The Cardiovascular Effects of Recreation Hockey in Middle-Aged Men

By

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A thesis submitted in conformity with the requirements for the degree of Master of Science
Graduate Department of Exercise Science
University of Toronto

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Abstract

The present study examined the hemodynamic response to recreational hockey (n= 22) in middle-aged men (53±6 yrs). Study participants were equipped with ambulatory blood pressure and heart rate monitoring equipment prior to a weekly hockey games. Participants were monitored throughout the duration of their hockey game for “On-Ice” responses and during seated bench time (“Bench”), and for a brief period afterwards. On-Ice HR’s and blood pressures were significantly higher than values obtained during maximal cycle exercise (HR 174±8.9 vs. 163±11.0 bpm) (SBP 17%; DBP 15%) (p<0.05), Blood pressures decreased throughout the duration of the game while HR increased significantly. The On-Ice endocardial viability ratio (EVR), an index of myocardial oxygen supply and demand, did not change from early (1.56±0.05) to late (1.44± 0.06) in the game. In conclusion, recreational hockey is an extremely vigorous form of interval exercise that produces cardiovascular responses exceeding intensities commonly recommended for continuous training.
Acknowledgement

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## Abbreviations

<table>
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<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>AED</td>
<td>Automated External Defibrillators</td>
</tr>
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<td>Alx</td>
<td>Augmentation Index</td>
</tr>
<tr>
<td>AMI</td>
<td>Acute Myocardial Infarction</td>
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<td>BRS</td>
<td>Baroreflex Sensitivity</td>
</tr>
<tr>
<td>CE</td>
<td>Continuous Exercise</td>
</tr>
<tr>
<td>CO</td>
<td>Cardiac Output</td>
</tr>
<tr>
<td>CV</td>
<td>Cardiovascular</td>
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<tr>
<td>DBP</td>
<td>Diastolic Blood Pressure</td>
</tr>
<tr>
<td>DPTI</td>
<td>Diastolic Pressure Time Index</td>
</tr>
<tr>
<td>DTF</td>
<td>Diastolic Time Fraction</td>
</tr>
<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
</tr>
<tr>
<td>E:R</td>
<td>Exercise to Rest Ratio</td>
</tr>
<tr>
<td>EVR</td>
<td>Endocardial Viability Ratio</td>
</tr>
<tr>
<td>HIIE</td>
<td>High Intensity Interval Exercise</td>
</tr>
<tr>
<td>HR</td>
<td>Heart Rate</td>
</tr>
<tr>
<td>HRR</td>
<td>Heart Rate Reserve</td>
</tr>
<tr>
<td>HRV</td>
<td>Heart Rate Variability</td>
</tr>
<tr>
<td>MET</td>
<td>Metabolic Equivalent</td>
</tr>
<tr>
<td>MVO₂</td>
<td>Myocardial Oxygen Consumption</td>
</tr>
<tr>
<td>NO</td>
<td>Nitric Oxide</td>
</tr>
<tr>
<td>PEH</td>
<td>Post Exercise Hypotension</td>
</tr>
<tr>
<td>PWA</td>
<td>Pulse Wave Analysis</td>
</tr>
<tr>
<td>PWV</td>
<td>Pulse Wave Velocity</td>
</tr>
<tr>
<td>RPP</td>
<td>Rate Pressure Product</td>
</tr>
<tr>
<td>SAC</td>
<td>Systemic Arterial Compliance</td>
</tr>
<tr>
<td>SBP</td>
<td>Systolic Blood Pressure</td>
</tr>
<tr>
<td>SCD</td>
<td>Sudden Cardiac Death</td>
</tr>
<tr>
<td>SPTI</td>
<td>Systolic Pressure Time Index</td>
</tr>
<tr>
<td>STTI</td>
<td>Systolic Time Tension Index</td>
</tr>
<tr>
<td>SV</td>
<td>Stroke Volume</td>
</tr>
<tr>
<td>TPR</td>
<td>Total Peripheral Resistance</td>
</tr>
<tr>
<td>TTI</td>
<td>Time Tension Index</td>
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Chapter I: Introduction

Rationale

It is estimated that more than 500,000 men over 30 years of age participate in recreational hockey across Canada. Most participants are engaged in semi-organized, recreational hockey with full equipment, without referee support, for weekly games lasting 60 minutes. Apart from musculo-skeletal injuries that may impede continued involvement by some, there are anecdotal reports of frequent cases of non-fatal adverse cardiovascular events or sudden cardiac death (SCD) during recreational hockey. There are 35,000 to 40,000 incidents of SCD in Canada per year. These concerns, along with less common cases of SCD in younger athletes, have in part, led to the placement of automated external defibrillators (AED) at hockey rinks across Canada. In spite of these efforts, little is known about the cardiovascular response to this type of activity in the adult population.

Despite the known benefits of physical activity at moderate to vigorous levels of intensity, the risks of coronary events increases with both age and intensity of effort. Hockey is an unusual form of high intensity intermittent exercise (HIIE) that involves bouts of near maximal effort combined with periods of total rest, typically in the seated position. However, little is known about the effect of bench sitting immediately following a brief period of near maximal exercise. It is also known that long-standing prior exposure to intensive physical activity can reduce the acute risks significantly. Notwithstanding, there are concerns of SCD or non-fatal cardiovascular events occurring during adult recreational hockey, due to the increased risk of adverse cardiac events associated with aging. As a response to the
growing concern of heart related incidents in Canada, the “Fit for Hockey” program was created in 2005, which provides education about cardiac risk. Unfortunately, there is a paucity of specific scientific evidence that is available to guide players, associations and health professionals towards specific strategies to lower risk, or to identify the physiological profile of a typical hockey game.

A major concern is the presence of occult coronary heart disease when myocardial oxygen supply/demand imbalance exists during intensive exercise. As exercise intensity is increased, an increase in systolic blood pressure combined with a marked increase in heart rate in response to exercise stress greatly increases myocardial oxygen demand. In addition, the increased heart rate during exercise shortens diastolic perfusion time, thereby reducing coronary reserve, resulting in a potential supply/demand mismatch. Little information is known regarding the potential implications of bench sitting immediately following intensive exercise, on myocardial oxygen supply and demand. Based on the observations of post exercise hypotension, a substantial decrease in diastolic blood pressure combined with an attenuated heart rate recovery may lead to an acute oxygen supply/demand mismatch following, rather than during vigorous exercise.

The purpose of the present study is to determine the cardiovascular consequences of a game of recreational hockey in middle-aged men. Holter electrocardiograms (ECG) for the assessment of heart rate and cardiac rhythms, and ambulatory measures of blood pressure during play, rest and recovery will be recorded. In addition, HR and BP will be used to estimate the myocardial supply/demand ratio during different points throughout a game.
Objectives
1) To compare the blood pressure and heart rate responses during maximal graded exercise to those recorded during a typical recreational hockey game in middle-aged men.
2) To assess how blood pressure and heart rate progress throughout the course of a game of hockey.
3) To assess the effect of a recreational hockey game on myocardial oxygen supply and demand.
4) To assess the effect of a 1-minute recovery on blood pressure, HR and myocardial oxygen supply and demand.

Hypotheses
1) Both blood pressure and heart rate will exceed the recommended training values and will approach, but not exceed maximal levels measured during a progressive cycle ergometer stress test.
2) HR will gradually increase throughout the course of the game. Blood pressure values will continuously increase as the game progresses.
3) There will be a significant reduction in myocardial oxygen supply/demand during the game when compared with rest. Supply/demand will be more compromised towards the latter stages of the game.
4) Following an on ice shift there will be a significant reduction in blood pressure while the player is seated on the bench. HR will recover rapidly following a player's shift.
Chapter II: Review of Literature

Cardiovascular Response to Exercise

At the onset of exercise there is an increasing oxygen demand from the exercising skeletal muscle, which is accommodated by an increase in cardiac output (CO) in order to increase blood and oxygen supply to the active tissues. Cardiac output is determined by heart rate (HR) and stroke volume (SV). The relationship between CO and the dynamic response to exercise has been summarized in numerous reviews. The summary below will focus on the cardiovascular physiology of high intensity interval exercise, continuous exercise and the time period immediately following exercise.

Continuous Exercise

The cardiovascular response to prolonged continuous exercise starts with an increase in cardiac output achieved with proportionate increases in HR and SV. However, during prolonged continuous exercise there is a gradual decrease in SV and CO is maintained by an increase in HR, known as cardiovascular (CV) drift. Rowell states CV drift is the result of a progressive increase in skin bloodflow, associated with thermoregulation and is accompanied by reductions in central venous pressure which ultimately leads to a reduction in SV that occurs as part of CV drift during prolonged exercise.

High Intensity Interval Exercise

High-intensity interval training (HIT) describes physical exercise that is characterized by brief, intermittent bursts of vigorous activity, interspersed by periods of rest or exercise that is at a much lower intensity. When compared to traditional endurance training, HIT can serve as an appropriate time-saving
alternate, for it can elicit equivalent or even superior changes in a range of physiological, performance and health related markers in both healthy individuals and diseased populations.\(^{15}\)

There are extremely limited data on the acute cardiovascular response to individual bouts of intermittent exercise. Briefly, one study noted that the recurring short recovery periods of intermittent exercise results in an improved matching of \(O_2\) delivery to \(O_2\) utilization (improved muscle perfusion) during the subsequent work periods compared to continuous exercise performed at the same work rate.\(^{16}\) The period following strenuous intermittent exercise is extremely important for the cardiovascular apparatus. For both a performance and health outcome, it is important to determine what form of recovery is most effective following HIT. The primary recovery modes for HIT are active upright recovery, and passive upright recovery.\(^{17}\)

Considering the limited data describing the acute cardiovascular responses to HIIT, it is important to examine the post exercise recovery following other high intensity exercise. Crisafulli et al.\(^{17}\) investigated the differences in the cardiovascular response during active and passive recovery following a single bout of supramaximal exercise. This study demonstrated that passive recovery was associated with a lower CO compared to active recovery. The lower CO observed during passive recovery was mediated by a faster reduction in heart rate and stroke volume following the cessation of exercise.

Active recovery appears to enhance subsequent exercise performance. It has been demonstrated that active recovery increases exercise performance and
decreases blood lactate levels during repeated bouts of moderate and high intensity exercise when compared with passive recovery. Spierer et al. examined the effects of active recovery following Wingate tests on both sedentary individuals and moderately trained hockey players. The results of the study indicated that active recovery increased the total work achieved during the repeated Wingate tests in both the sedentary and moderately trained hockey groups. Conversely, Dupont et al. showed that the time to exhaustion for intermittent runs (15s) at 120% of maximal aerobic speed was significantly longer following passive recovery compared to active recovery. The authors concluded that the energy required during the active recovery phase would result in less oxygen being available to the active muscles during the sprinting phase. Based on the aforementioned literature, it appears that there is no definitive answer as to which form of recovery is more effective and future research is warranted on this topic.

**Post Exercise Recovery**

The period following strenuous exercise it thought to be extremely important for the cardiovascular system. In fact, on stopping exercise, sudden changes in cardiac preload, afterload and contractility occur. These physiological responses have further implications for the cardiovascular system and drive both the HR and BP responses following exercise. While little research has been done on the period of time in between bouts of intermittent exercise it is important to understand what happens to the cardiovascular system as result of any cessation of exercise.
Heart Rate Recovery

The rise in HR accompanying exercise is the result of the combination of parasympathetic withdrawal and sympathetic activation. Immediately following exercise there is a reduction in HR, often labeled Heart Rate Recovery (HRR). Darr et al. demonstrated that HRR is characterized by 2 distinct phases; an initial exponential drop followed by a slower decline to resting levels. Vagal reactivation appears to be the principal determinant of HRR during the first 30 seconds following exercise, and this mechanism is independent of age and the intensity of exercise. Other factors that influence HRR include exercise intensity, baroreflex sensitivity, aerobic capacity, post-exercise norepinephrine concentration and resting HR. Individuals with a higher cardiovascular fitness ($VO_2 \text{max}$) and lower BMI appear to have a significantly faster HRR than less fit and lean individuals.

Heart rate recovery has also been known as a predictor of sudden cardiac death, because increased vagal activity has been associated with a reduction in the risk of death. Vivekananthan et al. sought to determine whether HRR predicts mortality independent of coronary artery disease following exercise. The results of the study showed that even after taking into account the severity of coronary disease, HRR was a strong predictor of mortality following exercise. Other studies have also demonstrated the relationship between HRR and SCD, however, aside from the vagal reactivation theory the mechanism by which HRR can predict SCD is relatively unknown.
**Post Exercise Hypotension**

A single bout of exercise can result in a reduction of blood pressure following the cessation of that activity for up to 2 hours; this phenomena is called post exercise hypotension (PEH)\(^{34}\). PEH can persist up to 17 hours after exercise in patients with hypertension\(^{35}\). Whereas significant BP reductions during recovery have been well documented after a single bout of dynamic aerobic exercise in humans with hypertension, its occurrence in normotensive humans is inconsistent\(^{36}\). Dujic et al.\(^{34}\) tested the magnitude of PEH following short-term maximal exercise. Their results indicate that although PEH may be more prominent and longer lasting following long term, moderate intensity exercise, it can still occur following short maximal bouts, albeit briefly. The study also noted that the subjects with the highest resting BP values had the largest PEH. Several studies have observed blood pressure decreases of approximately 8/9 (SBP/DBP) mmHg in normotensive population\(^{37-39}\) and 14/9 mmHg in hypertensive individuals\(^{40,41}\). The magnitude of the pressure reductions can be exaggerated in the seated position when compared to a standing position\(^{8}\). The magnitude of the blood pressure response following exercise has been debated. The former studies all had significantly higher BP reductions following exercise when compared to Cornelissen et al.\(^{36}\) who only observed a reduction in systolic blood pressure of 4-6mmHg.

Nitric oxide (NO) production may also play a potential role in PEH. Jungersten et al.\(^{42}\) suggested that NO production is increased following an acute bout of exercise. NO may contribute to PEH by blunting the vasoconstrictor response to α-adrenergic receptor stimulation.
The role of exercise intensity and its contribution to PEH is debated, with some studies reporting a relationship between intensity and PEH\textsuperscript{43,44} and others reporting no such relationship \textsuperscript{37,45}. Lacombe et al.\textsuperscript{46} sought to determine the PEH response following continuous vs. interval exercises, which were matched for total energy expenditure. The authors observed significant and prolonged PEH following both exercise conditions, with no difference between the subjects during the repeated measures design. Additionally, it was observed that baroreflex sensitivity (BRS) was reduced only following interval exercise, and that the changes in heart rate variability (HRV) were more pronounced following interval exercise.

Temperature also plays an important role in blood pressure regulation during exercise. With increasing body temperature, as a result of thermoregulation, blood vessels are dilated in order for blood to reach the skin\textsuperscript{47}, which has the effect of reducing systemic blood pressure. Physical exertion poses a challenge to thermoregulation by causing a substantial increase in metabolic heat production\textsuperscript{48}.

**Aging and Exercise**

Aging is associated with a decline in maximal aerobic capacity (VO\textsubscript{2} max)\textsuperscript{49-51}. Previous studies have provided evidence that the decline in VO\textsubscript{2} max is a result of decreases in maximal heart rate, maximal stroke volume and maximal ā-vO\textsubscript{2} difference\textsuperscript{51-53}. Hagberg et al.\textsuperscript{54} compared cardiovascular fitness between older male master athletes and young control athletes. There were no significant differences between the older and younger athlete’s stroke volumes and ā-vO\textsubscript{2} difference. The researchers observed that about two-thirds of the reduction in VO\textsubscript{2} max in the older athletes was the result of a reduced maximal heart rate.
Alterations in the neural control of the heart associated with aging have a profound effect on the cardiovascular system. Evidence suggests that compared to younger control subjects older adults show impaired; 1) baroreceptor control of heart rate and cardiac function; 2) baroreceptor modulation of the sympathetic drive to the peripheral circulation, and 3) cardiopulmonary stretch receptors (which tonically inhibit sympathetic tone, and the renal release of renin and vasopressin)\textsuperscript{55}. These factors may account for the elevated blood pressure and increased sympathetic activity frequently seen in the older population.

One of the major age-associated alterations in the cardiovascular response to exercise is the striking decrease in maximal heart rate \textsuperscript{56}. As mentioned previously, one of the primary determinants of HRR is maximal aerobic capacity. Considering VO\textsubscript{2} max is decreased with advancing age, there would be an associated reduction in HRR. Other variables that contribute to a faster HRR, such as baroreflex sensitivity also decrease with age\textsuperscript{57}. Dimkpa et al.\textsuperscript{27} showed that older individuals have a blunted HRR following maximal exercise (22±2.6% decline in 1 min) when compared to young control subjects (39±3.5% decline in 1 min). The study concluded that the blunted HRR was accounted for by changes in VO\textsubscript{2} max, BMI and RPE (exercise intensity).

Aging is also associated with an increase in arterial stiffness, which is associated with a decrease in systemic arterial compliance (SAC) \textsuperscript{58-60}. Arterial compliance reflects the ability of an artery to expand and recoil with cardiac pulsation and relaxation \textsuperscript{61}. The age-related increase in arterial stiffness manifests itself in a progressive increase in systolic blood pressure (SBP), a widening of the
arterial pulse pressure and an increase in the pulse wave velocity. This increase in SBP may consequently enhance LV afterload and may lead to increases in myocardial energy cost. The increase in afterload can also impair LV pump function which can have serious implications related to exercise capacity. Tanaka et al. demonstrated that aging results in increased arterial stiffness regardless of prior habitual physical activity level. They also noted that the increase in arterial stiffness is correlated with reduced aerobic capacity. Additionally, although exercise training cannot prevent the increase in arterial stiffness throughout the aging process, the authors noted that lifelong physical activity could attenuate the increase, and therefore increase the cardiovascular fitness when compared to sedentary counterparts.

**Cardiovascular Response to Ice Hockey**

Limited data exist on the acute cardiovascular response to hockey in the general population. Hockey is characterized by periods of high intensity exercise interspersed with periods of complete rest. Montgomery reported that peak, on-ice heart rates exceed 90% of maximal value, while average heart rates are around 85%. Atwal et al. examined the heart rate response to hockey in men older than 35, through Holter ECG monitoring. The researchers observed that max HR recorded during the game was consistently 85-100% of the subjects’ age-predicted maximum, which exceeds the recommended exercise training intensity of 66-95% HR max. As a result of the intermittent play of hockey, heart rate values fluctuate constantly depending on whether a player is on ice, or on the bench. Figure shows the heart rate profiles of 12 forwards during a minor league hockey game.
Due to the nature of a Canadian Hockey League shift (extremely high intensity shifts lasting 30-90 seconds), the high heart rate is reflective of the high-energy requirements, and it can be speculated that oxidative metabolism is not fully capable of satisfying an athlete’s energy demands\textsuperscript{68}. It has also been reported hockey athletes require both highly developed aerobic and anaerobic glycolytic and phosphagen energy pathways, as it is these energy systems which provide the ATP needed to sustain repetitive high energy power and endurance outputs incurred during practice and competitive play\textsuperscript{69}. It is important to note that the nature of recreational hockey is far different and less intense than professional ice hockey.
Montgomery \textsuperscript{70} assessed a game of “old-timer” hockey using both heart rate telemetry and time-motion analysis. The study noted that in a typical 60-minute game, old-timers were on the ice for 27 minutes at an average of 70% maximal heart rate. An important observation from this study was that the exercise to rest (E:R) ratio was around 1 for old timer hockey, compared to elite hockey where players sit on the bench almost 3 times longer than they play. A major issue with long on-ice shifts at high intensity is the accumulation of lactate in the muscle. If sufficient lactate is produced the increase in muscle acidity causes metabolic and contractile disturbances that result in decreased work performance\textsuperscript{71}

Twist and Rhodes \textsuperscript{69} report that 60-70% of the energy requirements of the body during periods of moderate activity within an ice hockey game are fueled by aerobic metabolism, but note that this contribution is subject to variability due to inter and intragame variations in intensity, level of competition, position, and the player’s level of conditioning. The fact that such a high portion of hockey is driven by aerobic energy sources has large implications on the CV system and the CV response to hockey may be similar to that of continuous cardiovascular exercise as a result of longer shift durations than typical intermittent exercise. This study will help bring better understanding to demands placed on the cardiovascular system.

**Cardiovascular Risks and Exercise**

While a lot has been made of both the risks and benefits of physical exertion, it is important to look at the intensity required to elicit an adverse cardiac event. It is well recognized that heavy physical exertion sometimes immediately precedes, and indeed appears to trigger the onset of acute myocardial infarction (AMI) and
can lead to sudden cardiac death (SCD)\(^{72-74}\). Vigorous exercise is defined as an absolute exercise work rate of at least 6 metabolic equivalents (METs), which is equivalent to an oxygen uptake of \(21\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}\). Six METs is approximately the energy requirements of activities such as jogging. While METs are helpful in defining an objective and absolute level of exercise intensity, the relative intensity may be more relevant for any given individual. For this purpose, a more appropriate definition of vigorous, is exercise that elicits a heart rate of 77-93% of a healthy individual’s age-predicted maximum\(^{76}\). It has been shown that AMI can be triggered by bouts of vigorous activity\(^{77-79}\). However, it has not been demonstrated that low levels of physical exertion can trigger AMI suggesting that the exposure-response relationship may only elicit an adverse response at the higher end of the spectrum.

When looking at the intensity of physical exertion in relation to SCD it is important to examine individual’s habitual physical activity level as well. Individuals who engage in little physical activity are more susceptible to cardiac events following intense physical activity than those who are engaged in regular physical activity. Albert et al.\(^{80}\) conducted a prospective, cross sectional study to determine the triggers of cardiac death by vigorous exertion. The authors noted that men who rarely engage in vigorous exercise had a relative risk of sudden death of 74.1 in the period during and 30 minutes after exertion. The relative risk is the risk of sudden death during and 30 minutes after an episode of vigorous exertion, as compared with the risk during periods of lighter exertion or none. In comparison, men who exercise at least 5 times per week had relative risk of 10.9. Thompson et al.\(^{75}\) also noted that habitual physical activity could reduce the risk of adverse
cardiac events. They demonstrated that the overall incidence of SCD decreased from 18 events per 1 million person hours in the least physically active individuals to only 5 events per 1 million person hours in the most physically active. In a study done by Mittleman et al. the researchers interviewed 1228 patients who had experienced an acute myocardial infarction (AMI) as a result of intense physical exertion. Although the authors were not looking at SCD, in some cases AMI is a direct result of myocardial ischemia, which has also been linked to SCD. The results of the study indicated that those individuals who rarely participate in vigorous physical activity (less than once a week) had a relative risk of 107. This compared to a relative risk of 2.4 among those individuals who reported regular heavy physical exertion. Similar results regarding habitual physical activity level and a decreased risk of SCD and other cardiac events have been found in the literature as well. Siscovick et al. noted a higher incidence of primary cardiac arrest in those who were less habitually active versus those who maintained regular habitual activity. Exercise has been coined “a double edge sword” due to the nature of its protective and harmful effects.

Myocardial Oxygen Supply and Demand

The increase in CO during exercise is achieved by modest increases in SV and more importantly, significant increases in HR following the initiation of exercise. The increase in systolic blood pressure is proportional to a greater myocardial oxygen cost. The increased metabolic demand of the heart requires a matched increase in oxygen supply to the myocardium. Myocardial ischemia occurs when
there is inadequate myocardial oxygen supply predominantly in the subendocardial layer of the myocardium 7.

The subendocardial zone of the heart has a lower oxygen tension and therefore requires more anaerobic metabolism given that this region of the myocardium is subject to greater mechanical stress. As a result, there is increased vulnerability of the subendocardium to ischemia with increased risk of necrosis 86. During systole, there appears to be a redistribution of myocardial blood flow as a result of a transmural gradient of myocardial pressure 87. The net effect is reduced blood flow in the endocardium when compared to the epicardium.

**Factors Determining Oxygen Demand**

Exercise is the most important physiological stimulus that leads to an increase in myocardial oxygen demand. The major determinants of myocardial oxygen demands include heart rate, contractility and ventricular work 88. Afterload, BP and arterial stiffness also play a role in oxygen demand and will be discussed in detail later. It is estimated that contractility contributes 15-20% of the increase in myocardial oxygen consumption during exercise, while heart rate accounts for 50-70% of the increase in oxygen consumption 88. Myocardial oxygen consumption increases in conditions of tachycardia and it is possible to see a 5-fold increase in the transition from rest to exercise 89.

**Myocardial Blood Flow**

In order to maintain an adequate supply/demand balance during exercise, as oxygen demand increases there must be a proportional increase myocardial oxygen
supply. Considering resting oxygen extraction is typically around 70-80%\textsuperscript{88}, there is little reserve for an increase in oxygen demand. As a result, in order to compensate for an increase in demand, oxygen supply to the myocardium must be driven by increases in myocardial blood flow. Myocardial blood flow in human subjects at rest is approximately 0.5-1.5 ml\textbullet} min\textsuperscript{-1}•g myocardium\textsuperscript{-1} \textsuperscript{90,91}. During exercise, human myocardial blood flow can reach approximately 3 ml\textbullet} min\textsuperscript{-1}•g myocardium\textsuperscript{88}. Figure 2 demonstrates the relationship between heart rate and myocardial blood flow in humans. The figure also shows myocardial blood flow for rats, which are substantially higher at both rest and exercise values when compared to a human.

\begin{figure}
\centering
\includegraphics[width=\textwidth]{myocardial_blood_flow.png}
\caption{Myocardial Blood Flow vs. Heart Rate in Humans\textsuperscript{88}}
\end{figure}

Indirect Indices of Myocardial Oxygen Consumption
A primary determinant of coronary blood flow is the effective perfusion pressure of the coronary bed. However extravascular compressive forces mediate
the perfusion pressure. Extravascular forces are exerted on the compressible intramural coronary vasculature by the surrounding myocardium effectively creating a backpressure that acts to impede coronary flow\textsuperscript{88}. The effect of the extravascular forces appears to be much greater during systole. Gregg and colleagues (1957)\textsuperscript{92} were one of the first laboratories to conclude that coronary blood flow is in fact impeded during systole. The authors measured coronary flow in beating canine hearts, and in order to assess the role of systole, induced prolonged periods of asystole and fibrillation. The contracting myocardium generates an extremely high level of intramyocardial pressure that compresses the coronary microvasculature, thereby impeding blood flow\textsuperscript{93}. This systolic arterial flow impediment has been repeatedly observed in numerous studies on humans\textsuperscript{94,95}. In contraction, the total vascular volume is reduced which leads to an increase in vascular resistance thereby resulting in arterial inflow impediment and venous outflow augmentation\textsuperscript{96}. These findings emphasize that the main contribution of coronary blood flow to the endocardium takes place during diastole and not systole. As a result of the increased HR in response to exercise, the proportion of cardiac cycle time that the heart is in systole is greatly increased. With an increased proportion of time spent in systole the duration of diastole is thereby reduced and so is the time available for myocardial perfusion.

**Systolic Time Tension Index**

The systolic time tension index (STTI), more commonly referred to as the TTI has been related to the oxygen demand of the myocardium\textsuperscript{97,98}. Sarnoff and colleagues\textsuperscript{97} estimated the value of TTI to equal the mean value of systolic blood
pressure multiplied by the duration of systole. The TTI is measured in mmHg seconds and is an index of the total tension developed by the myocardium per beat. It has been established that the area under the left ventricular pressure curve in systole is related to left ventricular oxygen consumption\textsuperscript{97}. Studies have noted that wall pressure and wall tension under the area of the curve are equally effective in determining myocardial oxygen consumption\textsuperscript{97,98}. In addition it is much easier to measure pressure than it is tension. Consequently, it is more appropriate to describe the area under the left ventricular pressure curve in systole the pressure time index (SPTI) as opposed to the STTI.

**Other Indirect Indices of MVO\textsubscript{2}**

Rate pressure product (RPP) is equal to SBP*HR. This index is highly correlated (r=0.83) to myocardial oxygen utilization\textsuperscript{99} yet is inadequate for studies of coronary flow regulation\textsuperscript{100}.

Bretschneider et al.\textsuperscript{6} proposed an alternative method to the systolic time tension index for indirect determination of MVO\textsubscript{2}. The product of systolic blood pressure and ejection time was replaced by the square root of heart rate (SBP x $\sqrt{HR}$). The modified time tension index is based on the relationship between the total "electrical" systolic duration per minute and the square root of heart rate.

Baller et al.\textsuperscript{6} sought to compare the various indirect measurements of MVO\textsubscript{2} to the direct measurement via catheterization. The study demonstrated that ejection time bears an inverse statistical relationship with contractility. Therefore, the three indirect measures studied (RPP, TTI and modified TTI) will correlate differently with actual MVO\textsubscript{2} depending on the contractile state of the heart. At
normal and moderately increased contractility TTI had the strongest correlation with direct MVO₂ in the study (r=0.96). However, in periods of increased contractility TTI severely underestimates MVO₂ by about 25% because values of contractility are not taken into account. Interestingly, the modified version of TTI (SBP x √HR) achieves the best overall correlation to MVO₂ (r=0.80) when examining both moderately and severely increased contractile states. RPP and TTI have an overall correlation to direct MVO₂ of r=0.65 and r=0.63 respectively. This study demonstrates how the indirect measures of MVO₂ can vary with differing states of myocardial contractility and therefore, exercise intensity. It is therefore important to determine the appropriate indirect measure based on the intensity of each study.

**Diastolic Time Tension Index**

The diastolic pressure time index (DPTI) represents the area between the aortic and left ventricular pressures in diastole. DPTI can be calculated by the average diastolic pressure minus the mean left ventricular pressure (5mmHg) multiplied by the time in diastole. When the subendocardial vessels are maximally dilated, flow through them depends on the driving pressure (difference between the pressure in the coronary arteries and the opposing pressure in the small coronary vessels), time and blood viscosity. Therefore, DPTI is a measure of myocardial oxygen supply. In order for DPTI to have validity, it must be assumed that the coronary vessels are maximally dilated. Coronary vessels are assumed to be maximally dilated during exercise considering that during this stress coronary blood flow can increase up to fivefold.
**Endocardial Viability Ratio**

The endocardial viability ratio (EVR) is an indirect measurement of the ratio between oxygen supply and demand. EVR can be calculated using the indirect indices of myocardial oxygen supply and demand. EVR is equal to the Diastolic Pressure Time Index (DPTI) divided by the Systolic Time Tension Index (STTI).

Figure 3 demonstrates how the EVR can be affected by tachycardiac heart failure, as a result of severely reduced diastolic perfusion, paired with increased systolic oxygen demand.

**Figure 3.** DTTI and STTI in a physiological situation; inferior part: DTPT and STTI in a pathological situation of tachycardiac heart failure. Note that the area for DPTI is small leading to marked reduction of the DPTI/STTO ratio. Taken from Baller et al.102

As a result of the unique physiological responses to exercise cessation (HRR and PEH), one would expect that a diminished ability to recover heart rate following exercise, combined with a dramatic drop in diastolic blood pressure would lead to a severely reduced EVR. If HR cannot decrease adequately following an acute bout of exercise, time in systole remains elevated thereby reducing the diastolic phase. This
may reduce diastolic myocardial perfusion and lead to heightened risk of ischemia. Forjaz et al.\textsuperscript{45} demonstrated that at exercise intensities of 50% and greater myocardial oxygen consumption (as measured by RPP) did not reduce following exercise over the 5 to 30 minute post exercise-recording period. There was not a significant reduction in HR in order to elicit a significant decrease in RPP. Although Forjaz noted that RPP and myocardial oxygen consumption remain elevated following a bout of exercise, we do not know if this is true of the short resting bouts of HIT.

\textbf{Arterial Stiffness and Oxygen Supply/Demand}

Aortic stiffness is an independent predictor of cardiovascular mortality, and the relationship between aortic stiffness and cardiac death may be related to an increased cardiac oxygen demand. A study by Guelen et al.\textsuperscript{104} investigated whether aortic stiffness, estimated as aortic pulse wave velocity (PWV), is associated with decreased perfusion pressure estimated as the cardiac oxygen supply potential. The DPTI and the Diastolic Time Fraction (DTF, the ratio of the diastolic time and the pulse interval) were considered estimates of the cardiac oxygen supply potential. The researchers examined 2490 older adults and related their aortic stiffness to their myocardial oxygen supply and demand. The authors noted that as aortic stiffness is increased there is an increase in the cardiac oxygen demand. On top of an increased oxygen demand on the heart, the study also demonstrated that as aortic stiffness increased the ability of the heart to supply blood decreased. It has been reported elsewhere that increasing aortic stiffness can limit exercise capacity as a result of increased energetic cost to the myocardium \textsuperscript{105}. The authors also
noted that increasing arterial stiffness could actually lead to a reduced coronary perfusion pressure however it is unlikely that, in healthy coronaries with a high functional reserve, this effect would be limiting\textsuperscript{105}.

Several mechanisms may explain the association between aortic stiffness and coronary heart disease. Arterial stiffening may lead to early pulse wave reflection causing an increase of central systolic blood pressure, a decrease of diastolic blood pressure, and a consequent increase of pulse pressure\textsuperscript{106}. The elevation of systolic blood pressure increases myocardial oxygen demand, reduces ejection fraction, and increases ventricular load, thereby inducing left ventricular hypertrophy\textsuperscript{107}. Moreover, because myocardial blood supply depends largely on pressure throughout diastole and the duration of diastole\textsuperscript{108}, the contemporary decrease of diastolic blood pressure can compromise coronary perfusion, resulting in subendocardial ischemia.

Pulse wave analysis (PWA) is an accurate time-effective measure of arterial stiffness, which measures the pulse wave at a peripheral site to estimate the regional arterial stiffness. Arterial stiffness is estimated based on the augmentation index (AIx), which is a ratio calculated from the blood pressure waveform. A probe is typically placed on the skin overlying the radial artery, and pressure is applied to distort or applanate (flatten) the artery, creating a signal, which approximates the arterial pressure\textsuperscript{109}. A reproducibility study by Wilkinson\textsuperscript{110} was performed using a total of 33 subjects (aged 24-67). Two researchers determined Alx from the dominant radial artery, twice each, in random order. The mean Alx for the study group was 19.6±12.0\% (range -15-45\%) The within-observer difference
(mean+SEM) was 0.49±0.93% and the between observer difference was
0.23±0.66%. The SD of the within-observer differences was 5.37% and the between-
observer differences were 3.80%. These results demonstrate that PWA by
applanation of the radial artery is a reproducible method for determining Alx and
thus arterial stiffness. Reference values for Alx are presented in table 1.

Table 1. Alx Reference Values

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<th>Age(Years)</th>
<th>Population Mean (%)</th>
<th>Lower 5% Confidence Interval</th>
<th>Upper 5% Confidence Interval</th>
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<td>80</td>
<td>41.53</td>
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</tbody>
</table>

Summary and Conclusions
In summary, it is well known that increased exercise intensity is associated
with increased risk of adverse cardiovascular events. However, it has also been
demonstrated that regular physical activity can reduce the risk of these adverse
events. The sport of hockey can be characterized as a game of high intensity interval
activity. This involves periods of extremely high intensity exercise in combinations
with complete bench rest. The extremely high levels of activity required during
hockey have the effect of drastically increasing both HR and systolic blood pressure,
which in turn drive the increase in myocardial oxygen demand. As a result of a
marked increase in HR, the duration of diastole during exercise is consequently
reduced leading to a reduction in oxygen supply to the myocardium. This can lead to an oxygen supply/demand mismatch and result in myocardial ischemia. Little is known about the post interval recovery phase during high intensity interval activities effect on myocardial oxygen supply and demand, especially during a game of hockey. Based on the age-related decrease in the ability of an individual’s heart rate to recover following a bout of exercise, and a severe reduction in diastolic blood pressure following exercise (PEH), it can be assumed that the recovery period following an acute interval may have a greater oxygen supply/demand mismatch that the activity itself. This can have serious implications in a game of hockey, where the rest interval involves complete bench sitting immediately following an intense bout of work. Despite these concerns however, considering the large number of middle-aged men currently participating in recreation, there are a relatively low number of adverse events. It is therefore the purpose of this study to examine the hemodynamic response to a game of hockey in middle-aged men, focusing on the intensity level in comparison to maximal exercise testing, and how 1-minute of recovery effects the variables of HR, BP and oxygen supply/demand. This can help better understand both the risks and potential protective measures associated with this type of activity.
Chapter III: Methodology

Experimental Design
The present study is designed to assess the hemodynamic responses to recreational hockey in middle-aged men. Middle-aged men are at a higher risk for adverse cardiac events during high intensity exercise. The effect of a high intensity intermittent sport, such as hockey, on middle-aged men is relatively unknown. Considering the high prevalence of hockey participation in Canada it is important to assess the cardiovascular responses to this type of activity. Middle-aged men are more prone to increased arterial stiffness, and elevated blood pressure, which can raise their risk of myocardial ischemia.

The characteristics of our participants were assessed during a preliminary visit to our laboratory prior to the field-testing. The purpose of the preliminary test was to determine descriptive information of the subjects as well as to determine vascular function and aerobic power. For the field-testing, ambulatory BP and HR monitoring was established 15 minutes prior to subjects dressing into their equipment and was recorded throughout the pre-game, game and recovery period.

Subjects
A total of 23 men aged 53±6.6 years were recruited for participation in this study. Inclusion criteria included males between the ages of 40 – 65. The only exclusion criterion was no prior history of any cardiac abnormalities. One participant only completed baseline testing and did not participate during the on-ice
testing portion of the study. All subjects were recruited from men’s recreational ‘pick up’ hockey games by postings at local Toronto rinks and word of mouth.

**Experimental Procedure**

*Preliminary Testing and Measurements*  
Participants attended a single session in the laboratory prior to any field-testing. Upon arriving to the laboratory, subjects completed a current activity questionnaire (Appendix D) and the PAR-Q+ (Appendix A). Descriptive characteristics including age (years), height (cm) and weight (kg) were recorded. Other measures are as follows.

*Vascular Stiffness and Blood Pressure*  
Participants were placed in a seated position and relaxed in a quiet room for 5 minutes. Following this, resting blood pressure was recorded using an automated sphygmomanometer (BP Tru). A total of 5 consecutive recordings over a 2-minute period were obtained, with the average recorded. Following this, participant’s arterial stiffness was measured through pulse wave analysis (Sphygmocor, AtCor Medical, Australia). Arterial stiffness was measured from non-invasive estimates of central and peripheral pulse wave analysis (PWA), using a tonometer applied against the radial artery on the right wrist. The measure of peripheral and central vascular stiffness (augmentation index, Aix) was obtained from the mean of 10 consecutive waveforms, with an operator index (reliability) of at least 90%\textsuperscript{110}.
**Aerobic Power (VO₂ max)**

VO₂ max was determined by indirect calorimetry using an incremental maximal exercise test on a cycle ergometer. A Polar Heart Rate monitor was used to monitor heart rate. The test was performed on a programmable cycle ergometer with respiratory gases analyzed by AEI Moxus Metabolic Cart (USA) and interfacing software. Participants warmed up for 5 minutes at a pace producing a heart rate of roughly 60% of their age-predicted maximum. Subjects selected their cadence based on their own comfort level (recommended between 60-80RPM). The power output began at 25Watts and increased by 50 Watts every 2 minutes until the 8th minute of the test where the power output was increased by 25Watts per minute. Subjects exercised until they reached complete exhaustion. VO₂ max was taken as the average of 15 seconds around the peak or plateau observed.

**In Game Field Testing**

For the field-testing, ambulatory monitoring was established 15 minutes prior to subjects dressing into their equipment and was recorded throughout the pre-game, game and recovery period. Electrocardiographic (ECG) data were obtained continuously throughout the time period using a Holter recorder (Holter) with data stored on flash memory for subsequent post-processing and analysis. For measures of BP, a motion-tolerant ambulatory BP monitor (Accutraker II, SunTech) was placed on the right arm. The equipment was placed and secured to allow for unrestricted movement. Resting measures were obtained in the change-room on two occasions to ensure data recording was successful. Participants engaged in their pre-game and game as usual. Due to the difficulty in acquiring accurate blood
pressure readings while on the ice the “on-ice” component of blood pressure was taken as an immediate blood pressure recording upon returning to the bench while the participant was still standing. Following the first reading, a second reading was made approximately 1-minute after the cessation of exercise. An activity log was maintained for each subject by an investigator. The player’s position (forward or defensemen) was also recorded on the activity log. Given the nature of timed ambulatory recordings, it was not possible to trigger BP recordings on demand while participants were skating; this is recognized as a limitation as their state of play may vary considerably when the recordings start. Follow the cessation of the game, data recording continued for 15 minutes while participants remain in the change-room. Participants were allowed to remove their hockey equipment during this time period. Following 15 minutes of recording at rest, all equipment and electrodes were removed.

**Measures**

Data for heart rate and blood pressure were temporally aligned where possible, with each discrete measure associated with the time log (pre-game to post-game). The timing of BP measurements was recorded using a data collection sheet (Appendix E).
Figure 4. Model of Data Collection. Heart rate was recorded continuously so it can be matched with any particular blood pressure reading that was taken. Considering it wasn’t feasible to measure BP while the participants were skating, the arrows that indicate HR on the diagram denote the additional HR recordings that correspond to the actual on-ice measurement for each shift in addition to the 1-minute recovery HR corresponding to that on-ice value.

Hemodynamic Response

Heart rate was recorded constantly throughout the duration of the field-testing protocol. HR was aligned with the timing of each BP reading so there were 2 pairs of HR and BP readings for each shift (immediate and 1-minute). In addition, considering the HR that was recorded in combination with the BP was taken almost 15 seconds after the participants returned to the bench, another HR reading was recorded while participants were still on the ice in order to get a true measurement of their HR while they were skating. The on-ice HR elicited higher values than the HR corresponding to the BP reading at end of shift and allowed for better
assessment of the true intensity of recreational hockey. All Holter data were analyzed with reports indicating the nature and frequency of any ectopic beats, segmental and interval disturbances in addition to the heart rate using Holter LX Analysis Software (NorthEast Monitoring, Inc.)

Blood pressure data were analyzed using the Accutraker analysis software, Ambulatory blood pressure monitoring using the Accutraker II has been previously validated. White et al. demonstrated that the Accutraker II is quite accurate compared with both the mercury column and intra-arterial methods of measuring BP. Prior to beginning the study, the Accutraker II was used to compare blood pressure results with our BPTru device, with both displaying similar values of blood pressure, showing consistency through the different recording devices.

**Myocardial VO₂**

Estimated myocardial oxygen uptake (MVO₂) was obtained through the BP and HR data recorded throughout the periods of game play, bench sitting and post game recovery. Based on the research of Baller et al. it was determined that the modified time tension index (MTTI) was the most accurate indirect method of acquiring MVO₂ during both exercise and rest. Given the nature of the sport of hockey (high intensity intermittent exercise) and likely increase in left ventricular contractility that is associated with the increased heart rate, the use of the modified time tension index appears to be the most appropriate. Considering MTI has the highest overall correlation (r=0.80) to direct measurement of MVO₂ at both normal contractility and markedly increased contractility it should serve as an appropriate
estimation of the myocardial oxygen demands of this specific activity. The formula for MTTI is as follows,

Modified time tension index (MTTI) = SBP x \sqrt{HR}.

**Myocardial Oxygen Supply**

Myocardial oxygen supply was estimated using the Diastolic Pressure Time index (DPTI). DPTI = (DBP-LV diastolic BP) x duration of diastole. Left ventricular diastolic BP was estimated at 5mmHg. Duration of diastole was determined using the ECG tracings reported by the Holter monitor (see Appendix). Duration of diastole has been demonstrated to be the time from the end of the P wave to the end of the QRS complex.\(^{112}\) This was determined by an investigator using calipers to measure these distances on various R-R intervals. For a given BP value, the HR corresponding to 15 seconds following the onset of inflation of the cuff (BP takes around 15 seconds to inflate) was used for analysis of diastolic duration. The diastolic duration was taken as the average of 3 separate R-R intervals to allow for any minor variation in HR. For each ECG printout there were approximately 20 R-R intervals, which would correspond to roughly 6 seconds in time. Initially, diastolic duration was measured for each R-R interval to determine whether there was a significant difference in diastole throughout the duration of the ECG printout. Data from 3 participants demonstrated consistent values (ICC=0.99); it was decided to average 3 R-R measurements to determine the duration of diastole for that particular time point.
**Endocardial Viability Ratio**

Endocardial Viability Ratio (EVR) was used to compare myocardial oxygen supply and demand. Typically, EVR = DPTI/STTI. In the present study, since we used MTTI instead of STTI EVR was calculated with the following formula;

$$EVR_{MTTI} = \frac{DPTI}{MTTI}$$

EVR’s were compared throughout the duration of the game in order to compare the differences amongst game play, bench sitting and post game recovery.

**Data Analysis**

Data for BP, HR, DTTI and EVR were analyzed separately using Repeated Measures and Post Hoc analysis to determine any significant main effects of time throughout the course of the hockey game. In addition ANOVA’s were performed on these same variables to test for significant differences between the immediate blood pressure reading and the 1-minute recovery reading throughout the game.

Individual t-tests were used to compare the maximal SBP, DBP and HR obtained during the baseline exercise test and the highest recorded values of these variables during the hockey game. Finally Pearson correlations were used to assess the relationship between AIx and $VO_{2\text{max}}$ and AIx and SBP during the maximal exercise test. All differences were deemed significant at P<0.05.
Chapter IV: Manuscript for Journal Submission

Introduction

Recent data indicate that over 500,000 men over 30 years of age regularly participate in recreational hockey across Canada. Most participants are engaged in semi-organized, recreational hockey with full equipment, without referee support, for weekly games lasting approximately 45-60 minutes. While participation in regular vigorous exercise is known to contribute to lowered risk of cardiovascular disease, there are anecdotal reports of exercise-related non-fatal adverse cardiovascular events or sudden cardiac death (SCD) during intensive exercise has led to concern about the safety of intensive exercise in general and pickup hockey in particular, in middle-aged men.

A major concern is the presence of occult coronary heart disease when myocardial oxygen supply/demand imbalance occurs during intensive exercise. As exercise intensity is increased, systolic blood pressure (SBP) increases rapidly, concurrent with a marked increase in HR which greatly increases myocardial oxygen demand. In addition, the increased heart rate during exercise decreases diastolic perfusion time, thereby reducing coronary reserve, potentially inducing a supply/demand mismatch. Notwithstanding, the cardiovascular response to pickup hockey on the general population has not been well described. Hockey is
characterized by periods of high intensity exercise interspersed with periods of complete rest. Previous reports indicate that peak, on-ice heart rates can exceed 90% of predicted maximal heart rate (HRmax), ranging between 85% and 100% of HRmax in men older than 35. These values match, but often exceed the widely accepted recommendations for physical activity intensity of 55 or 65% to 90% of an individual's age predicted HRmax. Beyond measures of HR, a more comprehensive understanding of the cardiovascular response to hockey in this age group is warranted. Therefore, the purpose of this study is to examine the hemodynamic effects of an entire game of recreational hockey in middle-aged men. We hypothesized that BP and HR values obtained during hockey would be lower than the values obtained during a maximal exercise test, blood pressure would progressively increase throughout the course of a hockey game and that there would be significant reductions in both HR and BP following 1-minute of rest. It was also hypothesized that myocardial oxygen supply/demand would be more compromised while on the bench than during actual game-play and the imbalance would be more marked towards the latter stages of the game.

Methods

Participants
A total of 23 men between the ages of 40 and 65 were recruited to participate in the study. All participants were recruited from men’s recreational ‘pick up’ hockey games by postings at local rinks and by word of mouth. All participants completed informed consent, with all experimental procedures approved by the University of Toronto’s research ethics board. All participants were then screened to
exclude a history or symptoms of cardiovascular disease or use of cardiovascular medications. Participants were excluded from participation if they indicated any instances of the aforementioned conditions.

*Baseline Resting Characteristics*

Participants attended a single session in the laboratory prior to any arena testing to determine baseline measures of height (cm) and weight (kg), resting BP and HR. Arterial stiffness was determined non-invasively using central and peripheral pulse wave analysis (PWA), using a tonometer applied against the radial artery on the right wrist (Sphygmocor, AtCor Medical, Australia). The measure of peripheral and central vascular stiffness (augmentation index, Aix) was obtained from the mean of 10 consecutive waveforms, with an operator index (reliability) of at least 90%.

*Aerobic Power (VO₂ max)*

Following baseline resting measures, VO₂ max was determined during graded exercise testing on an electronically-braked cycle ergometer. Direct gas-exchange measurements were obtained from breath-by-breath samples averaged over 15 seconds (AEI Moxus, USA). Participants warmed up for 5 minutes at a pace producing a HR (Polar 810i) of approximately 60% of their age-predicted maximum at a preferred cadence (typically 60-80RPM) starting at a power output of 25 Watts (W) with increments of 50 W every 2 minutes until the 8th minute of the test where the power output was increased by 25 W each minute. VO₂ max was defined as a plateau in oxygen consumption or less than a 150 ml/min increase despite a rise in work rate. VO₂ peak was recorded as the highest value if no plateau was observed.
Cardiovascular Monitoring During Hockey

For the arena testing, ambulatory monitoring was established 15 minutes prior to players dressing into their equipment and was recorded throughout the pre-game, game and recovery periods. Electrocardiographic (ECG) data were obtained continuously using a 3-lead Holter recorder (Holter DR200/HE, NorthEast Monitoring) with data stored on flash memory for subsequent post-processing and analysis. Blood pressure was recorded using a motion-tolerant ambulatory, automated BP monitor (Accutraker II, SunTech). This device has been validated against intra-arterial measures (White et al.111). The BP cuff was placed on the right arm, with recorder secured to allow for unrestricted movement. Resting (pre-game) measures were obtained in the change-room to ensure data recording was successful.

Participants engaged in their pre-game and game routines as usual. The “on-ice” component of BP was obtained by manually initiating recordings immediately upon returning to the bench (within 3 seconds) while participants remained on the bench in the standing position. The total time required for BP measurements were approximately 45 seconds. A second reading was completed following 60 s of rest after the cessation of exercise, in the seated position (‘Bench-Rest’). HR was determined from Holter ECG recordings, obtained following the processing of the Holter data at a later time.

An activity log that monitored the player’s position (forward, defence) and the timing of each blood pressure reading was maintained for each subject by an investigator. Following the cessation of the game, data recording continued for 15
minutes while participants remained in the change-room. Players were allowed to remove their hockey equipment during this time period. Given that the duration of shifts (each time period of on-ice play) and bench-time would vary considerably within and between study participants, data from the game were binned into 4 segments, each representing 25% of the game duration.

Data for heart rate and blood pressure were temporally aligned with each discrete measure associated with the time log (pre-game to post-game).

**Figure 1.** Model of Data Collecting
All Holter ECG data were analyzed for frequency of any ectopic beats, segmental and interval disturbances using Holter proprietary software.

The myocardial oxygen demand was estimated using the modified time tension index (MTTI)\(^{113}\) from the BP and HR data recorded throughout the periods of game play. MTTI was used over the regular TTI due to the overestimation of MVO\(_2\) that TTI yields at markedly increased contractility levels. A critical evaluation of indirect measurements of MVO\(_2\) is presented elsewhere.\(^6\) Bench sitting and post game recovery period using the following equation (1):

\[ (1) \quad MTTI = SBP \times \sqrt{HR}. \]

An estimate of myocardial oxygen demand was determined using the diastolic time tension index (DPTI), where:

\[ (2) \quad DPTI = (DBP-LV \text{ diastolic BP}) \times \text{duration of diastole}. \]

Left ventricular diastolic BP was assumed to be 5mmHg\(^{102}\). The duration of diastole was estimated using the ECG tracings from Holter ECG recordings, defined as the time interval between the end of the P wave to the end of the QRS complex, using calibrated calipers to determine the average duration of three R-R intervals during each measurement window. The reliability of these measures was determined in a blinded fashion on 7 participants (intra-subject variation) to be 99\%. For a given BP value, the HR corresponding to 15 seconds following the onset of inflation of the cuff was used for analysis of diastolic duration, to account for the cuff-inflation time. In addition, the endocardial viability ratio (EVR) was determined
as an index of myocardial oxygen supply and demand, using a modified equation (3),

\[ \text{EVR}_{\text{MTTI}} = \frac{\text{DPTI}}{\text{MTTI}} \]

where:

\[ \text{(3)} \]

We used the MTTI rather than the systolic time tension index (STTI) because during periods of increased contractility the TTI underestimates MVO\textsubscript{2}; the MTTI achieves the best overall correlation to direct measures of MVO\textsubscript{2} \((r=0.80)\) when taking into account both moderately and severely increased contractile states\textsuperscript{6}. Accordingly, the EVR was calculated using the following formula (4):

\[ \text{(4)} \]

The EVRs were compared at intervals throughout the game representing game play, bench sitting and post game recovery.

**Data Analysis**

All data are reported as means ± standard deviation (SD). Data for BP, HR, DTTI and EVR was analyzed separately using repeated measures of analysis (rANOVA) with Bonferroni post hoc analysis to identify significant main effects of time throughout the course of the hockey game. Significant differences between the immediate blood pressure reading and the 1-minute recovery reading throughout the game were tested using rANOVA. Individual t-tests were used to compare the maximal cardiovascular endpoints obtained during the baseline maximal exercise testing and the highest recorded values recorded during the hockey game. Finally, Pearson correlations were used to see examine relationships between AIx and VO\textsubscript{2}\text{max} and AIx and SBP during the maximal exercise test. All data were considered significant at \(p<0.05\).
Results

Participants

Of the 23 participants recruited, all completed the study without adverse responses. One participant only completed baseline testing and did not participate during the on-ice testing portion of the study. The mean age of the group was 53.0±6.6 with additional baseline characteristics presented in Table 1. Participants were normotensive and slightly overweight based on their body mass index. VO₂ Peak is reported since only 5 out of 22 participants achieved a plateau in oxygen consumption during the graded exercise test. The participants VO₂ Peaks fell in the 40th – 50th percentile for their age-predicted VO₂ Max values\(^{114}\). The HR Peak was slightly below age-predicted HR Max based on the formula 220-age (also compared with 208-(0.7 x age)\(^{115}\)). Based upon the RAPA self-reported data, participants were moderately active and scored an average of 5 out of 7, with most participants reporting they exercised at least 1 additional time per week on top of their normal hockey game.

Table 2. Subject characteristics following baseline assessment.

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<tr>
<td>BMI</td>
<td>26.8 ± 3.5</td>
<td>21 – 35</td>
</tr>
</tbody>
</table>
Peak values of HR, SBP and DBP achieved during hockey are presented in Table 2. In all cases, the values exceeded those observed during maximal exercise testing. Average shift duration was 2:30±30 min. Average HR (including both on-ice and 1-minute recovery values) was 137 ± 22 bpm.

### Cardiovascular Response to Hockey

<table>
<thead>
<tr>
<th>Variable</th>
<th>Maximal Exercise Test</th>
<th>On-ice</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Max HR Attained</td>
<td>163.0±11.0</td>
<td>174.8±8.9</td>
<td>0.00018</td>
</tr>
<tr>
<td>Max SBP Attained</td>
<td>172.7±30.5</td>
<td>201.6±19.9</td>
<td>0.0083</td>
</tr>
<tr>
<td>Max DBP Attained</td>
<td>88.6±10.6</td>
<td>102.2±5.7</td>
<td>0.00003</td>
</tr>
</tbody>
</table>

### Trends Throughout the Game: On-Ice and Bench Data

#### Systolic Blood Pressure:

Following an initial rise in SBP after the second shift, SBP declined significantly through the game (Figure 5).
declined progressively and significantly throughout the entire game \((p<0.05)\). In addition, the SBP during bench-rest remained elevated and was not different from values obtained on-ice recordings at any point in the game.

**Diastolic Blood Pressure**

There was a significant decrease in DBP throughout the course of the game for on-ice and bench values \((p<0.05)\)(Figure 6). However, a non-significant increasing trend for DBP was observed from the 3\textsuperscript{rd} to 4\textsuperscript{th} quartile of the game during bench-rest readings. Similar to that of SBP, on-ice and bench values were similar throughout the game.

**Heart Rate**

A progressive upward drift of HR was observed throughout the course of the game for both the on-ice and bench-recovery readings, amounting to approximately 20 b/min \((p<0.05)\)(Figure 7). HR fell significantly between the immediate and 1-minute recovery readings across all game points \((p<0.05)\).

**Diastolic Time Tension Index**

There is a significant decrease in DTTI throughout the course of the game in both the immediately following shift and 1-minute recovery readings \((p<0.05)\)(Figure 8). There is also a significantly lower DTTI during the post game readings when compared to pre-game readings. There was no significant difference in DTTI between the immediate and 1-minute recovery readings at any game point throughout the course of the game.
Endocardial Viability Ratio

There was a significant decrease in EVR during the immediately following shift reading up until the 3rd quartile of the game, which was then followed by a small non-significant increase towards the 4th and final portion of the game. EVR’s for 1-minute recovery did not change throughout the course of the game. There is a significant difference between the immediate and 1-minute recovery reading during the 3rd game point ($p<0.05$). There is also a significant decrease in EVR between the pre game reading and the post game reading (Figure 9).

Electrocardiographic Recordings:

Of the 22 participants, only two participants had abnormal Holter ECG recordings. One subject had evidence of a bundle branch block pattern at rest and exercise, while another participant had 124 supraventricular ectopic beats during the 60-minute recording period. No clinically abnormal segmental changes were observed during recording for any participants.

Discussion

To our knowledge, this is the first study that has looked at both the HR and BP responses during a game of recreational hockey in middle-aged men. There are two novel findings of our study: first, we have demonstrated that the cardiovascular demands of recreational hockey in middle-aged men can exceed those observed during graded maximal exercise testing. In addition, an upward drift in HR throughout the game during both on-ice and bench time occurred, systolic blood
pressure decreased. No difference was noted between participants playing defense or forward. Average shift time was approximately 150 seconds.

**Cardiovascular Responses of Recreational Hockey Vs. Maximal Exercise Testing**

Our hypothesis that HR and BP values obtained during a maximal exercise test would be greater than those obtained during hockey was refuted and indicate that peak values of HR and BP response during a recreational game of hockey exceed that of a maximal exercise test. These data are consistent with an earlier report\(^66\) that showed HR's during recreational hockey that were greater than the age-predicted maximal values of the majority of their participants, and well beyond target HR values recommended for intensive physical activity\(^67\). It is important to note that the recommended training intensity mentioned refers to continuous exercise and not HIIE. While there is no set criteria for determining the recommended intensity of interval exercise it is important to note that the high heart rates elicited during HIIE are offset by periods of much lower intensity. HIIE has even been effective in helping patients with coronary disease\(^116\). Moholdt et al.\(^116\) demonstrated that coronary patients improved their cardiovascular function following HIIE protocol consisting of 4-minutes at 80-95% of their \(HR_{\text{max}}\) followed by 3-minutes at 60-70% \(HR_{\text{max}}\).

While the average heart rate, when combining the on-ice and 1-minute recovery values, was only 137 ± 22 (well within the recommended training level), the importance of the on-ice values should be stressed. The high HRs obtained during recreational hockey are clinically relevant, since most individuals who
undergo an exercise stress test are typically exercised until they reach a HR of approximately 80-85% of their age predicted maximal value. This brings concern as to whether this form of testing is truly assessing the nature of what an individual may experience while participating in recreational hockey. In addition, the greater SBP that was observed during hockey will contribute to an increased myocardial oxygen cost. These data suggest that graded exercise testing may pose a significantly lower cardiovascular challenge, particularly myocardial oxygen demand, compared to recreational hockey, and therefore standard clinical exercise testing may underrepresent the ceiling for both HR and BP expected during this form of exercise.

**Cardiovascular Responses During the Game:**

In agreement with our hypothesis, we observed a progressive rise in HR throughout the game, similar to the phenomenon known as cardiovascular drift seen during prolonged continuous exercise. While it was not feasible to quantify fluid-loss or biomarkers during our study, it is likely that a combination of dehydration and elevated catecholamines during exercise contributed to the upwards drift in HR. In addition, a progressive increase in skin bloodflow associated with rising core temperature may further reduce central venous pressure, lowering SV, contributing to cardiovascular drift. While no research has been done on the thermal load of hockey players while wearing full protective gear, some research has been done on football players. Armstrong et al. compared the effect of exercise on 3 different groups, complete equipment (FULL), partial equipment (PART) and no equipment (CON). The study concluded the FULL and
PART conditions resulted in greater physiologic strain than the CON condition. These findings indicated that critical internal temperature and hypotension were concurrent with exhaustion during uncompensable (FULL) or nearly uncompensable (PART) heat stress and that anthropomorphic characteristics influenced heat storage and exercise time to exhaustion.

Contrary to our hypothesis, throughout the course of the game, study participants demonstrated a progressive decrease in both systolic and diastolic blood pressure. After an initial upswing SBP (Figure 5), demonstrated a continuous and significant reduction. This reduction in blood pressure may be secondary to a reduction in total peripheral resistance (TPR) \(^{119}\).

Contrary to the SBP response and in agreement with our hypothesis, there was an average decrease in HR of 20bpm with 1 minute of bench rest. During clinical exercise testing, a failure to reduce HR less than 12 beats per minute following 1-minute of exercise is considered abnormal\(^{120}\) and can be a predictor of mortality\(^{32}\).

**Myocardial Oxygen Supply/Demand**

Our results indicate that myocardial oxygen supply (as noted through the DTTI) during hockey significantly decreased throughout the course of the game, most likely as a result of reductions in blood pressure. This was offset by an increase in myocardial demand (as noted through a decreased MTTI). As expected, EVRs during gameplay are significantly lower than pre-game or post-game levels (Figure 9). Hoffman et al.\(^{7}\) demonstrated that the DPTI:STTI ratio can predict periods of ischemia and is proportional to the endocardial:epicardial flow ratio, with values
below 0.7 reflecting subendocardial ischemia\(^{121, 122}\). Other researchers have found the critical value of DPTI:STTI ratio to be as low as 0.45 to 0.6\(^{121, 123}\), suggesting that a much larger supply/demand mismatch is required in order to have myocardial ischemia. DPTI values in the present study decreased significantly throughout the course of the game, possibly due to the progressive reduction in DBP throughout the game. Conversely, the STTI was preserved, negating this effect and preserving the EVR. Ironically, the bench-rest time period offered no ‘recovery’ in the EVR throughout the game, suggesting that myocardial oxygen demand remains equally high during bench rest, and in potential cases where occult coronary disease with flow-limiting coronary reserve is present, there is no opportunity during recreational hockey to restore myocardial perfusion.

Holter ECG recordings were unremarkable for either playing time or bench-rest. For the 2 participants that reported abnormal ECG findings there was no relationship with an inability to reduce HR following 1-minute and myocardial supply/demand was no more compromised for those individuals. For all participants, only one participant had more than 1% of their total beats determined as ectopic. While this does not confirm an absence of coronary disease, BP and HR responses were appropriate and suggest a well-compensated response to increasingly strenuous exercise and rapid cessation from exercise.

**Limitations**

Our study has limitations. It is possible that our exercise test on the cycle ergometer may have underestimated true VO\(_2\) max values. Leg fatigue was reported as a primary reason for cessation of the test on a number of occasions. It is possible
that treadmill VO₂ max testing would have led to higher recorded values for our subjects, however it is unlikely that this would have changed the significance in our results. True ambulatory BP measures during the participant’s hockey shift were attempted during pilot testing, but both limitations of automated timing and the obstructive nature of cuff inflation during a shift, precluded its use. However, standing BP recordings were initiated within 3 seconds of participant returned to the bench, taken in the standing position and while maintaining foot motion to minimize orthostatic-related BP reductions. We measured diastolic duration as the duration between the end of the QRS complex and the end of the P wave using calibrated calipers. We acknowledge that the estimation of myocardial oxygen supply/demand by this method is limited, particularly without heart sounds to confirm mitral valve closure.

Conclusions

In conclusion, recreational hockey in middle-aged men elicits high HR and BP responses that can meet or exceed values obtained during maximal graded exercise testing. Throughout the game, on-ice heart rates drift upwards significantly and SBP decrease, paralleling the bench-recovery pattern. While these results indicate that recreational hockey in this age group becomes increasingly demanding on the cardiovascular system, local myocardial supply and demand is well regulated, and is associated with unremarkable ECG responses. Caution is recommended when using standard clinical exercise testing to establish an upper-limit to gauge an individual’s ability to safely participate in this activity.
**Figure 5.** Averaged systolic blood pressures throughout the duration of a hockey game. *Significantly lower than 1st, 2nd and 3rd game point (Immediate BP). # Significantly lower than 1st and 2nd game point (Immediate BP). + Significantly lower than 2nd game point (Recovery BP). No significant difference between immediate and 1-minute recovery over any time point *(p<0.05)*

**Figure 6.** Averaged diastolic blood pressures throughout the duration of a hockey game. *Significantly lower than 1st, 2nd and 3rd game point (Immediate BP). + Significantly lower than 1st and 2nd game point (Immediate BP). # Significantly lower than 1st game point (Recovery BP). No significant difference between immediate and 1-minute recovery over any time point *(p<0.05)*
Figure 7. Averaged heart rates throughout the duration of a hockey game. * Significantly higher than 1st game point. Significant difference between On-ice and 1-minute Recovery during all game points ($p<0.05$)

Figure 8. Averaged diastolic pressure time index’s throughout the duration of a hockey game. * Significantly lower than 1st game point. No significant difference between On-ice and 1-minute Recovery during any game points ($p<0.05$)
Figure 9. Averaged endocardial Viability ratio’s throughout the duration of a hockey game. * Significantly lower than 1<sup>st</sup> and 2<sup>nd</sup> game point. Significant difference between Immediate and 1-minute Recovery at the 3<sup>rd</sup> 25% of the game (p<0.05). # Significant Difference between Pre Game and Post Game.
Chapter V: General Discussion, Future Direction and Conclusions

Participants

Recruitment for the study was straightforward and no difficulty was experienced. Most of the participants were recruited through word of mouth once a player on their team had already participated. An article on this study was written for the Toronto Star and this generated a great deal of interest from the Toronto population. In order to increase our sample size, we increased the initial maximal age requirement from 60 to 65; this allowed a number of other participants to be included. Contrary to what was expected, our participants were moderately active throughout a given week.

Data Analysis, Limitations, and Future Directions

The assessment of myocardial oxygen supply and demand by indirect measures is not a widely recognized methodology. Currently, positron emission tomography (PET) using C-acetate is the most accurate and commonly used method of measure $MVO_2$ noninvasively\textsuperscript{124}. The use of DPTI/MTTI ratio in order to assess supply and demand characteristics has only mildly, but successfully been validated\textsuperscript{6}. As a result, our use of EVR as calculated through DPTI and MTTI is a novel approach in determining myocardial oxygen supply and demand. In order to assess the effect of human error a reliability study was performed. Using 3 different participants a particular ECG tracing was selected for each individual. The researcher was blinded to each tracing and was required to measure the duration of diastole on a number of
different occasions. The researcher proved to have an intraclass coefficient of 99.5%.

Pre-game systolic and diastolic BP’s were higher than our participants resting values that were recorded during their baseline visit. These results are similar to that observed by Atwal and colleagues prior to their hockey testing as well. The minor activity of putting on equipment (tying skates) might have elevated resting blood pressure.

While significant differences were observed between the maximal values of HR and BP on the ice and during the maximal exercise test, it is possible that the graded test could have underestimated our subject’s true maximal values. A number of our participants did not reach a plateau in either VO₂ or HR, and often complained of leg fatigue as opposed to cardiovascular fatigue. This is consistent with work done by Mckay, who reported >7% greater maximal oxygen consumption following a treadmill exercise test versus a cycle test. That being said, our subjects average maximal RER were above the standard criteria of 1.15 for determining VO₂ max. Although this may diminish the amount of difference between on-ice and exercise test values, the HR’s that are elicited during a hockey game still reach or exceed age-predicted maximums.

The largest limitation to the current study was the inability to measure blood pressure while players were on the ice. This is recognized as an equipment limitation, and it is believed that such readings may be possible with more sophisticated ambulatory blood pressure monitors. This should be considered in future research.
When examining the effect of HR throughout the duration of a recreational hockey game it appears that cardiovascular drift is occurring. Our participants HR were continuously rising throughout the course of the game. Work done by Coyle\textsuperscript{12} demonstrates that cardiac drift can occur as a result of dehydration. Logan-Sprenger et al.\textsuperscript{127} also demonstrated that dehydration can cause increases in HR following progressive cycling. Future research on dehydrations effect during recreational hockey is warranted. It would be valuable to monitor individual's fluid intake during the game to assess the impact that possible dehydration may play on the cardiovascular response to this activity.

While Alx was included in the data acquisition period in order to assess the effects of arterial stiffness on our participants this was omitted from the manuscript. Our participants Alx values were well within the normative range for their particular age. A simple Pearson correlation was run to determine the effect of arterial stiffness on aerobic fitness (assessed by VO\textsubscript{2 peak}). The results indicated a moderate negative correlation ($r=0.33$), indicating stiffer arteries were associated with lower aerobic fitness. A positive correlation ($r=0.44$) was also noted between Alx and maximal SBP indicating that lower arterial stiffness in individuals would induce a more blunted systolic response to maximal exercise testing. A weak negative correlation ($r=-0.14$) was also observed between Alx and average EVR. A strong correlation ($r=0.49$) was also observed between Alx and pulse pressure indicating that higher values of Alx would elicit larger pulse pressures, which could suggest a more susceptible heart.
As mentioned in the literature review the effects of body temperature can play a role in PEH. While recording core body temperature was initially attempted on participants early on in the study due to the difficulty in acquiring this data, it was not continued. In order for this to be collected, more than one researcher would have been required in order to help obtain the measurements. In addition to core temperature, future studies should examine the ambient temperature in the arena, for this may also play a role in cardiac drift or PEH.

Conclusions

In conclusion, this was the first study to look at both HR and BP responses in a game of recreational hockey in middle-aged men. Our hypothesis that assumed that the intensity of recreational hockey in terms of HR and BP would exceed the recommended training values, as described by ACSM was correct. Our second hypothesis stating that both HR and BP would continuously rise throughout the course of the game was only partially correct. HR significantly increased throughout the game, while BP significantly decreased. Our hypothesis that predicted that there would be significant reductions in BP and HR following 1-minute of recovery after each shift should also only be partially accepted. BP did not reduce significantly at any time point following recovery after individuals had completed a shift. However there were significant reductions in HR following 1-minute recovery after each shift. Our final hypothesis also was correct in stating that the myocardial oxygen supply/demand ratio (as depicted by EVR) would be significantly lower during and following the game when compared with pre gam resting levels. However EVRs remained relatively stable as opposed to the progressive reduction that was
proposed. Hockey appears to an extremely vigorous form of activity for all variables were significantly higher during hockey than those obtained from the maximal exercise test.
References

2. Gardiner M, Leather R, Teo K. Prevention of sudden cardiac death from ventricular arrhythmia. 1999
11. Laughlin MH. Cardiovascular response to exercise. Am J Physiol. 1999;277:S244-259


28. Lind L, Andren B. Heart rate recovery after exercise is related to the insulin resistance syndrome and heart rate variability in elderly men. *Am Heart J.* 2002;144:666-672


68. Madean E. A theoretical review of the physiological demands of ice hockey and a full year periodized sport specific conditioning program for the Canadian junior hockey player.


76. Goodman JM, Thomas SG, Burr J. Evidence-based risk assessment and recommendations for exercise testing and physical activity clearance in apparently healthy individuals (1) (1) This paper is one of a selection of papers published in this Special Issue, entitled Evidence-based risk assessment and recommendations for physical activity clearance, and has undergone the Journal’s usual peer review process. *Appl Physiol Nutr Metab*. 2011;36 Suppl 1:S14-32


96. Westerhof N, Boer C, Lamberts RR, Sipkema P. Cross-talk between cardiac muscle and coronary vasculature. Physiol Rev. 2006;86:1263-1308


125. McKay GA, Banister EW. A comparison of maximum oxygen uptake determination by bicycle ergometry at various pedaling frequencies and by


Appendix A: Par-Q+

PAR-Q+

6. Do you have a Respiratory Disease? This includes Chronic Obstructive Pulmonary Disease, Asthma, Pulmonary High Blood Pressure
   If the above condition(s) is/are present, answer questions 6a-6d
   If NO go to question 7
   6a. Do you have difficulty controlling your condition with medications or other physician-prescribed therapies?
      (Answer NO if you are not currently taking medications or other treatments)
      YES  NO
   6b. Has your doctor ever said your blood oxygen level is low at rest or during exercise and/or that you require supplemental oxygen therapy?
      YES  NO
   6c. If asthmatic, do you currently have symptoms of chest tightness, wheezing, laboured breathing, consistent cough (more than 2 days/weeks) or have you used your rescue medication more than twice in the last week?
      YES  NO
   6d. Has your doctor ever said you have high blood pressure in the blood vessels of your lungs?
      YES  NO

7. Do you have a Spinal Cord Injury? This includes Tetraplegia and Paraplegia
   If the above condition(s) is/are present, answer questions 7a-7c
   If NO go to question 8
   7a. Do you have difficulty controlling your condition with medications or other physician-prescribed therapies?
      (Answer NO if you are not currently taking medications or other treatments)
      YES  NO
   7b. Do you currently exhibit low resting blood pressure significant enough to cause dizziness, light-headedness, and/or fainting?
      YES  NO
   7c. Has your physician indicated that you exhibit sudden bouts of high blood pressure (known as Autonomic Dysreflexia)?
      YES  NO

8. Have you had a Stroke? This includes Transient ischemic Attack (TIA) or Cerebrovascular Event
   If the above condition(s) is/are present, answer questions 8a-8c
   If NO go to question 9
   8a. Do you have difficulty controlling your condition with medications or other physician-prescribed therapies?
      (Answer NO if you are not currently taking medications or other treatments)
      YES  NO
   8b. Have you experienced a stroke or impairment in walking or mobility?
      YES  NO
   8c. Have you experienced a stroke or impairment in nerves or muscles in the past 6 months?
      YES  NO

9. Do you have any other medical condition not listed above or do you have two or more medical conditions?
   If you have other medical conditions, answer questions 9a-9c
   If NO read the Page 4 recommendations
   9a. Have you experienced a blackout, fainted, or lost consciousness as a result of a head injury within the last 12 months or have you had a diagnosed concussion within the last 12 months?
      YES  NO
   9b. Do you have a medical condition that is not listed (such as epilepsy, neurological conditions, kidney problems)?
      YES  NO
   9c. Do you currently live with two or more medical conditions?
      YES  NO

GO to Page 4 for recommendations about your current medical condition(s) and sign the PARTICIPANT DECLARATION.
PAR-Q+

The Physical Activity Readiness Questionnaire for Everyone

Regular physical activity is fun and healthy, and more people should become more physically active every day of the week. Being more physically active is very safe for MOST people. This questionnaire will tell you whether it is necessary for you to seek further advice from your doctor OR a qualified exercise professional before becoming more physically active.

GENERAL HEALTH QUESTIONS

Please read the 7 questions below carefully and answer each one honestly: check YES or NO.

<table>
<thead>
<tr>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Has your doctor ever said that you have a heart condition OR high blood pressure?</td>
<td>☐ ☐</td>
</tr>
<tr>
<td>2) Do you feel pain in your chest at rest, during your daily activities of living, OR when you do physical activity?</td>
<td>☐ ☐</td>
</tr>
<tr>
<td>3) Do you lose balance because of dizziness OR have you lost consciousness in the last 12 months? Please answer NO if your dizziness was associated with over-breathing (including during vigorous exercise).</td>
<td>☐ ☐</td>
</tr>
<tr>
<td>4) Have you ever been diagnosed with another chronic medical condition (other than heart disease or high blood pressure)?</td>
<td>☐ ☐</td>
</tr>
<tr>
<td>5) Are you currently taking prescribed medications for a chronic medical condition?</td>
<td>☐ ☐</td>
</tr>
<tr>
<td>6) Do you have a bone or joint problem that could be made worse by becoming more physically active? Please answer NO if you had a joint problem in the past, but it does not limit your current ability to be physically active. For example, knee, ankle, shoulder or other.</td>
<td>☐ ☐</td>
</tr>
<tr>
<td>7) Has your doctor ever said that you should only do medically supervised physical activity?</td>
<td>☐ ☐</td>
</tr>
</tbody>
</table>

If you answered NO to all of the questions above, you are cleared for physical activity. Go to Page 4 to sign the PARTICIPANT DECLARATION. You do not need to complete Pages 2 and 3.

- Start becoming much more physically active – start slowly and build up gradually.
- Follow Canada's Physical Activity Guidelines for your age (www.csep.ca/guidelines).
- You may take part in a health and fitness appraisal.
- If you have any further questions, contact a qualified exercise professional such as a Canadian Society for Exercise Physiology - Certified Exercise Physiologist® (CSEP-CEP) or a CSEP Certified Personal Trainer® (CSEP-CPT).
- If you are over the age of 45 yr and NOT accustomed to regular vigorous to maximal effort exercise, consult a qualified exercise professional (CSEP-CEP) before engaging in this intensity of activity.

If you answered YES to one or more of the questions above, COMPLETE PAGES 2 AND 3.

Delay becoming more active if:

- You are not feeling well because of a temporary illness such as a cold or fever - wait until you feel better
- You are pregnant - talk to your health care practitioner, your physician, a qualified exercise professional, and/or complete the ePARmed-X at www.eparmedx.com before becoming more physically active
- Your health changes - answer the questions on Pages 2 and 3 of this document and/or talk to your doctor or qualified exercise professional (CSEP-CEP or CSEP-CPT) before continuing with any physical activity program.
PAR-Q+

If you answered NO to all of the follow-up questions about your medical condition, you are ready to become more physically active - sign the PARTICIPANT DECLARATION below:

1. It is advised that you consult a qualified exercise professional (e.g., a CSEP-CEP or CSEP-CPT) to help you develop a safe and effective physical activity plan to meet your health needs.
2. You are encouraged to start slowly and build up gradually - 20-60 min of low to moderate intensity exercise, 3-5 days per week including aerobic and muscle strengthening exercises.
3. As you progress, you should aim to accumulate 150 minutes or more of moderate intensity physical activity per week.
4. If you are over the age of 45 yr and NOT accustomed to regular vigorous to maximal effort exercise, consult a qualified exercise professional (CSEP-CEP) before engaging in this intensity of activity.

If you answered YES to one or more of the follow-up questions about your medical condition:

You should seek further information before becoming more physically active or engaging in a fitness appraisal. You should complete the ePARmed-X+ at www.eparmed.com and/or visit a qualified exercise professional (CSEP-CEP) to work through the ePARmed-X+ and for further information.

Delay becoming more active if:

1. You are not feeling well because of a temporary illness such as a cold or fever - wait until you feel better
2. You are pregnant - talk to your health care practitioner, your physician, a qualified exercise professional, and/or complete the ePARmed-X+ at www.eparmed.com before becoming more physically active
3. Your health changes - talk to your doctor or qualified exercise professional (CSEP-CEP) before continuing with any physical activity program.

You are encouraged to photocopy the PAR-Q+. You must use the entire questionnaire and NO changes are permitted.

The PAR-Q+ Collaboration, the Canadian Society for Exercise Physiology, and their agents assume no liability for persons who undertake physical activity. If in doubt after completing the questionnaire, consult your doctor prior to physical activity.

PARTICIPANT DECLARATION

I, the undersigned, have read, understood to my full satisfaction and completed this questionnaire. I acknowledge that this physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if my condition changes. I also acknowledge that a Trustee (such as my employer, community/fitness centre, health care provider, or other designate) may retain a copy of this form for their records. In these instances, the Trustee will be required to adhere to local, national, and international guidelines regarding the storage of personal health information ensuring that they maintain the privacy of the information and do not misuse or wrongfully disclose such information.

NAME

DATE

SIGNATURE

WITNESS

SIGNATURE OF PARENT/GUARDIAN/CARE PROVIDER

For more information, please contact www.eparmed.com or
Canadian Society for Exercise Physiology www.csep.ca

ThePAR-Q+was created using the evidence-based AGREEX process by the PAR-Q+ Collaboration chaired by Dr. Zinner E., R. Workman with Dr. Norman Gelboin, Dr. Jeronim Jaremik, and Dr. Donald C. McKenderreid. Production of this document has been made possible through financial contributions from the Public Health Agency of Canada and the BC Ministry of Health Services. The views expressed herein do not necessarily represent the views of the Public Health Agency of Canada or BC Ministry of Health Services.

Key References:
1. Do you have Arthritis, Osteoporosis, or Back Problems?
   If the above condition(s) is/are present, answer questions 1a-1c
   (Answer NO if you are not currently taking medications or other treatments)
   1a. Do you have difficulty controlling your condition with medications or other physician-prescribed therapies?
   YES NO
   1b. Do you have joint problems causing pain, a recent fracture or fracture caused by osteoporosis or cancer, displaced vertebra (e.g., spondylolisthesis), and/or spondylolysis/pars defect or a crack in the bony ring on the back of the spinal column?
   YES NO
   1c. Have you had steroid injections or taken steroid tablets regularly for more than 3 months?
   YES NO

2. Do you have Cancer of any kind?
   If the above condition(s) is/are present, answer questions 2a-2b
   (Answer NO if you are not currently taking medications or other treatments)
   2a. Does your cancer diagnosis include any of the following types: lung/bronchogenic, multiple myeloma (cancer of plasma cells), head, and neck?
   YES NO
   2b. Are you currently receiving cancer therapy (such as chemotherapy or radiotherapy)?
   YES NO

3. Do you have Heart Disease or Cardiovascular Disease? This includes Coronary Artery Disease, High Blood Pressure, Heart Failure, Diagnosed Abnormality of Heart Rhythm
   If the above condition(s) is/are present, answer questions 3a-3e
   3a. Do you have difficulty controlling your condition with medications or other physician-prescribed therapies?
   YES NO
   3b. Do you have an irregular heart beat that requires medical management?
   (e.g., atrial fibrillation, premature ventricular contraction)
   YES NO
   3c. Do you have chronic heart failure?
   YES NO
   3d. Do you have a resting blood pressure equal to or greater than 160/90 mmHg with or without medication?
   (Answer YES if you do not know your resting blood pressure)
   YES NO
   3e. Do you have diagnosed coronary artery (cardiovascular) disease and have not participated in regular physical activity in the last 2 months?
   YES NO

4. Do you have any Metabolic Conditions? This includes Type 1 Diabetes, Type 2 Diabetes, Pre-Diabetes
   If the above condition(s) is/are present, answer questions 4a-4c
   4a. Is your blood sugar often above 13.0 mmol/L? (Answer YES if you are not sure)
   YES NO
   4b. Do you have any signs or symptoms of diabetes complications such as heart or vascular disease and/or complications affecting your eyes, kidneys, and the sensation in your toes and feet?
   YES NO
   4c. Do you have other metabolic conditions (such as thyroid disorders, pregnancy-related diabetes, chronic kidney disease, liver problems)?
   YES NO

5. Do you have any Mental Health Problems or Learning Difficulties? This includes Alzheimer's, Dementia, Depression, Anxiety Disorder, Eating Disorder, Psychotic Disorder, Intellectual Disability, Down Syndrome
   If the above condition(s) is/are present, answer questions 5a-5b
   5a. Do you have difficulty controlling your condition with medications or other physician-prescribed therapies?
   YES NO
   5b. Do you ALSO have back problems affecting nerves or muscles?
   YES NO
Appendix B: Study Information and Consent Form

UNIVERSITY OF TORONTO
Faculty of Physical Education and Health

Study Information for the Participant

Investigating the Hemodynamic Responses to a Game of Recreational Hockey in Middle-Aged Men

Principle Investigator:
Dr. Scott Thomas, Associate Dean, Department of Exercises Science, University of Toronto

Co-Investigators:
Dr. Jack Goodman, Associate Professor, Department of Exercise Science, University of Toronto

Student Investigator
Zack Goodman, Master Candidate, Department of Exercise Science, University of Toronto,

Background Information
Over 500,000 male adults in Canada play recreational “pick up” and organized hockey. The objectives of this study is to determine the cardiovascular consequences of a game of pick up hockey in middle-aged men and identify the potential risks that may contribute to adverse cardiovascular events. The novel aspect of this study is both blood pressure and ECG recordings will be obtained during play, rest and recovery, providing estimates of the cardiac oxygen costs, and the interaction between intensive activity interspersed with periods of sitting. This information will provide insights of the cardiovascular consequences of hockey and possibly strategies to minimize the risk of adverse, catastrophic events.

Purpose
You are invited to participate in a study that will examine the cardiovascular effects of a game of recreational hockey. Due to the intense nature of the sport, it is not uncommon to see heart rates approach near maximal values. This in combination with increased blood pressure can lead to increased work place on the heart. Following a players shift, upon returning to the bench, as a result of a unique post exercise physiological mechanism called post exercise hypotension (PEH) there is marked reduction in blood pressure. This reduction in blood pressure along with an elevated heart rate has the effect of decreasing the potential supply of oxygen to the heart. The purpose of this study is therefore to determine what are the cardiovascular consequences of both game-play and bench rest in middle age recreationally active males.

Procedures
A week before the actual in game measurements, you will be required to undergo a baseline test at the University of Toronto. First, resting blood pressure will be recorded. You will be required to abstain from caffeine 8 hours and vigorous physical activity 48 hours before the measurement. The blood pressure measurement will be taken after you have been seated for 10 minutes. The blood pressure machine will take a measurement every 2 minutes for a total of 5 times, you blood pressure is the average of the 5 measurements.

Following your blood pressure measurement your arterial stiffness will then be recorded. Arterial stiffness will be measured with pencil-like device that will be gently pressed over an artery at the wrist.

The final preliminary test will the exercise test (VO$_{2\text{max}}$ test) which tests your maximum exercise capacity and it will take 15-20 minutes. The test will be carried out on a treadmill with increased exercise intensity every minute until exhaustion.

For the field testing, ambulatory monitoring will be established 15 minutes prior to you dressing into your equipment and will be recorded throughout the pre-game, game and recovery period. Electrocardiographic (ECG) data will be obtained continuously throughout the time period using a Holter recorder (Holter). For measures of BP, a motion-tolerant ambulatory BP monitor will be placed on the left arm. The equipment will be placed and secured to allow for unrestricted movement. During the game, the blood pressure cuff will be set to record on a 3-minute interval. When the cuff begins to inflate you will feel tension on your left arm. Once you feel this tension you are to drop your arm to the side of your body in order to obtain a more accurate reading of your blood pressure. When you return to the bench after each of your shifts, the researcher will take a manual recording of your blood pressure, he will repeat this measure after 2 minutes of rest.

Following the game, the equipment will be left on for a total of 30 minutes in order to obtain appropriate recovery information. Following this period you are free to leave.

**Risks and Benefits**

All of the tests at the University of Toronto are routinely performed and are associated with minimal risk. More specifically, the intensity of the exercise performed is commonly prescribed and recommended for obtaining benefits from physical activity. The risk of cardiac events associated with the exercise stress test is less than 1 in 10,000 people. It is possible that you will have sore legs after exercise or the following day. There are no known risks associated with the non-invasive measurements involved in this study.

In terms of the in-game field-testing, there is nothing that you are being asked to do physically outside what your normal routine is.

**Confidentiality and Privacy of Information**

Your personal information including the results of the above tests will be kept strictly confidential. All results will be coded with a study ID number. The document connecting your name to your identification number will be kept in a locked filing cabinet in the primary investigator's office. All study data inputted into a computer will be password protected and access will be limited to study investigators only.

**Voluntary Participation**

Your participation in this study is completely voluntary. Your decision to participate in this study (or not), will in no way affect your membership or status at the University of Toronto. Similarly, you are free to withdraw from the study at anytime.
Compensation
You will be offered compensation for transportation or parking.

Contacts and Questions
If you have any questions please contact: Mr. Zack Goodman, phone: 647-458-3451 or email zack.goodman@mail.utoronto.ca Or Dr. Scott Thomas, Phone: 416-978-6957, email scott.thomas@utoronto.ca If you have questions about your rights as a research participant, please contact Jill Parsons, Health Sciences Ethics Review Officer, Ethics Review Office, University of Toronto, at 416.946.5806 or email: jc.parsons@utoronto.ca
Investigating the Hemodynamic Responses to a Game of Recreational Hockey in Middle-Aged Men

Participant’s Informed consent to participate

I, _________________________________, agree to participate in this study, (Print participant’s first and last name)

Conducted by the University of Toronto, which will provide knowledge about the cardiovascular consequences of recreational hockey. I understand the above information and have had an opportunity to discuss any questions or concerns with the primary, or student investigator. As a participant in this study I will receive a copy of this signed informed consent form. As a participant I understand that the investigators will be available throughout this study to answer any question that may arise.

I voluntarily consent to participate in this study.

Participant’s signature: _____________________________ Date: ________________

Witness name (print): _____________________________ Date: ________________
Witness signature: _____________________________ Date: ________________

Investigator name: _____________________________ Date: ________________
Investigator signature: _____________________________ Date: ________________
Appendix C: Recruitment Poster

THE HEART OF HOCKEY
A Study of the Cardiovascular Consequences of Hockey

STUDY PURPOSE
Our study is attempting to determine the cardiovascular consequences of recreational pick-up hockey in middle-aged men. The risks associated with exercise are increased with advancing age in addition to increasing exercise intensity. Little is known about the cardiovascular response to hockey. Considering the intense nature of hockey it is important to determine what exactly is going on with individuals heart rates and blood pressure during a game of pick-up hockey. It is also important to examine whether an individuals fitness level plays a role in their response to

Participant Requirements
• Men currently involved in a weekly, recreational game of pick-up hockey
• Aged 40-60 years
• Free from any history of cardiovascular disease

Benefits for the participants
• Learn your aerobic fitness level
• Gain knowledge on what is happening to you during your weekly game of pick-up hockey

Study Requirements
• Time commitment in order to come in for baseline laboratory assessment (1x, approximately 1.5 hours)
• Maximal exercise testing (VO2max)
• Non-invasive assessment of vascular stiffness and blood pressure
• Ability to arrive at hockey arena 40 minutes prior to your scheduled time of play

Please Contact
zack.goodman@mail.utoronto.ca
Cardiovascular Regulation Laboratory: 416-978-0762
How Physically Active Are You?

An assessment of level and intensity of physical activity
<table>
<thead>
<tr>
<th></th>
<th>How physically active are you? (Check one answer on each line)</th>
<th>Does this accurately describe you?</th>
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<tbody>
<tr>
<td>1</td>
<td>I rarely or never do any physical activities.</td>
<td>Yes □ No □</td>
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<tr>
<td>2</td>
<td>I do some <strong>light</strong> or <strong>moderate</strong> physical activities, but not every week.</td>
<td>Yes □ No □</td>
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<tr>
<td>3</td>
<td>I do some <strong>light</strong> physical activity every week.</td>
<td>Yes □ No □</td>
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<tr>
<td>4</td>
<td>I do <strong>moderate</strong> physical activities every week, but less than 30 minutes a day or 5 days a week.</td>
<td>Yes □ No □</td>
</tr>
<tr>
<td>5</td>
<td>I do <strong>vigorous</strong> physical activities every week, but less than 20 minutes a day or 3 days a week.</td>
<td>Yes □ No □</td>
</tr>
<tr>
<td>6</td>
<td>I do 30 minutes or more a day of <strong>moderate</strong> physical activities, 5 or more days a week.</td>
<td>Yes □ No □</td>
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<tr>
<td>7</td>
<td>I do 20 minutes or more a day of <strong>vigorous</strong> physical activities, 3 or more days a week.</td>
<td>Yes □ No □</td>
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<tr>
<td>8</td>
<td>I do activities to increase muscle <strong>strength</strong>, such as lifting weights or calisthenics, once a week or more.</td>
<td>Yes □ No □</td>
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<td>9</td>
<td>I do activities to improve <strong>flexibility</strong>, such as stretching or yoga, once a week or more.</td>
<td>Yes □ No □</td>
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ID # ____________________________
Today’s Date ____________________
Scoring Instructions

RAPA 1: Aerobic

To score, choose the question with the highest score with an affirmative response. Any number less than 0 is suboptimal.

For scoring or summarizing categorically:

Score as sedentary:

1. I rarely or never do any physical activities.

Score as under-active:

2. I do some light or moderate physical activities, but not every week.

Score as under-active regular - light activities:

3. I do some light physical activity every week.

Score as under-active regular:

4. I do moderate physical activities every week, but less than 30 minutes a day or 6 days a week.

5. I do vigorous physical activities every week, but less than 20 minutes a day or 3 days a week.

Score as active:

6. I do 30 minutes or more a day of moderate physical activities, 5 or more days a week.

7. I do 20 minutes or more a day of vigorous physical activities, 3 or more days a week.

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RAPA 2: Strength & Flexibility

I do activities to increase muscle strength, such as lifting weights or calisthenics, once a week or more. (1)

I do activities to improve flexibility, such as stretching or yoga, once a week or more. (2)

Both. (3)

None (0)
# Appendix E: Data Collection Sheet

Subject Name:_____________________

<table>
<thead>
<tr>
<th>Time</th>
<th>BP Reading</th>
<th>RPE</th>
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Figure 2 was taken from:

Duncker DJ, Bache RJ. Regulation of coronary blood flow during exercise. Physiol Rev. 2008;88:1009-1086

Figure 3 was taken from:

Baller D, Sigmund-Duchanova H, Zipfel J, Hellige G. Prediction of myocardial blood flow by DPTI and prediction of the adequacy of myocardial O2 supply by the DPTI/STTI ratio under maximal coronary dilation. Basic Res Cardiol. 1979;74:378-388