td>
<td>75.0±14.1</td>
<td>t(4)=0.24 p=0.820</td>
</tr>
<tr>
<td>Post walk test HR (bpm)</td>
<td>82.2±6.6</td>
<td>89.2±10.8</td>
<td>t(5)=-2.98 p=0.031</td>
<td>0.78</td>
<td>91.0±8.7</td>
<td>96.8±19.2</td>
<td>t(4)=0.89 p=0.423</td>
</tr>
<tr>
<td>Pre walk test SBP (mmHg)</td>
<td>106.7±15.9</td>
<td>105.8±8.9</td>
<td>t(5)=0.25 p=0.815</td>
<td>-</td>
<td>112.6±9.9</td>
<td>117.4±19.5</td>
<td>t(4)=0.52 p=0.633</td>
</tr>
<tr>
<td>Pre walk test DBP (mmHg)</td>
<td>65.5±10.4</td>
<td>65.3±9.6</td>
<td>t(5)=0.04 p=0.966</td>
<td>-</td>
<td>70.6±9.0</td>
<td>73.4±5.0</td>
<td>t(4)=0.57 p=0.597</td>
</tr>
<tr>
<td>Post walk test SBP (mmHg)</td>
<td>112.0±12.9</td>
<td>129.0±22.3</td>
<td>t(5)=-2.78 p=0.039</td>
<td>0.93</td>
<td>135.6±24.8</td>
<td>125.8±20.3</td>
<td>t(4)=0.58 p=0.593</td>
</tr>
<tr>
<td>Post walk test DBP (mmHg)</td>
<td>65.5±14.6</td>
<td>74.5±12.5</td>
<td>t(5)=-5.73 p=0.002</td>
<td>0.66</td>
<td>73.0±5.0</td>
<td>73.6±7.4</td>
<td>t(4)=0.17 p=0.872</td>
</tr>
<tr>
<td>Pre walk test SaO₂ (%)</td>
<td>96.7±1.0</td>
<td>96.3±1.4</td>
<td>t(5)=0.79 p=0.465</td>
<td>-</td>
<td>96.2±2.1</td>
<td>97.6±1.7</td>
<td>t(4)=1.36 p=0.245</td>
</tr>
<tr>
<td>Post walk test SaO₂ (%)</td>
<td>96.0±2.0</td>
<td>96.2±3.1</td>
<td>t(5)=-0.11 p=0.920</td>
<td>-</td>
<td>95.2±1.9</td>
<td>95.6±3.2</td>
<td>t(4)=-0.41 p=0.704</td>
</tr>
<tr>
<td>Walk test RPE</td>
<td>12.6±1.3</td>
<td>13.2±1.3</td>
<td>t(4)=-0.80 p=0.468</td>
<td>-</td>
<td>11.8±1.6</td>
<td>12.8±0.8</td>
<td>t(4)=-1.41 p=0.230</td>
</tr>
<tr>
<td>Walk test distance (m)</td>
<td>477.0±127.0</td>
<td>539.3±113.9</td>
<td>t(5)=-3.01 p=0.030</td>
<td>0.51</td>
<td>483.6±48.1</td>
<td>537.2±72.2</td>
<td>t(4)=-2.66 p=0.056</td>
</tr>
</tbody>
</table>

Note: HR = Heart Rate, SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, RPE = Rating of Perceived Exertion, SaO₂ = Oxygen Saturation
Table 8. *The Systolic Timing Intervals pre and post 12 weeks of exercise training for the two aetiologies of heart failure.*

*Values are mean ± SD.*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Ischemic (n=6)</th>
<th>Non-Ischemic (n=5)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial 12 Weeks Sig. Effect</td>
<td>Initial 12 Weeks Sig. Effect</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>61.2±3.1 62.5±5.8 t(179)=-2.99 p=0.003 0.28</td>
<td>73.0±12.8 72.1±11.4 t(149)=0.86 p=0.390 -</td>
</tr>
<tr>
<td>LVET index (ms)</td>
<td>452.9±23.1 450.9±21.9 t(179)=1.32 p=0.190 -</td>
<td>449.6±36.0 438.4±30.5 t(149)=4.28 p=&lt;0.001 0.34</td>
</tr>
<tr>
<td>PEP index (ms)</td>
<td>125.8±26.3 126.0±30.1 t(179)=0.24 p=0.813 -</td>
<td>128.0±23.5 119.9±18.5 t(149)=6.87 p=&lt;0.001 0.38</td>
</tr>
<tr>
<td>PEP index/LVET index</td>
<td>0.28±0.06 0.28±0.08 t(179)=1.11 p=0.268 -</td>
<td>0.29±0.06 0.28±0.05 t(149)=3.61 p=&lt;0.001 0.18</td>
</tr>
</tbody>
</table>

*Note: HR = Heart Rate, LVET = Left Ventricular Ejection Time, PEP = Pre-Ejection Period*
CHAPTER 6: Discussion

The purpose of this thesis was to determine whether the SCG can be used to detect altered cardiac mechanics in people with HF, to determine if a combined aerobic and resistance training program will produce significant benefits in the timing aspects of the cardiac cycle in people with HF, and to determine whether a combined aerobic and resistance training program will improve exercise intolerance when assessed using the 6MWT. The most significant finding in this study was the positive adaptations that occurred with exercise to the mechanical timing of the heart, especially the pre-ejection period (PEP). It is well known that as the left ventricle (LV) becomes more diseased there is a degradation of the aspects of timing that occurs during the cardiac cycle; the PEP elongates and the LVET shortens [42, 70-73]. A reversal of those factors would indicate a positive change to the deteriorating cardiac mechanics. Previous studies have focused their research on using SCG as a tool of investigation and diagnosis [46, 48, 51, 52, 73, 74], but not as adaptation of actual exercise training over a prolonged period of time. Physiologists have established proof that exercise is linked to increased myocardial performance and improvements in mechanics of the beating heart, including, improvements in preload, cardiac output [5, 9, 31], and development of non-pathological left ventricular hypertrophy [31, 72, 75, 76].

The results of this study indicate that exercise is capable of improving indices of cardiac timing and positive changes related to functional adaptations in individuals with HF. The PEP has the most influence on the systolic timing intervals and cardiac cycle due to the direct relationship it has with ventricular preload [76]. An increase in ventricular preload will allow the LV to accept more blood during diastole, create a
greater stretch to the LV and therefore increase SV. Since preload varies inversely with
the PEP, any decrease in the PEP would indicate an increase in SV by way of enhanced
ventricular filling [73] and indicate an increase in myocardial contractility [42]. Since
individuals with HF have issues with both contractility and SV, the PEP provides
valuable insight to the workings of the LV. As discussed earlier, exercise is known to
produce changes to both preload and contractility. When PEP was examined after 12
weeks of exercise, there was a significant reduction in the PEP, and thus in agreement as
hypothesized. The PEP decreased significantly from 126.8±25.0 ms to 123.2±25.7 ms
(Table 7). This suggests that contractility was improved after the 12 week exercise
program in this study.

When looking at the LVET results there appears to be a paradoxical effect; i.e.,
the LVET shortened after the 12 weeks of the cardiac rehabilitation exercise program.
According to previously published studies, a shortening of the LVET is associated with a
reduction in ventricular function [42, 70-73]. These results differs from the normative
results reported by Weissler et al. for the expected time frame of the LVET for
individuals with HF. The established mean for LVET that Weissler et al., reported in
their 1968 study, cited an average LVET of 413±10ms [70], whereas, this study reported
a LVET of 451.4±29.6ms (Table 7). However, the etiology of patients reported in
Weissler et al. study are different from those reported in the current study, and could
account for this difference. One of the entry criteria for participation in this study was to
have a current dilated cardiomyopathy with a reduced EF, where Weissler et al.’s study
had no criteria for EF or LV wall dimensions. Also, there were three distinct forms of
HF that they based their study from: arteriosclerotic heart disease, hypertensive
cardiovascular disease, and primary myocardial disease; there were no definitions given for these etiologies.

It is known that an increase in afterload, that is prevalent in LV dilation, will produce significantly longer LVET times [77]. Another possible explanation for differences in studies is the effect of negative inotropic drugs. Negative inotropic drugs are primarily used to treat high blood pressure, decrease the electrical activity of the heart and are primarily used to treat HF; they are also known to increase LVET [78]. During the study by Weissler et al., all medications were withheld 48 prior to participation and specific medication classes were not listed as part of the study. It is plausible that with medical advances and the ever increasing reliance on medications to treat chronic conditions that some of the differences noted were due to a greater prevalence in afterload and optimal pharmaceutical care.

So why did the LVET shorten after 12 week of exercise training programming? The participants in our study had no indication of any worsening symptoms associated with their disease; there was no change in medication, there was no change in HR, systolic or diastolic blood pressure, and yet they experienced an increase in 6MWT distance. This resultant decrease in the LVET after exercise may be attributed to an increase rate of myocardial fiber shortening/contractility [5, 9, 76]. The increased rate of myocardial fiber shortening can directly be attributed to the aforementioned increase in preload. As more blood enters the left ventricle a greater stretch of that left ventricle occurs, a greater interaction of actin and myosin cross bridges occur, and more tension is developed and stored in the ventricle (Frank-Starling Mechanism). This increased tension produces a greater velocity of shortening and therefore a quicker LVET. As
discussed earlier, individuals with HF were thought not to be able to use the full extent of the Frank-Starling Mechanism. It is conceivable that the results from this study may indicate that our individuals with HF were able to make use of the Frank-Starling Mechanism. As mentioned previously, afterload impacts how the LVET behaves. Since afterload has a direct impact on the LVET, any reduction in afterload will produce a decrease in LVET. However, more research is warranted to examine the influence of exercise training on LVET in a similar group of HF patients.

The 6MWT is one of the most commonly used assessment metric in cardiac rehabilitation settings to assess functional capacity. As greater physical limitations occur, and the HF disease process advances, there is a greater decrease in oxygen consumption, ultimately leading a reduction in QOL [5, 9]. Any tools that can be used to demonstrate an increase in oxygen consumption is vital to the individual to maintain and/or improve their ADLs. As mentioned previously, a peak VO₂ of 14 mL·kg⁻¹·min⁻¹ will give the same prognosis as individuals who may have benefitted from heart transplantation, and any improvement of 2 mL·kg⁻¹·min⁻¹, to a level greater than 12 mL·kg⁻¹·min⁻¹, will lead to a 2-year survival rate of 100% [29]. When comparing the results of this study, (i.e., achieved 14.7 mL·kg⁻¹·min⁻¹ upon completion of 12 weeks of exercise), to the aforementioned standards, the improved VO₂ outcome would have indicated a positive influence on the participant’s functional capacity and physical well-being. Although the participants in this study failed to achieve the documented 2 mL·kg⁻¹·min⁻¹ standard to achieve the benefits associated with that functional capacity, these participants achieved an improvement of 1.2 mL·kg⁻¹·min⁻¹. The values that we are reporting could be viewed as misleading, as they are based on a sub-maximal, self-paced walking test which is then
used to predict VO$_{2\text{peak}}$. The reported values in previous studies [21, 30] are based upon a maximal symptom limited exercise tests. The possibility exists that if we had the capability of performing maximal exercise tests as part of this study; results would mirror those of the previous studies (see **Limitations** below).

Duration of exercise training programming is an important factor that should be addressed when comparing study outcomes. The participants in this study exercised three times per week for a period of 12 weeks which falls in-line with a majority of other studies. One of the largest trial investigating the safety of exercise in HF had individuals exercise 3 times per week for a period of 12 weeks; they saw an overall improvement in VO$_2$ of 10% or 1.4 ml·kg$^{-1}$·min$^{-1}$ [21]. In another study by Friemark et al., they had 42 subjects exercise twice per week for 18 weeks which resulted in an improvement of 1 ml·kg$^{-1}$·min$^{-1}$ upon completion of their study[6]. Finally, in a study that most closely resembled our format, Beckers et al., had participants exercise, using a combined resistance and endurance training, 3 days per week for a period of 6 months. At the end of their study, the combined training methodology produce an improvement of 2.1 ml·kg$^{-1}$·min$^{-1}$ [30]. A couple of smaller trials also produced similar results. Kiilavuori et al., had participants exercise three times per week for three months using a home based program. That study produced an improvement in VO$_2$ of 2.4 ml·kg$^{-1}$·min$^{-1}$. However, these participants started off with a VO$_2$ considerably higher (19.3 ml·kg$^{-1}$·min$^{-1}$) than participants in other studies [16]. Finally, Tabet et al. had individuals exercise “3 to 5 training sessions for 4 to 8 weeks” in a cardiac rehabilitation centre. Their trial produced an improvement of 7.1% or 1.2 ml·kg$^{-1}$·min$^{-1}$ [26]. It becomes clear that the process of beginning and undertaking an exercise training program will produce benefits to an
individual’s aerobic capacity and overall functioning. This study is no different. We had our participants exercise three times per week for a period of 12 weeks following a combined resistance and aerobic program and produced an improvement of 9.4% or 1.2 mL·kg\(^{-1}\)·min\(^{-1}\), which falls in-line with the studies previously mentioned.

The minimal clinical important difference (MCID) is another way to assess improvements in functional capacity by looking at changes in the 6MWT as absolute improvements opposed to relative improvements in distance walked. The MCID is a term that is used to determine the smallest change that can occur, from an intervention, that would produce clinically significant changes to the individual’s physical or mental well-being [79, 80]. The MCID has been studied quite often in individuals with HF due to the degree of impairment these individuals have as a result of their disease. A systemic review by Shoemaker et al. looked at the 6MWT and health related QOL and determined that a change of 45m in the 6MWT would produce the aforementioned MCID [81]. When applying the results of our study to the standards set out by Shoemaker et al., it appears that the improvement in 6MWT distance achieved the MCID set forth by Shoemaker et al. Our study yielded an improvement of 58m after completion of the 12 weeks of exercise programming. This would suggest that significant changes occurred to the individual’s physical well-being.

Another area of discussion is the impact of gender on outcomes of rehabilitation in HF. This study looked at two distinct outcomes; myocardial performance measured using the systolic timing intervals (PEP and LVET) and 6MWT distance. The patient demographics for this study leaned heavily towards being male dominant with males comprising 9 of the 11 participants (81.8%). The largest randomized trial investigating
the outcomes of exercise and HF was the HF-ACTION trial. This study randomized 2,331 patients into control and exercise arms, with the exercise arm receiving 1,159 patients. The overall gender breakdown for this study also present with a male dominant enrollment of 1,670 participants or 71%. They concluded that 6MWT distance was initially 7-10% lower in females when compared to males and females had a significantly lower baseline peakVO$_2$. However, upon completion of the study, no significant interaction occurred between gender and peakVO$_2$ after 3 months of exercise [82].

When looking at the impact of gender on the systolic timing intervals, Weissler et.al., concluded that small but significant changes occurred during the LVET and there was no change in the PEP (equations 5-8) [70]; this study adhered to the proper gender based indexing equations as proposed by Weissler et.al. Although gender was weighted heavily in the male favor for this study, it would appear that the effect of gender had little to no impact on the outcomes of this study.

The results from this study also compared the two etiologies of HF; the ischemic and the non-ischemic groups. We observed a non-significant decrease in LVET (452.9±23.1 ms to 450.9±21.9 ms), and no change in PEP (125.8±26.3 ms to 126.0±30.1 ms) for the ischemic group. Whereas, the non-ischemic group showed a statistically significant change in both LVET (449.6±36.0 ms to 438.4±30.5 ms, t(149)=4.28 p=≤0.001 with a small to medium effect size of 0.34, and PEP (128.0±23.5 ms to 119.9±18.5 ms, t(149)=6.87 p≤0.001 and a small to medium effect size of 0.38). When looking at the results of the 6MWT, both groups had similar improvements. The ischemic group had a significant improvement of 62m (t(5)=−3.01 p=0.030) and the non-ischemic group improved by 54m. Although the non-ischemic group failed to reach
statistical significance, the resultant 54m increase in 6MWT distance still achieved the MCID set forth by Shoemaker et al. [80, 81].

A closer inspection of the two different groups, ischemic and non-ischemic, revealed a number of important findings: 1) the ischemic group had a lack of mechanical changes to the heart after 12 weeks of exercise training, although they had significant improvements in 6MWT distance; 2) the non-ischemic group had significant improvements to the mechanical properties of the heart, but resulted in non-significant changes in the 6MWT. In part, the lack of significance in these variables could be related to the low participant numbers in each group (n=6 and n=5, respectively), as well it appears from the 6MWT the non-ischemic group was more homogeneous. Another possibility for the disparity between the two groups is that the myocardial damage, which occurred as result of ischemic nature of HF, produced enough cell damage that the myocardium was unable to receive the benefits of exercise training to alter the disordered pumping mechanics. Whereas, a “healthier” heart (non-ischemic group), that has no myocardial damage, was able to improve their pumping efficiency due to better functioning myocardium. When it comes to the difference in 6MWT distance between the two groups, the increase in walking distance for the ischemic groups could be related to an improvement in oxygenation and oxygen extraction to the periphery.

There has been a long standing debate as to whether the central or peripheral mechanisms are the driving force that leads to exercise and quality of life improvements [29, 83]. Recent research has suggested that peripheral muscular adaptations in HF patients occur with exercise training [5, 18, 19]. Thus it is possible that the ischemic
group had greater improvements in their peripheral muscular efficiency than in cardiac related changes.

One method to determine oxygenation of the periphery is to measure the hemoglobin of working muscles. Hemoglobin is contained within the red blood cells and is primarily responsible to carry oxygen to those working muscles [84]. In an unpublished pilot study, our laboratory also assessed the degree of oxygenation to the periphery while performing the aforementioned 6MWT. We assess oxygenation of the quadriceps at two time points (initially and at the 6-week point) and amongst the two etiologies of heart failure (ischemic and non-ischemic) by looking at the total hemoglobin available (tHb) in the quadriceps. We observed a statistically significant difference for tHb at the initial and 6-week points amongst the etiology of heart failure groups and more importantly observed a lesser drop in tHb from initial to 6-week assessment point for the ischemic group (non–ischemic: -4.0 vs. -3.3, ischemic -7.9 vs. -1.5). This potentially confirms the hypothesis that the improvement in functionality for individuals with ischemic based HF is as a result of improved oxygenation to the periphery opposed to improved cardiac mechanics.

CHAPTER 7: Limitations

This study had a number of limitations. The primary limitation was the small sample size. Although statistically underpowered, research studies focusing on HF often have sample sizes in the range of 10-30 participants [16, 50, 69, 76, 85, 86]. This may be in part to the relatively low prevalence of HF in the community. Although the completion percentage of the exercise program was 55% (11 of 20 participants), this is
similar to other reports in Cardiac Rehabilitation programs. The Mayo Clinic in Rochester, Minnesota, reported an improvement in completion rates from 14% to 39% following a Motivational Program intervention [87], and the Cardiac Wellness Institute of Calgary reported that from 1996 to 2009, of the 5,886 attendees of Cardiac Rehabilitation, only 49.3% completed the exercise program [88]. Finally, the largest randomized control trial investigating the safety and efficacy of exercise training in HF (HF-ACTION) reported that only 31.5% of participants completed the allotment of 36 exercise sessions [33]. Often cited reasons for non-completion of Cardiac Rehabilitation are: time of exercise program, difficulty/lack of difficulty, returned to work, access to exercise location, difficulty in changing lifestyle factors and the stigma associated with Cardiac Rehabilitation [89]. Unfortunately, reasons for non-completion were not gathered as part of this study. However, to mitigate the sampling difficulties, if all participants would be of the same pathologic background, unlike ours, that could strengthen that sample of participants.

Another potential limitation was related to the annotation and interpretation of the SCG signal to calculate the systolic timing intervals (LVET and PEP). An optimal situation would be to verify the findings from the systolic timing intervals with that of a standard procedure such as ECHO to confirm the reported findings. A number of limited studies have correlated the relationship between ECHO timing and the systolic timing intervals examined in this study [50, 51, 54, 56, 68]. More research in this area is also warranted to confirm these variables in heart failure patients. The ability of the SCG to detect the mitral valve opening/closing would also provide an enhanced ability to look at a greater number of timing intervals such as, the iso-volumetric relaxation and contraction
time. This ability could potentially give greater insight into the altered myocardial mechanics.

Also, the use of symptom limited stress tests to assess exercise performance before and after the training program would be an asset in conjunction with using the predictive 6MWT. Although the 6MWT is a reproducible and affordable test used for assessing cardiopulmonary exercise symptoms and developing cardiac rehabilitation programs, there is a degree of error associated with them. Carvalho et al. tested a cycle based cardiopulmonary exercise test against the 6MWT and determined, although significantly correlated, that the 6MWT overestimated PeakVO\textsubscript{2} by 6.37 mL/kg/min (20.49±1.99 vs. 14.12±4.11) [90]. There are more inherent limitations with using the 6MWT such as motivational factors [91] and physiological limitations such as walking speed and gait [92].

Additionally, physical activity outside the study design was not controlled. This allows the possibility for some participants to be more physically active than other participants and ultimately impacting outcomes in either walking distance or the STI. For example, the ischemic HF group may have been so debilitated prior to enrolment in the study and since beginning exercise they may have gained more self-confidence and began undertaking more physical activity at home and impacting their walking distance at the end of their 12 week exercise program.

Furthermore, a potential selection bias by purposefully selecting individuals may have impacted study outcomes. This limits the randomization since we are looking for an “ideal” participant for this study. By selecting participants only from the DPSC Cardiac
Rehabilitation Program, we have appealed already to the individuals who are prepared to exercise.

The time from diagnosis to program entry may have had some impact on the outcome of this study. The ischemic group had a time from diagnosis to program entry of 27.0±24.6 months, where the non-ischemic group had 4.6±3.4 months from diagnosis to program entry. This wide disparity between entrance months may be a reason why the ischemic group failed to show changes in the LVET and PEP. The individuals from the non-ischemic cohort may be more prone to myocardial adaptations since they were relatively new into their myocardial remodeling process whereas, the ischemic group had a greater time under disease and this may leave the myocardium less prone for adaptations.

Finally, the use of medications could potentially be an area of concern. As our participants maintained their prescribed medication regime throughout the study, the degree and duration of anti-remodeling properties that occurred as a result of the medication is unknown. The benefits of using β-blockers and ACE inhibitors in regard to myocardial remodeling may persist for months after initiation of dosing and therefore may have skewed the observed results.

**CHAPTER 8: Conclusion**

In conclusion, the SCG was able to detect changes that occurred to the myocardium as measured by the systolic timing intervals, LVET and PEP, after exercise training for individuals with HF. Secondly, walking distance significantly increased following the 12 week exercise training program to document an improvement in
functional capacity. Future research is needed to provide further insights into the mechanisms for improvements in exercise capacity. Research studies that include both central (cardiac) and peripheral (muscle) monitoring could also help to delineate the physiological mechanism(s) for the adaptations observed as a result of exercise training in heart failure patients. [93]
List of References


40. Klabunde, R.E., Cardiovascular Integration and Adaptation, in Cardiovascular Physiology Concepts 2004, Lippincott Williams and Wilkins.


List of Figure References


Figure 2. Number of Hospitalizations for CHF (actual and projected) in Canada 1980-20205. Available from: http://www.chfn.ca/facts-about-chf-in-canada.

Figure 3. Wilson, R.A., et al., Diagnostic accuracy of seismocardiography compared with electrocardiography for the anatomic and physiologic diagnosis of coronary artery disease during exercise testing. Am J Cardiol, 1993. 71(7): p. 536-545.
List of Table References

APPENDIX A – Ethics application

UNIVERSITY OF REGINA

RESEARCH ETHICS BOARD

Application for Approval of Research Procedures

Section I: Identification and Purposes

1. Date: March 29, 2012

Name of Applicant(s): Dr. J. Patrick Neary, Professor

Co-Applicants: Jonathan E. Silbernagel, BKin, CEP, MSc candidate

Student # (if applicable): 200206800

Full Address: Faculty of Kinesiology & Health Studies

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Telephone #: (306) 585-4361

E-mail: silbenj@uregina.ca

Title of Research: Effects of exercise training on heart failure measured using seismocardiography.
2. If the project will be part of a thesis, or class requirement, give the name of the supervisor:

   Supervisor: Dr. J. Patrick Neary, Professor
   Department or Faculty: Kinesiology & Health Studies

3. If you are a student, please indicate your level:

   □ Graduate
   □ Undergraduate

4. Provide an overview of the main features and variables of the research problem. Include a brief review of the relevant literature, a statement that describes the significance and potential benefits of the study, your hypotheses (if applicable), a brief description of your measures and some information about your design and analytic approach (e.g., "narrative data will be analyzed through grounded theory methodology"; "a 2 x 2 Multivariate Analysis of Variance will be employed for the data analysis").

   **Background:** This project is aimed at understanding the improvements in heart function between individuals with heart failure before and after an exercise training program. Heart failure is a growing health concern as the population of “baby boomer” ages and advances in health care lead to more people surviving myocardial infarctions that were previously unable to. Exercise training has now become a common treatment modality for many chronic disease conditions including heart failure. As the disease process in heart failure advances, the myocardium goes through a remodeling phase that alters
the myocardium and reduces the heart’s pumping efficiency. Exercise is known to lead to a positive remodeling of the myocardium in healthy populations, but the degree to which exercise reverses pathological remodeling in people with heart failure remains to be determined.

The SCG device is an accelerometer which records movement and measures the forces generated by the contraction and relaxation of the heart. This technology is considered more sophisticated than other tools for measuring heart function (i.e. electrocardiogram (ECG), cardiac and arterial blood pressures generated). As such, the SCG identifies cardiac problems at an earlier stage allowing preventative interventions, allows for monitoring the response to interventions (such as exercise, rehabilitation or in drug management), and provides more sophisticated and meaningful monitoring of patients in acute management situations. The value of the seismocardiogram in diagnosing cardiac anomalies was established more than fifty years ago (Starr 1965), but with the innovative application of modern computer technology and microchip sensing it will bring the digital seismocardiogram to clinical practice in Canada and worldwide. 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 on how the heart adapts to different types of exercise training.

Procedures: The testing will be performed within the constructs of the medically supervised Dr. Paul Schwann Centre (DPSC) Cardiac Rehabilitation program.
The individuals will be referred to the Cardiac Rehabilitation program by the patient’s general practitioner or cardiologist for the purpose of rehabilitation and will be deemed physically able to participate in the exercise training program by the referring physician. The participants will perform their individualized exercise training program three times a week for a period of eighteen weeks. Participants who consent to participate in the study will have a seismocardiogram (SCG) tracing done at the start of their exercise program, at the six week point, the twelve week point and the eighteen week point. To obtain the SCG tracing, participants will be placed in resting conditions, lying supine with arms resting to their sides. A standard 12-Lead ECG with also be performed at the same time, along with non-invasive blood pressure (Finometer) for 3 minutes. A DXA (dual energy x-ray absorptiometry) scan will also be performed at week 0 and 18. This procedure requires the participant to lay supine on a table while the body is scanned by an x-ray beam. This measurement takes approximately 15 minutes and is used to assess the participant’s body composition. Physiological variables that are routinely measured at DPSC Cardiac Rehabilitation program that are to be included in this study include functional capacity measured via six minute walk test (6MWT) at the start of the exercise program, six week point, twelve week point, and eighteen week point, bi-weekly blood pressures, bi-weekly 2-lead ECG, height, weight, waist girth, hip girth and grip strength measurements. In addition to these measurements, muscle oxygenation will be assessed during the routine 6MWT by using the non-invasive PortaMon NIRS device. The sensor
will be placed on the thigh (quadriceps) muscles and will be attached using an elastic bandage. The participants will be required to perform their 6MWT with the sensor attached to their leg.

**Exercise group with heart failure (n=20)** – Participants for the research study will be required to have a period of medical stability for a minimum period of three months to meet criteria for entrance in the research study. Medical stability will be defined as no medication change or adverse events/hospitalizations due to nature or course of the disease. Participants will also be required to be either assessed as New York Heart Association (NYHA) Class II or III with aetiology of ischemic heart disease. Participants will be excluded from participation from the study if there has been a medication change within three months of attending the exercise program or will be removed as a participant from the study if a medication change occurs during the study. Participants will also be excluded from the study if any complications or adverse events occur within three months of the study or during the study. Other factors to exclude participants include chronic obstructive pulmonary disease, documented exercise-induced ischemia or exercise-induced ventricular tachycardia, NYHA class IV symptoms, uncontrolled hypertension and orthopaedic, peripheral vascular or neurological disease limiting the ability to exercise (Wielenga 1999).
Safety of exercise training in heart failure: According to the American Heart Association’s statement on exercise and acute cardiovascular events (2007), surveys across exercise based cardiac rehabilitation centers reported 1 cardiac arrest for every 116,906 hours of exercise, 1 myocardial infarction every 219,970 hours of exercise, 1 fatality every 752,365 hours of exercise and 1 major complication every 81,670 hours of exercise (Thompson 2007). Although the potential for complications do exist, the incidence remains low because of prior medical screening and the ability of staff to handle emergencies as they arise. This compares to one non-fatal event every 1,124,200 hours of recreational exercise and one fatal event every 887,526 hours of recreational exercise for healthy people (Thompson 2007). Smart and Marwick (2004) also conducted a review of 30 single-center trials and reported no exercise-related death during more than 60,000 hours of exercise training among HF participants (Keteyian 2010a, Tabet 2009). Similarly, the HF-ACTION trial (which was the largest study exploring the benefits of HF and exercise) concluded that regular aerobic-type exercise is, indeed, safe in patients with chronic, stable HF (Keteyian 2010b).

All subjects will be monitored by trained and fully qualified professionals (See section 10)
References:


Section II: Application Checklist

1. Do you consider that this project involves:

☐ HIGH RISK to subjects

☒ MORE THAN MINIMUM RISK to subjects

☐ MINIMUM RISK to subjects
Researchers are advised that "Risk" is defined broadly to include not only threats to one's physical integrity or health but also temporary as well as permanent psychosocial consequences (e.g., experiencing negative mood for a brief period as a result of research participation, potential for violations of privacy, potential for upsetting a third party because of research participation).

If other than MINIMUM RISK, please explain and submit the full research proposal (e.g., grant application, thesis proposal) or, if a full proposal is not available, contact the REB Chair.

_There is a potential risk of bodily harm to occur by the participants during this research study. The risks exist by having participants exercise with heart failure. However, the risks are attenuated by having employees of the Dr. Paul Schwann Applied Health and Research Centre (DPSC) Cardiac Rehabilitation Program monitor the exercise sessions. The staff has experienced many emergency situations before as a routine part of their job. Also, all participants would be attending the rehabilitation program regardless of participation in this research study. All participants of the study and rehabilitation program are referred by their family physician or cardiologist and are deemed physically able to exercise. During the exercise sessions, all participants will be monitored by emergency trained staff, a registered nurse and a physician from the Regina Qu’Appelle Health Region. All participants have bi-weekly electrocardiograms and blood pressure readings recorded by the registered nurse and each_
participant self-records post exercise heart rate under supervision of DPSC staff. Consent will be obtained for participation in the exercise program and for recording of the SCG tracings.

2a. Do you think that the research findings from this project might result in a financial benefit to the researchers?

☐ Yes
☒ No

If Yes, please explain.

2b. Do you think that the research findings from this project might be commercially valuable to others (e.g. the researchers’ employers, the project sponsors)?

☒ Yes
☐ No

There is interest in making SCG devices commercially available in the future. As of now, the device and technology are in the testing phases with one company seeking USFDA approval and approval from Health Canada.

3. Would this research project or its findings place you or any member(s) of your research team in potential conflict of interest situation (e.g., being both a researcher and an employee of the organization being studied)?
Jonathan Silbernagel as an MSc Student (researcher) is also an employee of the DPSC Cardiac Rehabilitation Program. There may be “perceived” conflict due to the relationship that may be created between researcher and participant. However, the added personal relationship will not directly result in skewing results because personal relationships should not affect the outcomes of this study. Also, Jonathan will not be able to spend more time with the participants during their exercise sessions as the role he holds at the DPSC requires him to monitor 250-300 people per day performing their cardiac rehabilitation exercise program.

4. How long do you expect your research project (contact with human subjects for data collection) to last?
   ☒ Less than one year from the date of approval
   ☐ More than one year from the date of approval (an annual renewal will be needed every year)

5. What are the sources of funding (if any) for the proposed research?

   No sources of funding.
Section III: Subjects

1. Briefly describe the number and characteristics of participants required for the study, and how a potential sample of such participants will be identified.

_The focus of this research is to determine if exercise training produces any mechanical benefit to the myocardium in people with heart failure, and at what rate (if any) these adaptations occur. Participants will be selected from the DPSC Cardiac Rehabilitation program after referral by the family physician or cardiologist. All participants will be deemed medically able to participate in an exercise program as determined by the participant’s family practitioner or cardiologist. Participants for the research study will be required to have a period of medical stability for a minimum period of three months to meet criteria for entrance in the research study. Medical stability will be defined as no medication change or adverse events/hospitalizations due to nature or course of the disease. Participants will also be required to be either assessed as NYHA class II or III with aetiology of ischemic heart disease._

2. Describe the recruitment procedures. Who will approach potential participants and how (e.g. by phone, mail)?

_Subjects will be approached by other members of the DPSC cardiac rehabilitation team regarding the possibility of participating in the research study. An information sheet outlining the research study’s goals and_
requirements of the participants will be given to each potential participant. If more information is required regarding the study, questions will then be directed to Jonathan Silbernagel. All participants will be approached during the initial first or second session of the exercise program. The delay in recruitment will occur to ensure the client feels comfortable in the exercise setting and no symptoms were exacerbated due to the exercise program development. The subjects will be required to have a specific medical history (absence of chronic obstructive pulmonary disorder, musculoskeletal limitations, NYHA class IV symptoms, history of complicated heart rhythms, and uncontrolled hypertension) as to not confound to current physical state of the individual. A complete medical history is obtained via initial consultation with each participant upon entering the DPSC Cardiac Rehabilitation program. If further information is required regarding their medical history consent is obtained to gather test results from the Regina Qu’Appelle Health Region.

3. What will the participants be required to do in the course of the project?

The participants will be required to perform an exercise training program under supervision of the DPSC Cardiac Rehabilitation staff, registered nurse and physician. The exercise training program is individually tailored to the individual’s current functional state. The primary focus for the participant is to enter the cardiac rehabilitation program as per physician’s referral; the only difference is the acquisition of the SCG tracing, a blood pressure recording with
the Finometer (finger plethysmography), muscle oxygenation using the non-invasive PortaMon NIRS device, and Dual-emission X-ray absorptiometry (DXA). The SCG is placed over the sternum of the chest under resting conditions and will be recorded at the start of the exercise program, at the six week point, the twelve week point and eighteen week point. The SCG tracing is similar to that of an electrocardiogram tracing and places the individual in undo discomfort. The blood pressure recording is non-invasive and will be performed using a finger cuff (plethysmography) around the left index finger. The DXA is also a non-invasive tool used to assess body composition. The DXA testing will be performed at the start of the exercise program and at the 18 week point. The participants are required to lay supine for a period of 15 minutes as the body is scanned using an x-ray beam. The use of all equipment, the SCG, Finometer, DXA, and PortaMon devices has already received ethical clearance for other projects in our laboratory (#75R0910; #75R0708; #70R0910).

Participants will also be required to keep track of and record any exercise that occurs away from the DPSC Cardiac Rehabilitation program.

4. What information about the research project and their role will participants be given during the initial contact?

The subjects will be informed that the SCG device being used in this research project utilizes micro-accelerometer technology to measure the contraction forces of the heart. They will also be informed that testing occurs under resting
conditions and tracings will be recorded at four time intervals; start of exercise program, six week point, twelve week point and eighteen week point. This data will allow us to compare the mechanical functioning of the heart in people with HF pre and post exercise training, with hopes to use this information for rehabilitation and training. The SCG, Finometer, and PortaMon devices are non-invasive and possess no harm to the subject. The DXA scan does emit minute amounts of radiation. They will be informed that the DXA has been used previously in our research in Kinesiology and has received ethical approval (#).

5. Will a consent form be used? If so, when will it be presented (e.g. immediately before interviews take place)?

Two informed consent forms will be used to obtain permission from the involved subjects. The first form obtains consent from the participant to enter DPSC Cardiac Rehabilitation program acknowledging the potential risks associated with an exercise program and the second consent obtains permission to obtain the SCG tracing and blood pressure data. The consent for the DPSC Cardiac Rehabilitation program is presented at the initial intake time and the second consent will be obtained upon agreeing to participate in the research study (see attached).
6. What assurances will participants be given and what precautions will be taken regarding the confidentiality of the data or information which they provide in the study? (Please refer the following descriptions of anonymity & confidentiality)

**Anonymity**: No link can be established between a participant and the research (i.e. no one knows who has participated in the study).

**Confidentiality**: No link can be established between the collected information and a participant’s identity (i.e. no one can identify who contributed a given piece of information)

*Any information that is obtained during this study will be kept confidential to the full extent permitted by law. Subjects will be given a unique identification number and any information provided will be marked with this ID number. Materials will be locked in Jonathan Silbernagel’s desk and/or stored on a password-protected computer in Jonathan Silbernagel’s office and will only be available to him and, Dr. Patrick Neary (Research supervisor). The ID number master list will be kept separately from the data collected. Physiological data that is routinely collected through the DPSC Cardiac Rehabilitation Program as means of determining outcomes will be housed on a password encrypted database stored on University servers with access only granted to specific usernames.*
7. Will children be used as a source of data?

☐ Yes
☒ No

If Yes, indicate how consent will be obtained on their behalf.

8. Will the researcher or any member of the research team be in a position of power or authority in relation to the subjects? (For example: A teacher doing research and having a class as subjects or a counsellor collecting research data from clients).

☒ Yes
☐ No

If Yes, indicate how coercion of subjects will be avoided.

*Jonathan Silbernagel is directly involved in the daily operation of the DPSC Cardiac Rehabilitation program which may introduce a perceived influence on the clients in the DPSC. The influence may exist as part to Jonathan being responsible for the health of the participant as well as the exercise programs of the participants of the DPSC. It would not be ethical in any way to alter the exercise programs of the participant for the benefit of this study; the primary goal is rehabilitation, secondary goal is research. The DPSC Cardiac Rehabilitation program operates in a team environment and at all possible*
times other members of the rehabilitation team will perform the exercise
program updates and advancements and Jonathan Silbernagel will be
responsible for data collection of the physiological variables.

9. Describe any apparatus, substance, element of the physical environment or other
materials that could cause harm to a participant if a side effect, malfunction,
miuse accident or allergic reaction were to occur. If the participant comes into
contact with a potentially hazardous apparatus or material, who is responsible for
checking defects or malfunctions, and on what schedule will inspections be made?
If participants come into contact with some substance that could cause harm,
please document your safeguards.

The DPSC Cardiac Rehabilitation program operates in a gym setting involving
various types of exercise equipment. The exercise equipment is maintained and
service by IronMax Fitness.

10. If the research involves participants who have special vulnerabilities (e.g., victims
of sexual abuse, children, cardiac patients engaging in physical exercise), describe
any special competencies (e.g., professional qualifications, other specialty
training) that qualify you or any other member of your team to work with these
people.
All testing will be conducted under the direct supervision of licensed medical professionals. All testing of heart failure patients will be conducted in the Dr. Paul Schwann Centre Cardiac Rehabilitation Program, or the Exercise Physiology laboratory (SCG, Finometer, and DXA). The staff involved with the rehabilitation programs are qualified individuals trained in dealing with any type of cardiac emergency that may present itself throughout daily operations. The staff of the Dr. Paul Schwann Centre Cardiac Rehabilitation Program conforms to the highest standards of their professional organization. Exercise leaders hold Certified Personal Trainer designation from the Canadian Society of Exercise Physiology (CSEP), Consultants hold Certified Exercise Physiologist designation form the CSEP, there are also individuals who hold Exercise Specialist designation from the American College of Sports Medicine and individuals are also CPR instructors. Registered nurses are fully licensed and physicians primarily staff from the emergency department at the two Regina hospitals with Advanced Cardiac Life Support designations. A radiology technician will take all DXA scans.

The Dr. Paul Schwann Centre Cardiac Rehabilitation Program operates a fully stocked “crash cart” on site along with Automated External Defibrillators, portable 12-lead electrocardiogram units, multiple blood pressure units, blood glucose units and oxygen in case of emergencies. Individuals who hold designations from CSEP are required to re-certify their CPR skills on a yearly basis and the staff of the DPSC holds Health Care Provider CPR designations.
Emergency situations are handled as per protocol developed between the DPSC Cardiac Rehabilitation program, Campus Security, EMS and communication with the Regina General Hospital (see attached).

11. Will deception of any kind be necessary in the project?
   
   ☑ No

12. Describe any debriefing procedures that will be used. (Note that if deception is used, debriefing is necessary).

   Once the data is analyzed, the subject, upon request, will have full access to his or her individual test results. Any questions regarding the results will be answered by a member of the research team.

13. Will participants be compensated?
   
   ☑ No
Section IV: Access to Data and Findings

1. Who will have access to the original data? (For example co-investigators, students) How will all those who have access to the data be made aware of their responsibilities concerning privacy and confidentiality?

All members of the research team will have primary access to all data collected throughout the duration of this study. All data will be securely stored as described above and any access will follow the guidelines set forth by the REB as discussed above (#6).

2. How do you anticipate disseminating your research results? Check all that apply.

- Thesis/Dissertation/class presentation
- Media (e.g. newspaper, radio, TV)
- Presentations at scholarly meetings
- Published article, chapter or book
- Internet
- Other, explain: Case Studies

Directly to participants, describe how**:

The subjects who participate in the study will also, upon request, be granted full access to his or her results once the data has been analyzed.

3. Describe your plans for protecting data as well as preserving or destroying data after the research is completed. For all data (e.g. paper records, audio or visual recordings, electronic recordings), indicate the:
a) means and location of storage (e.g. a locked filing cabinet, password protected computer files)

b) time duration of storage. (REB requires that data be archived for a minimum of three (3) years)

c) final disposition (archive, shredding, electronic file deletion)

(See Section IV-3 of the Guidelines)

All records related to this study will be securely stored in Jonathan Silbernagel's office in a locked filing cabinet and/or a password-protected folder on his computer. Upon completion of data analysis, all data will be stored with Dr. Patrick Neary. We will comply with the REB requirement of keeping data for the minimum three year period. However, due to the upcoming advancement in technology for the SCG device, data may be kept for years to come. If decided to dispose of the data, all paper records will be shredded and all electronic records will be securely deleted and formatted from their place of storage.

Your signature(s) below acknowledges that:
- the information in this application is correct to the best of your knowledge
- you will notify the REB of any changes or amendments to this application
- contact with human subjects in the proposed research will not commence until ethical approval is obtained
all members of the research team are aware of, and adhere to, University of Regina regulations and policies for conducting research, including the Tri-Council Policy Statement (TCPS).

Signature of Applicant(s)  
Signature of Advisor or Instructor

Reminder: Please attach a copy of your recruitment letter, consent form, questionnaire, interview questions, etc.

Enclosures:

- DPSC Cardiac Rehabilitation referral form
- Informed consent form for exercise program
- Informed consent form for SCG collection
- DPSC Emergency procedures
Cardiac Rehabilitation and Risk Reduction Classes

I  Staff: Physician, Nurse, Consultant and students:

1. Nearest Staff member goes to the patient and evaluates as per CPR Protocol. Call for help and send someone to call 911 if deemed necessary. The Nearest Blue Alarm should be pulled to notify other staff on the floor and the FLC staff as well as the medical staff in the Allied Health Centre. Other class clients are instructed to go to the opposite side of the area from the emergency

2. Physician and/or other staff proceed to the side of the patient. The first person brings the AED (nearest person to cart brings it) and another brings the crash cart and portable blood pressure cuff with them. Once by the victim’s side, the staff member gets the AED ready, applies and activates it if the client is unconscious or has no pulse. One person retrieves the oxygen tube from the cart, and gets the ECG (3) leads ready for placement. He/she will access cardiac rhythm and follow appropriate ACLS guidelines (if ACLS trained). He/she will assist the Nurse/Doctor with medicine and procedures.

3. Emergency procedures activated if necessary:
   i) Nearest staff will: Establish unresponsiveness and attempt to arouse patient.
ii) He/she designates a volunteer/student to wait for EMS at the Building south doors. The staff member (consultant) will also be in charge of crowd control or will designate a volunteer/student to do so. If there is a Cardiac Arrest, 2 volunteers will go to the main entrance. One will guide one of the ambulance attendants up the south west stairs. The second will guide the other to the elevator.

iii) The remaining staff (Physician and nurse and/or consultant/student) performs emergency procedures as indicated by physician. These may include 2 man CPR with bag-mask breathing apparatus, defibrillation, drug administration and intubation.

iv) The staff member who made 911 call returns to patient and assists with his/her care, records emergency procedures, checks on other clients, etc. One staff member should record all emergency procedures implemented on the Emergency Rescue Record kept on clipboard by crash cart.

v) The EMT’s take over when they arrive

vi) One staff member calls the patient’s Cardiologist and/or Family Doctor and spouse or next of kin. Staff should check other class participants to make sure they are all right, and then make sure emergency records are complete.
II Staff: Nurse, Consultant and students:

i) The nearest Staff member goes to patient and evaluates as per CPR Protocol. The Nearest Blue Alarm should be pulled to notify other staff on the floor and the FLC staff as well as the medical staff in the Allied Health Centre. The other class clients are instructed to go to the opposite side of the work out area.

ii) The second responder brings the AED (nearest person to cart brings it) and another brings the crash cart and portable blood pressure cuff with them. Once by the victim’s side, the staff member gets the AED ready, applies and activates it if the client is unconscious or has no pulse.

iii) The consultant calls the Cardiac Ambulance (911) from the phone in the cardiac area and designates a volunteer to wait by the east doors to bring EMS to the cardiac area.

iv) The remaining staff performs emergency procedures as necessary. These may include 1 man CPR and possibly defibrillation and/or drug administration at the Nurse’s discretion.

v) The staff who made 911 call returns to assist with patient.
vi) All procedures should be recorded at the time implemented or immediately following arrival of the EMT’s. Emergency Rescue Record forms are kept on a clipboard hanging on the crash cart.

vii) The other class participants should be watched as closely as possible.

viii) The EMT’s take over when they arrive.

ix) The Nurse or consultant calls patients cardiologist and/or family doctor and next of kin/spouse. The consultant should check other participants to make sure they are all right, and then make sure emergency records are completed.

Procedure with a client with Chest Pain:

1. First person to reach the client will have the Client stop exercising if they are and sit/lay down in the most comfortable position possible.

2. If physician is present, notify physician.

3. The Nurse will check vitals and Nitro spray will be administered if appropriate by the client him or herself.
4. EMS is activated after the third dose of Nitro spray if the chest pain continues.

5. The crash cart and AED are readied in case of Cardiac arrest.

6. A 12-lead ECG will be conducted if deemed appropriate by the nurse/consultant.

7. The consultant/student will phone EMS if necessary and send a volunteer to wait for them at the South Doors and ask FLC staff to control elevator.

8. A copy of the Clients book with meds and the ECG will also be given to EMS or to the client if he is being driven to the Hospital.

9. An incident report will be filled out after the client has been safely dealt with.
### Protocol for General Emergencies:

<table>
<thead>
<tr>
<th>Staff Member</th>
<th>Responsibilities</th>
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<tr>
<td>1. First to Witness</td>
<td>Pull the BLUE Alarm on the nearest corner post or in the labs. Blow the whistle 3 times. Call help to get someone. Alert RN to attend and follow CPR Protocol. Assess: Level of consciousness, Airway, Breathing, Circulation and then start CPR.</td>
</tr>
<tr>
<td>2. Exercise Specialist(s)</td>
<td>Bring AED followed by the second Nurse and/or physician person bringing the Crash Cart to client. Assist the RN and take over CPR as needed.</td>
</tr>
<tr>
<td>3. Exercise leader</td>
<td>Call 911 as per instructions from other staff. He/she will direct patients away from the emergency and control the crowd and spouses if present. Also retrieve the personal belongings of the client.</td>
</tr>
</tbody>
</table>
4. FLC Staff

   FLC Staff to control elevator on 1st floor and hold elevator while ambulance gets here. 2nd floor FLC staff makes sure the big double doors of the FLC Centre are open. Send 1 or 2 volunteers (2 if Cardiac arrest) to meet ambulance at south doors emergency fire exit by the west end of the track.

4. RN, ACLS Trained staff

   Assess patient status via ECG. Defibrillate as per AED guidelines and proceed until Ambulance arrives and EMT’s take over CPR.
Dr Paul Schwann Centre Referral

Name: 
Address: 
Home Phone: 
Date of Birth: (dd/mm/yy) 
Hospitalization #: 
Postal Code: 
Work Phone: 
SGI □ WCB □ Claim # 

Reason for Referral (please check one only):
- Cardiac
- CGPD
- Musculoskeletal Injury
- Osteoporosis Program (please list history of fractures below)
- Personal Training
- Risk Reduction/Lifestyle Changes
- TIA/Stroke (must be ambulatory)
- Other (Please Specify)

Musculoskeletal Diagnosis/Findings/Contraindications

Medical History
□ "Normal"  OR  □ Prone to Coronary Heart Disease

Risk Factors Present (please check all that apply):
- Cigarette Smoking
- Dyslipidemia
- Diabetes Mellitus IDDM or NIDDM (please circle one)
- Family Hx Premature CHD
- Hypertension
- Obesity

Other significant medical conditions (please check and comment)
□ Cardiac Client
- □ Myocardial Infarction
- □ CABG
- □ PTCA
- □ CAD
- □ Valvular Disease
- □ Other (please explain)

□ Accidents
□ Allergies
□ Epilepsy
□ Infections
□ Mental Illness, Neuromuscular Impairment
□ Osteoporosis, Osteopenia
□ Respiratory Disease
Laboratory Data (if available)

Blood Pressure: / Medication: (check one) □ Yes  □ No

Blood Lipids: Total-C HDL-C LDL-C TG Hemoglobin:

Fasting: (please check one) □ Yes  □ No  Blood Glucose:  HbA1C:

12 Lead Electrocardiogram: (please check applicable boxes & attach if available)
□ Not Available
□ Within Normal Limits
□ Abnormal (please explain)

Present Medications (Type & Dosage)

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Date of last physical examination: ___/___/___ (dd/mm/yy)

Other Comments:

________________________________________________________
________________________________________________________

IMPORTANT: The above-listed person is capable of participating in a laboratory
controlled physical fitness test under the direct guidance and supervision of:

□ Laboratory Technician OR □ Physician (Please check one)

Referring Physician: __________________________ Telephone: ( )

(Please Print)

Signature: __________________________ Date: ___/___/___ (dd/mm/yy)

Please return to patient, mail or fax to:
Dr. Paul Schwann Applied Health & Research Centre
University of Regina, Regina, SK S4S 0A2
Fax: (306) 585.3563  Tel: (306) 585.4004

For more information on our programs and services or to download referral forms please
visit our website at http://www.uregina.ca/dpsc

□ Please check here to receive more referral pads

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Page 2 of 2
Effects of Exercise Training on Heart Failure Measured Using Seismocardiography

May 1, 2012
May 1, 2015
April 9, 2014

Dr. Larena Hoeber
Chair, Research Ethics Board
INFORMATION SHEET FOR RECORDING A SEISMOCARDIOGRAPHY TRACING

Project Title: Effects of exercise training on heart failure measured using seismocardiography.

EXPLANATION OF THIS RESEARCH

Seismocardiography (SCG) is a non-invasive technology that provides a measure of the forces generated by the contraction and relaxation movements of the heart. SCG is a more sophisticated measurement than the electrical activity (ECG) of the heart, and can help to identify cardiac problems at an earlier stage. This device is safe and has been used previously in 100’s of clinical trials at the Pasqua General Hospital in Regina, SK, and we have used this technology previously on 100’s of participants in the Faculty of Kinesiology at the University of Regina.

In this experimental trial you will be asked to wear an electrode similar to an ECG electrode, which you have had in the past. The SCG will be attached to this electrode, and then it will be attached to your skin at over the sternum (middle) of your chest. You will then be asked to relax and breathe normally for a couple of minutes before we record a resting sample. The resting sample will take approximately 3 minutes to collect, and therefore the entire procedure will take less than 5 minutes. In addition to collection of the
SCG, your blood pressure will be recorded using a finger blood pressure cuff (called plethysmography) as well as a 12-lead ECG. You will also be asked to wear a portable muscle oximeter called the PortaMon NIRS device at the time of the 60m walk test. You will be required to complete SCG tracing, ECG tracing, blood pressure, and PortaMon on four separate occasions; at the start of your exercise program, at six weeks into your exercise program, at twelve weeks into your exercise program and at the eighteen week point of your exercise program. You will also be given two DXA (dual-emission X-ray absorption) scans, at week 0 and 18, to determine changes in your muscle mass during the eighteen week program. During participation in this study, you will be required to keep track of and record any exercise that you participate in away from the Dr. Paul Schwann Centre using exercise journals that will be given to you. Your results will be kept completely confidential, and all data collected in this project will be secured in a locked office. Only group means will be used in potential publications.

**BENEFITS TO BE EXPECTED**

Although this trial will not provide you with an immediate benefit, it is proposed that our SCG testing and data collection will allow better preventative interventions programs to be developed in the future, and provide more sophisticated and meaningful monitoring of patients in acute care and rehabilitation management situations.

**POTENTIAL RISKS AND INJURIES**

There is potential risk for bodily harm to occur as a result of participation in this study. As with most exercise programs the possibility exists that injuries of physical nature may occur.
along with exacerbation of cardiac symptoms and on rare occurrences even death. The staff of the Dr. Paul Schwann Centre tries to minimize the risks of exercise by monitoring the performance of your exercise sessions along with blood pressure, heart rates and ECGs. If a medical emergency occurs, the staff of the Dr. Paul Schwann Centre is trained in Health Care CPR and employs similar equipment to that of an ER. All emergencies will be handled by physician on staff along with a registered nurse and will operate in conjunction with local Emergency Medical Services and the local hospitals.

INQUIRIES

We would invite you to ask any questions about the SCG device, the procedures and protocols to be used during this testing. If you have any doubts about what is expected, please ask for further clarification and we will be happy to explain. The principle investigator for this trial is Dr. Patrick Neary, and Mr. Jonathan Silbernagel will assist in the data collection.
APPENDIX C – Informed consent for seismocardiography collection

Consent for Subjects to Participate in this Research Project

Title of Project:  *Effects of exercise training on heart failure measured using seismocardiography.*

I acknowledge that I have been referred by my general practitioner or cardiologist for participation in the Dr. Paul Schwann Centre Cardiac Rehabilitation program.

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. If I wish, my data will be deleted, destroyed and omitted upon withdrawal.

I am aware that any information obtained by the staff of the Dr. Paul Schwann Centre regarding my personal history may be used for the purposes of this research study.

I have been informed that all information collected from me for the purpose of this study will be treated confidentially by unique identification number, and will be locked in the desk of the research assistant, Mr. Jonathan Silbernagel. In the event of an emergency, applicable health information will have to be disclosed to Emergency Medical Services for treatment in the situation.
I have been assured that I may contact Dr. Patrick Neary at patrick.neary@uregina.ca (306-585-4844) or Jonathan Silbernagel silbernj@uregina.ca (306-585-4361) at any time if I have questions or would like more information about the study.

I am aware that all data collected for the purpose of this research study will be held by Jonathan Silbernagel and upon completion of data analysis; Dr. Patrick Neary will maintain the data indefinitely.

I may obtain a copy of my results, upon completion of the study, by contacting either of the above persons.

I am aware that Jonathan Silbernagel, an employee of the Dr. Paul Schwann Centre Cardiac Rehabilitation program, will also act as a research assistant and co-applicant for this research study.

This project was approved by the Research Ethics Board, University of Regina (REB #). If I have any questions or concerns about their rights or treatment as subjects, I may contact the Chair of the Research Ethics Board at (306) 585-4775 or by e-mail at research.ethics@uregina.ca.

I understand the contents of this form, and I agree to participate in this research study under my own volition.
I have received a copy of the information sheet and this informed consent form for my records.

NAME (Please print legibly): __________________________________________

ADDRESS: _________________________________________________________

SIGNATURE: _______________ WITNESS: ______________________

DATE: _________________________
INFORMED CONSENT FOR EXERCISE REHABILITATION

1. Purpose and Explanation of Procedure

To improve my physical capacity and generally aid in my medical treatment for heart or chronic disease, I hereby consent to enter a cardiac rehabilitation and/or chronic disease and/or risk reduction program that may include cardiovascular monitoring, physical exercise, dietary counselling, stress reduction, and health education activities. The levels of exercise that I will perform will be based on the condition of my heart and circulation at the time of entry to the program. I will be given explicit instructions regarding the amount and kind of exercise I should do. Organized exercise sessions will be available on a regularly scheduled basis. The exercise specialist in consultation with the exercise program director and/or physician, and depending on my progress, may adjust my exercise sessions for continued improvement and safety. I understand that I am expected to attend regularly and to follow physician and staff instructions with regard to any medications that may have been prescribed, exercise, diet, stress management, and smoking cessation. If I am taking prescribed medications, I have already informed the program staff and further agree to inform them promptly of any changes my doctor or I have made with regard to use of these.
I have been informed that in the course of my participation in exercise, I will be asked to complete the activities unless such symptoms as fatigue, shortness of breath, chest discomfort, or similar occurrences appear. At that point, I have been advised that it is my complete right to stop exercise and that it is my obligation to inform the program personnel of my symptoms. I recognize and hereby state that I have been advised that I should immediately upon experiencing any such symptoms inform the program personnel of my symptoms.

2. Monitoring

I understand that during the performance of exercise, I will report to the nurse for a pre-exercise blood pressure and ECG every two weeks, or as needed. I will also monitor my own pulse rate as instructed, before, during and after each session. I also understand that the staff may reduce or stop my exercise program when findings indicate that this should be done for my safety and benefit.

3. Risks and Discomforts

There exists the possibility during exercise of certain changes occurring during the exercise sessions. These include abnormal blood pressure, fainting, disorders of heart rhythm, and in rare instances heart attack, stroke, or even death. Every effort will be made to minimize the risks by proper staff assessment of my medical condition before designing my program. Thereafter, all observations made by the staff will be used to carefully control my exercise effort. I have also been informed that emergency equipment and personnel are readily available to deal with unusual situations should
occur. I understand that there is a risk of injury, heart attack, stroke, or even death as a result of my exercise, but knowing those risks, it is my desire to participate as herein indicated.

4. Benefits to Be Expected

I understand that participation in the rehabilitation program may or may not benefit me in any way. The results obtained may help in evaluation in what types of activities I may engage in safely during my daily life. No assurance can be given that the rehabilitation program will increase my functional capacity although widespread evidence indicated that improvement is usually achieved.

5. Confidentiality and Use of Information

I have been informed that the information obtained from the rehabilitation program will be treated as privileged and confidential as described in the Health Insurance Portability and Accountability Act of 1996. It will not be released or revealed to any person except my referring physician or specialist without my express written consent, I do, however, agree to have my data and use of any information for research and statistical purposes with my right to privacy retained. Any other information obtained, however, will be used only by the program staff in the course of prescribing exercise for me, planning my rehabilitation program, or advising my relevant care provider(s) including professional, and administrative staff within the Allied Health Centre on a “need to know” basis.
I also understand that it is my responsibility to take care in not leaving my information book unattended while at my session and that it is my right to take my book home after my exercise session, and bring it back for each session. I understand that if I choose to leave my book at the Dr. Paul Schwann Centre (DPSC) I am assuming the risk of my personal information being potentially exposed in extreme case such as theft.

6. Responsibility of the Participant

Information I possess about my health status or previous experiences of heart-related symptoms (e.g. shortness of breath with low-level activity, pain, pressure, tightness, heaviness in the chest, neck, jaw, back, and/or arms) with physical effort may affect the safety of my exercise session. My prompt reporting of these and any other unusual feelings with effort during exercise session itself is very important. I am responsible for fully disclosing my medical history, as well as symptoms that may occur during the sessions.

To gain expected benefits, I must give priority to regular attendance and adherence to prescribed amounts of intensity, duration, frequency, progression, and type of activity.

To achieve the best possible preventative health care:

**DO NOT:**

Withhold any information pertinent to symptoms from the Exercise Consultant, Nurse, Physician, Exercise Program Director, or other professional personnel in the DPSC.

A. Exceed my target heart rate.
B Exercise when I do not feel well.
C Exercise within two hours of eating a large meal
D Exercise after drinking alcoholic beverages.
E Use extremely hot water during showering after exercise (stay out of sauna, steam bath, and similar extreme temperatures).

**DO:**

A Report any unusual symptoms, which I experience before, during, or after exercise, after my scheduled session.
B Only perform exercises/activities prescribed by my consultant(s), and only in the correct technique that has been previously demonstrated by my consultant(s). I accept responsibility for myself for all other exercises I choose to perform, and understand that they will be performed at my own risk.
C Understand that I may stop or delay any further participation in the activity I desire and that the activity may be terminated by the Exercise Consultant based on any symptoms of distress, abnormal response or safety concern.
D Understand that I may ask any questions or request further explanation or information about the procedures and program at any time before, during and/or after the physical activity.
E Understand that the use of the facility, preceding or following the allotted length of time for the service being provided will not be monitored by or under the responsibility of a DPSC consultant and will be performed at my own risk. I will inform my Exercise Consultant if I plan to use other
facilities at the site. At that time I must accept responsibility for myself, and exercise at my own risk.

Any questions about the rehabilitation program are welcome. If you have any doubts or questions, please ask us for further explanation.

7. Attendance and Refund Policy

Because the DPSC’s Cardiac Rehabilitation and/or Chronic Disease and Risk Reduction Program does not open until 8:00am on Monday, Wednesday and Friday and 2:30pm on Tuesday and Thursday, please do not arrive prior to 7:45am or 2:15pm on the day of your program.

All services and fees are NON-REFUNDABLE and cannot be extended. Your visits must be used up within one year from the date of purchase. Once the book is expired or you have used up all your sessions, you have the option of purchasing more sessions providing there is room in the program. You may be asked to attend the continuing program at a different time.

8. Inquiries and Freedom of Consent

I further understand that there are remote risks over than those previously described that may be associated with this program. Despite the fact that a complete accounting of all remote risks is not entirely possible, I am satisfied with the review of these risks that was provided to me, and it is still my desire to participate.
I acknowledge that I have read this document in its entirety or that it has been read to me and I understand the Rehabilitation Program in which I will be engaged. I accept the rules and regulations set forth. I consent to participate in this Program.

9. Privacy Pledge

_________________________________________  ______________
Patient’s Signature                      Date

_________________________________________  ______________
Witness’s Signature                       Date

_________________________________________  ______________
Program Staff Signature                   Date

APPENDIX E – Sample Initial Exercise Prescription

***BLOOD PRESSURE AND ECG CHECK EVERY 2 WEEKS***

Your Training Heart Rate range is $\frac{\leq 20}{\text{beats/10 sec.}}$

Your exercise prescription includes:

<table>
<thead>
<tr>
<th>Activity</th>
<th>Time/Duration</th>
<th>Intensity</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Walk</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bike</td>
<td>5 min</td>
<td>BS, C 20-30 Km/h x1</td>
<td></td>
</tr>
<tr>
<td>Recumbent Bike</td>
<td>5 min</td>
<td>Random 1 20-30 Km/h x1</td>
<td></td>
</tr>
<tr>
<td>Row</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arm Ergometer</td>
<td>200 - 300 Reps</td>
<td>0-10 Watts</td>
<td>x1-3</td>
</tr>
<tr>
<td>Weight Circuit</td>
<td>10 Reps</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elliptical Trainer</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Floor Hockey</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Biodex</td>
<td>5 min</td>
<td>Level 2-3 60-80 RPM</td>
<td>X1</td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Keiser Weight Equipment:

- Upper Back 40
- Leg Press 210
- Chest Press 40
- Leg Extension 45
- Triceps 90
- Seated Leg Curl 50
- Lat Pulldown 50
- Hip Abductor 75
- Arm Curl 20
- Military Press 30