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Quantifying changes in accelerations and heart rate indicative of fatigue during condensed competitions in elite youth ice hockey players

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Quantifying Changes in Accelerations and Heart Rate Indicative of Fatigue During Condensed

Competitions in Elite Youth Ice Hockey Players

by

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Thesis

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Abstract

Thirty-three elite youth ice hockey players wore Bioharness-3 (Zephyr, MD) sensors to compare

accelerations (ACC) and heart rate (HR) over four games (G1–G4) in three days, in order to

establish changes in cardiovascular stress and physical exertion associated with fatigue. Peak

ACC and HR across multiple time frames were quantified and analyzed in conjunction to

determine exertion profiles for each game. MANOVAs for peak ACC and HR, at each time

point across G1–G4 and multiple games per day (M1, M2) for magnitude and time as main

effects were performed. HR beats per minute decreased between G1 and G3/G4 in time segments

(3–20 minutes) although ACC were not different. Peak ACC were lower for M2 vs M1 at 60, 90,

120 and 180 seconds. Results concluded the decline in HR, but not ACC, across games indicates

a cardiovascular adaptation. The reduced ACC between games M1 and M2 indicate fatigue.

Table of Contents

List of Figures

Introduction

At the professional, collegiate and 20-and-under developmental performance levels of ice hockey, teams rarely compete on more than two consecutive days and never in multiple games per day ("2017-2018 WCHA Composite Schedule," 2018; NHL, 2018). Fatigue and its effects on player performance and safety are the major concerns. It has been established that fatigue can reduce performance and may contribute to an increased risk of injury (Luke et al., 2011; Yaggie & McGregor, 2002). Since fatigue is possibly manageable, developing strategies to reduce the effect on athletes may have a positive impact on performance and reduce injury risk (Nuno et al., 2016). Yet at the youth levels of the sport, there is little regard for the potential negatives of playing and competing on many consecutive days without a break. The general perception is typically "the more ice time, the better," without regard to athlete fatigue.

Across the USA Hockey youth development landscape, there exist a number of regional clubs that aggregate some of the nation's best youth players. These clubs begin to run national showcase events with 14-to-18-year-olds that bring teams together for weekend events, which include multiple competitions per day. The typical showcase tournament involves traveling to the competition site on a Thursday, playing one or two games on Friday, one or two games on Saturday then one game on Sunday before traveling back home. A general perception by scouts and those who regularly view these types of events is that there is a distinct drop in performance over course of the event and evaluations that take place at the end are far less valuable. Therefore, the purpose of this study is to quantify changes in peak accelerations and heart rate that would be indicative of fatigue over the course of a short-term event.

The research hypothesis states that there will be signifcant changes in peak acceleration and heart rate between games over the course of a short-term tournament event. The null

hypothesis for this project states that there will not be a significant change in peak accelerations

and heart rate between games over the course of a short-term tournament event.

Literature Review

Ice hockey is a high intensity, contact team sport, which requires both anaerobic and aerobic fitness (Cox, Miles, Verde, & Rhodes, 1995; H. J. Green, Daub, Painter, & Thomson, 1978; D. L. Montgomery, 1988; Peterson et al., 2015; Stanula, Roczniok, Maszczyk, Pietraszewski, & Zajac, 2014). It is classified as an intermittent sprint sport, punctuated by times gliding, stop-and-start accelerations, or potential engagement in some form of physical confrontation (Bracko, Fellingham, Hall, Fisher, & Cryer, 1988). Locomotion is done through skating, taking advantage of the low surface coefficient of friction on the playing surface and providing a distinctly different energy expenditure profile than walking or running (Formenti, 2014).

The professional game is broken into three 20-minute periods with an approximate 15-minute break between periods to resurface the ice (Cox et al., 1995). Coaches target on-ice shift time for players in games to be 40 to 45 seconds, but it can be up to 80 seconds or more if caught in a poor playing situation (Cox et al., 1995; D. L. Montgomery, 1988). Based upon data from the National Hockey League, seasons 2009 through 2011, the average shift (game time on ice) for a forward position player is 45.5 ± 3.9 seconds with average recovery intervals off ice lasting 73.4 ± 16.6 seconds (Peterson et al., 2015).

The physiological demands on the players can vary by position and potential rank on the team. First- and second-line athletes will typically accumulate more playing minutes than third- or fourth-line players (NHL, 2016). Players will cover an approximate range between 2,250 and 5,000 m per game with average speeds of 17 to 20 km/hr (Golich, 2014). Average sprint distances for individual players range from 13.41 m to 19.52 m (Golich, 2014). Again, player position, standing on the team, and game strategy plays a factor in this. Top speed for most elite

international players is over 30 km/h, with some players able to exceed 36 km/h (Omega Timing

Ltd., 2018). This is combined with high levels of agility and physical contact. This is an

equivalent to the current 100 m world-record running sprint speed of 10.44 m/s (International

Association of Athletic Federations).

Fatigue

Fatigue is defined as the inability to sustain the expected level of force (Hawley & Reilly,

1997). In a sport performance setting, it is the inability to maintain a desired work rate where

work rate is dictated by the immediate demands of the sport and not self-selected by the athlete

(Reilly, Drust, & Clarke, 2008).

Causes of fatigue are multifactorial, as there is no global mechanism for fatigue. Rather,

the mechanisms that cause fatigue are specific to the task being performed (Enoka & Duchateau,

2008). The development of fatigue is typically quantified as a decline in maximal force or power

capacity of muscle, which means submaximal contractions can still be sustained after onset.

(Enoka & Duchateau, 2008) This task dependency of fatigue is specific to the dominant

mechanisms that are being stressed within the exercise (Enoka & Duchateau, 2008). As ice

hockey is considered an intermittent-sprint sport requiring both aerobic and anaerobic fitness

(Cox et al., 1995; D. L. Montgomery, 1988), fatigue can and should be evaluated with respect to

specific nature of the activities involved.

Fatigue during sprint activity. Many team sports necessitate athletes to generate high-

intensity efforts over short time frames. This type of sprint activity has been defined by maximal

or near-maximal efforts with a duration of ≤ 10 seconds (D. J. Bishop, 2012). Yet the demands in

team sports can dictate varying levels of low to moderate activity levels and recovery segments

interspersed with sprints. A further distinction is made between intermittent-sprint exercise and

repeat-sprint exercise (Girard, Mendez-Villanueva, & Bishop, 2011). Intermittent-sprint exercise consists of short duration, maximal efforts ≤ 10 seconds with longer periods (60 to 300 seconds) that allow for greater recovery (Balsom, Seger, Sjodin, & Ekblom, 1992; David Bishop & Claudius, 2005; Duffield, King, & Skein, 2009) while repeat-sprint exercise consists of short duration maximal efforts ≤ 10 seconds separated by smaller duration recovery segments ≤ 60 seconds. This demonstrates a greater performance decrement and establishes the potential for causes of fatigue to be different (D. Bishop, Edge, Davis, & Goodman, 2004; Girard et al., 2011).

In the context of team sports, fatigue has been linked to a reduction in repeat-sprint capacity (Krustrup, Zebis, Jensen, & Mohr, 2010). Due to the intensities required in competition, a reduction in an athlete's sprint ability can adversely affect performance and outcomes on individual plays by limiting the athlete's ability to arrive at a desired location at the necessary time (Girard et al., 2011; McGregor, 2016).

Limiting Factors

Metabolite availability and depletion. Phosphocreatine (PCr) provides the most immediate source for phosphorylation of ATP and is rapidly depleted in repeat-sprint situations (Girard et al., 2011). Muscle PCr concentrations have been shown to drop as much as 57% over resting levels in a single 6-second maximal sprint (Gaitanos, Williams, Boobis, & Brooks, 1993). PCr does resynthesize rapidly, but can take more than 5 minutes to return to resting values (Bogdanis, Nevill, Boobis, Lakomy, & Nevill, 1995). Within the competition environment, the necessity for repeated maximal sprint efforts may not allow for adequate restoration of PCr levels and subsequent performance can be compromised (Bogdanis, Nevill, Lakomy, & Boobis, 1998; Girard et al., 2011).

It has also been noted that there are muscle fiber type differences in the phosphate utilization between Type 1 and Type 2 fibers with greater depletion in Type 2 fast-twitch fibers. (Karatzaferi, de Haan, van Mechelen, & Sargeant, 2001) Since maximal sprint capacity is reliant on fast-twitch muscle fibers, a reduction in PCr reserves may inhibit successive sprint efforts (Girard et al., 2011).

In contrast to conventional thinking, anaerobic glycolisis plays an important role in sprints as short as 6-seconds. It has been shown in that in a single 6-second sprint, roughly 40% of the total ATP provision is achieved through anaerobic glycolysis and progressively declines as sprints are repeated (D. J. Bishop, 2012; Gaitanos et al., 1993). In addition, over a 10 x 6-second repeat sprint test, there is an eightfold decrease in absolute ATP production between the first and last sprint (Gaitanos et al., 1993). This suggests that the rate of ATP provision through anaerobic glycolysis is a contributing factor to fatigue during intermittent sprint exercise (D. J. Bishop, 2012).

Although sprint efforts are generally accepted to be anaerobic in nature, in evaluating the oxidative system's input to the first and last sprints in a 5 x 6-second repeat sprint protocol, it was shown that the aerobic contribution to the first sprint is ~10% and ~40% for the fifth sprint (McGawley & Bishop, 2015). This demonstrates a substantial shift in energy system contributions as the decrease in anaerobic glycolysis is compensated by the increase in aerobic ATP provision. Significant increases in blood plasma FFA have been shown to be consistent over three periods of game play in ice hockey players, suggesting this is an important substrate in energy production (H. J. Green et al., 1978).

While there is considerable focus in the literature regarding repeat ability evaluating maximal 6-second sprint efforts or longer (Girard et al., 2011; Spencer, Bishop, Dawson, &

Goodman, 2005), analysis of team-based sports suggests that sprint efforts are of a much shorter duration (< 4 sec; Spencer et al., 2005). This would indicate a potentially even larger reliance on PCr contribution ATP provision. It has been estimated that for a 3-second sprint, which is potentially more typical in field-based team sports, the estimated energy system contributions are 10% from stored ATP, 55% from PCr, 32% from anaerobic glycolysis, and 3% from the aerobic system (Spencer et al., 2005). Yet, while the aerobic contribution to a single sprint is low, the contribution increases with repeated efforts and is influenced by sprint duration, number and length of recovery (Balsom et al., 1992; Spencer et al., 2005)

Muscle glycogen is well known to be a significant substrate during exercise, and as exercise intensity increases, there is an increased dependence on muscle glycogen (Ivy, 1991). The capacity to sustain high-intensity exercise has been associated with pre-exercise levels of muscle glycogen as reviewed in (Balsom, Gaitanos, Soderlund, & Ekblom, 1999; Ivy, 1991). In ice hockey, muscle glycogen depletion over the course of game has been reported in the range of 38% to 88% of resting values (Akermark, Jacobs, Rasmusson, & Karlsson, 1996; H. J. Green et al., 1978; D. L. Montgomery, 1988). This is of concern when consecutive competitions occur within 24 hours or < 8 hours, as insufficient restoration can effect performance (Burke, van Loon, & Hawley, 2017). It has been shown in ice hockey that players with higher muscle glycogen values in the third period retain the capacity to skate faster than players who are more glycogen depleted (Akermark et al., 1996). In other team sports such as soccer, under certain conditions with similar glycogen depletion, it has been shown to take over 48 hours to restore muscle glycogen to resting levels (Gunnarsson et al., 2013). This indicates that the timing of competitions may have an impact on fatigue and player performance by reducing the availability of muscle glycogen as a substrate source.

When looking at glycogen depletion patterns for ice hockey, specific to fiber type, Type 1 fibers show significant depletion in contrast to Type 2 fibers (H. J. Green et al., 1978). Even with differences in shift length, number of shifts per game, and blood substrates profiles, the amount and patterns of depletion were similar for forwards and defensemen (H. J. Green et al., 1978). This greater depletion in Type 1 fibers may indicate a potential greater reliance on oxidative metabolism.

Metabolite accumulation. During maximal repeat-sprint activity, PCr degradation and anaerobic glycolysis contribute the majority of initial energy to resynthesize ATP (Gaitanos et al., 1993). Anaerobic glycolysis is associated with increased acidosis in muscle (David Bishop & Claudius, 2005; Gaitanos et al., 1993) and blood (D. Bishop, Lawrence, & Spencer, 2003). H+ accumulation can lower pH within the muscle and may inhibit phosphofructokinase (PFK), slowing glycolysis as well as displacing Ca2+ from troponin and hindering muscle contraction (Brooks, Fahey, & Baldwin, 2005). However, there is more recent contrary evidence calling into question acidosis at physiological temperatures as a direct cause of muscle fatigue (Gaitanos, Nevill, Brooks, & Williams, 1991; Girard et al., 2011; Matsuura, Arimitsu, Kimura, Yunoki, & Yano, 2007; Westerblad, Allen, & Lannergren, 2002), and further research is appropriate to determine the effects H+ accumulation has on repeat-sprint ability.

It appears that the accumulation of inorganic phosphate as a result of PCr hydrolysis during anaerobic metabolism may have a more significant influence on fatigue (Westerblad & Allen, 2002; Westerblad et al., 2002). Studies have shown that, on isolated muscle fibers and enzymes, phosphate (Pi) interferes with PFK and cross-bridge binding of Ca2+ (Brooks et al., 2005; Westerblad et al., 2002). It appears that the altered cross-bridge function may reduce Ca2+ myofibrillar sensitivity and reduce force production (Westerblad & Allen, 2002; Westerblad et

al., 2002). It is also suggested that Pi directly acts on Ca2+ release and uptake in the sarcoplasmic reticulum, but results are as yet equivocal (Westerblad & Allen, 2002; Westerblad et al., 2002).

Skeletal muscle excitability may also play a role in fatigue as it has been well established that during contractile activity, muscles lose K+ and gain Na+ (Clausen, Nielsen, Harrison, Flatman, & Overgaard, 1998; Fenn, 1940) due to the inability of the Na+, K+ pump to restore K+ and can lead to a doubling in extracellular K+ and effecting ATPase activity (D. J. Bishop, 2012; Girard et al., 2011; Juel, Pilegaard, Nielsen, & Bangsbo, 2000). This has the effect of impairing cell membrane excitability and reducing force development (Ruff, Simoncini, & Stuhmer, 1988). Since most studies in this area have been performed in vitro, interpretation is still equivocal (Girard et al., 2011).

Central and neuromuscular fatigue. While the previously listed factors are potential contributors to peripheral fatigue, the research is still equivocal on the level of contribution central fatigue plays in repeat-sprint or intermittent-sprint exercise (D. J. Bishop, 2012; Girard et al., 2011). It takes a high level of neural drive to sprint at a maximum level (Ross, Leveritt, & Riek, 2001). When assessing muscle activation during repeat-sprint exercise through surface electromyogram (EMG), several studies show a decline in the amplitude of EMG signals (Mendez-Villanueva, Hamer, & Bishop, 2007, 2008); however, the findings are not consistent (Billaut & Basset, 2007; Hautier et al., 2000). Considering the influence with respect to the level of fatigue, low levels (< 10% reduction in a fatigue index or sprint performance) show neural activation to remain consistent (Billaut & Basset, 2007; Hautier et al., 2000; Perrey, Racinais, Saimouaa, & Girard, 2010). In contrast, when fatigue levels are high (> 10%) the body of evidence consistently shows a reduction in EMG amplitude and may indicate the inability to

reach full neural activation whether through motor unit recruitment or potentially firing rate (D. J. Bishop, 2012; Girard et al., 2011; Mendez-Villanueva et al., 2007, 2008; Racinais et al., 2007). There are, however, some acknowledged issues that create signal interferences that may factor in interpreting EMG data, such as excessive sweat, an amplitude cancellation phenomena, changes in fiber membrane, and motor unit properties. (D. J. Bishop, 2012; Farina, Merletti, & Enoka, 2004; Girard et al., 2011).

The extent to which the central nervous system (CNS) regulates neural drive in repeat sprint exercise has still not been fully explored (Girard et al., 2011). However, it is postulated that through various afferent signals the CNS can modify central neural drive (Amann & Dempsey, 2008). It has been shown that various levels of initial peripheral fatigue have an impact on central motor command over a 5 km cycling time trial as measured though quadriceps EMG, potentiated quadriceps twitch force, power output, and performance time (Amann & Dempsey, 2008). It has also been demonstrated that changes in arterial O2 content can attenuate power output in both endurance and repeat sprint exercise as measured through surface EMG despite a consistent level of peripheral fatigue (Amann et al., 2006; Billaut & Smith, 2010). These studies indicate a potential for afferent signals to influence central neural drive and the CNS participation in regulatory action.

Muscle recruitment and motor unit recruitment patterns may also contribute to fatigue during repeat sprint exercise (Girard et al., 2011). One study has highlighted a reduction in EMG root mean square (RMS) of knee flexor muscles and an unchanged RMS of knee extensor muscles over a 15 X 5-second repeat sprint cycling protocol (Hautier et al., 2000). This suggests fatigue induced reduction in co-activation as agonist force is lost (Hautier et al., 2000). These findings were interpreted as creating an inter-muscular coordination adaption to reduce force and

power while cycling (Hautier et al., 2000). The authors do caution that training status may play a factor in the results (Hautier et al., 2000). In the case of motor unit recruitment patters, it's been suggested that during repeat sprint exercise, due to greater muscle Type 2 fiber fatigability, there is great relative input by Type 1 fibers (Girard et al., 2011). Results interpretations are also cautioned here due to test validity on maximal voluntary contractions related to fatigue based upon methodological differences (Girard et al., 2011).

In many team sports, environmental factors such as extreme temperatures, humidity and altitude can potentially affect player fatigue. At the performance levels of ice hockey and the majority of youth levels, competition and training occur in enclosed facilities and are not exposed to extreme weather. There are also few locations where altitude plays a factor. However, hypohydration status due to impaired thermoregulatory capacity from the restricted heat dissipation of the required protective equipment and clothing has been demonstrated in at least one study on National Collegiate Athletic Association hockey players (Batchelder, Krause, Seegmiller, & Starkey, 2010). The overall effect on player fatigue is yet to be established.

Evaluating Load to Assess Fatigue

Monitoring training or competition load, fatigue levels, and performance in elite athletes through the use of new technology is an established practice in many sports, such as rugby (Gabbett, Jenkins, & Abernethy, 2012; Kempton, Sirotic, & Coutts, 2015; Sirotic, Coutts, Knowles, & Catterick, 2009), Australian football (Cormack, Mooney, Morgan, & McGuigan, 2013; Henderson, Cook, Kidgell, & Gastin, 2015), soccer (Akenhead, Hayes, Thompson, & French, 2013), and netball (Cormack, Smith, Mooney, Young, & O'Brien, 2014). It is, however, a recent development at the professional and collegiate levels in the sport of ice hockey. At the

National Hockey League level, the use of wearable technology is still prohibited during competition (NHL & National Hockey League Players Association, 2013).

Evaluation methods of the load placed upon athletes in competition can be divided into two categories: internal and external. Internal measures, as defined by Bourdon et al. (2017), are the biological stressors (physiological and psychological) placed on athletes during competition, while external loads are objective measures of actual work performed and are assessed independently of internal load.

Measures of internal load include heart rate (HR), oxygen consumption, ratings of perceived exertion (RPE), session rating of perceived exertion, HR-to-RPE ratio, training impulse (TRIMP), blood lactate concentrations, lactate-to-RPE ratio, HR recovery (HRR), HR variability, critical power, psychomotor speed, sleep, questionnaire/diary, and biochemical/hormonal/immunological assessments. Measures of external load include power output, speed, acceleration, time-motion analysis, and neuromuscular function (Borresen & Lambert, 2009; Bourdon et al., 2017; Halson, 2014). Each methodology has strengths and weaknesses due to the nature of the sport being monitored, ease to administer, cost, practicality and the level of reliability and validity (Bourdon et al., 2017). In team sports such as ice hockey, to monitor load during competition, the sport environment may preclude certain methods as unsuitable or unpractical. The most applicable methods evaluate physiological changes and assess movement patterns and indicators of skills specific to the sport (Halson, 2014).

A combined usage of both internal and external measures may provide even greater insight into athlete fatigue. Bourdon et al. (2017) points out that the uncoupling of internal and external loads may better demonstrate the fatigue or freshness level of the athlete. For example, two athletes who work at the same power output (external load) for the same duration may

display different internal loads such as HR and thus expose their potential for fatigue (Halson, 2014).

Monitoring individual load within the team setting is also of significance as individual athletes respond differently to stimulus and exposure may vary depending on factors inherent with the competition environment such as player position or amount of playing time (Halson, 2014). Factors influencing the variability in response may include age, sex, current fitness status, or training frequency (Borresen & Lambert, 2009; Bourdon et al., 2017). Thus, monitoring the individual load allows for better prescription by coaches to match the needs of the athlete with the needs of the team (Halson, 2014).

Monitoring Load in Team Sports

Monitoring large groups of athletes as opposed to an individual sport athlete presents different challenges. The nature of team sports require a wide range of movement patterns and that can be difficult to assess (Polglaze, Dawson, & Peeling, 2016; Spencer et al., 2005). Time motion analysis though video and global positioning system (GPS) is a common external methodology used in many team sports (Bourdon et al., 2017), while HR is a common internal measure (Achten & Jeukendrup, 2003). Both are relatively noninvasive and, through current technology, track large numbers of athletes simultaneously.

Heart rate. The exercise load is evaluated through three components: frequency of exercise, duration of exercise, and intensity of exercise (Achten & Jeukendrup, 2003; Borresen & Lambert, 2009), with frequency and duration being the easier components to measure. There are a number of methodologies that can be used to assess exercise intensity; however, there is a need to balance between validity and practicality (Achten & Jeukendrup, 2003). Using HR as an indicator of exercise intensity has become inexpensive, easy to use, and can be deployed in most

sporting activities (Achten & Jeukendrup, 2003). While HR monitors vary in their accuracy, there is general consensus that those devices using chest electrodes are considered valid and reliable for use during exercise in a group setting (Achten & Jeukendrup, 2003). HR monitoring is less accurate for individuals as day-to-day variability of approximately 6 beats/min, or < 6.5%, has been established (Achten & Jeukendrup, 2003; Bagger, Petersen, & Pedersen, 2003; Borresen & Lambert, 2009; Lambert, Mbambo, & St Clair Gibson, 1998). When conditions such as training status, environment, hydration status, and altitude are controlled, changes of 2 to 4 beats/min are still probable (Achten & Jeukendrup, 2003; Borresen & Lambert, 2009).

Exercise intensity, defined as the amount of energy expended to perform a specific task per minute (kJ/min; Jeukendrup & VanDiemen, 1998), is difficult to measure directly outside laboratory settings (Achten & Jeukendrup, 2003). HR as a marker of exercise intensity or energy expenditure relies on the established linear relationship between HR and VO2 consumption up to near maximal exercise (Achten & Jeukendrup, 2003; Borresen & Lambert, 2009). An estimation of energy expenditure can be calculated from HR once the individual HR–VO2 relationship is measured (Achten & Jeukendrup, 2003). Again, there is a consensus that HR can provide an acceptable estimation of energy expenditure for groups; the accuracy is diminished for individuals (Achten & Jeukendrup, 2003; Ceesay et al., 1989; McCrory, Mole, Nommsen-Rivers, & Dewey, 1997). Issues in team sports arise as the work rates are intermittent and changes in HR respond slowly. A rapid increase or decrease in work rate is not instantaneously reflected in HR and does not reflect the HR that would occur after several minutes of work at that steady-state level of exertion (Achten & Jeukendrup, 2003). This reduced accuracy suggests that HR can only be used as an estimate of energy expenditure and more specifically within a group (Achten & Jeukendrup, 2003; P. G. Montgomery et al., 2009).

In ice hockey and other team sports, HR has been used to estimate exercise intensity and energy expenditure in competition (A. Coutts, Reaburn, & Abt, 2003; Jackson, Snydmiller, Game, Gervais, & Bell, 2016; Seliger et al., 1972; Spiering, Wilson, Judelson, & Rundell, 2003; Stanula & Roczniak, 2014). In one example, Stanula and Rocziok, established HR zones based on off-ice incremental test to exhaustion to determine VO2 max and HRmax. Low, moderate, and high HR zones were established by playing position (forwards and defense) on measures based off of determined first and second ventilatory thresholds as a percentage of HRmax. Game time spent in each HR zone was then calculated to assess exercise intensity. The study found that defensive players spent 22% of their playing time, respectively, in the high-intensity zone (HR exceeding 94.55% of HRmax) and 22% of their time in the moderate-intensity zone (HR between 82.6 and 94.0% of HRmax). Forwards spent 19% of their time in the high zone and 26% of their playing time in the moderate zone (Stanula & Roczniak, 2014). It has been established that in-game intensity contrasts with training intensities (Cox et al., 1995; H. Green et al., 1976; Spiering et al., 2003; Stanula & Roczniak, 2014). And this study also demonstrated this as the means of HRmax were different between the recorded in-game indices and the off-ice incremental tests (Stanula & Roczniak, 2014). This points to the need for specificity when practical as in-competition measures provide the most relevant information.

Heart rate limitations. Limitations to using HR as a measure of exercise intensity beyond the lag in response to changes in work rate and overall day-to-day variability include cardiac drift, hydration status and environmental factors such as temperature and altitude (Achten & Jeukendrup, 2003).

Cardiac drift refers to the gradual decrease in stroke volume and increase in HR over time during exercise. HR has been shown to increase as much as 15% over 5 to 60 minutes of exercise

(Achten & Jeukendrup, 2003; Ekelund, 1967). It has been proposed that cardiac drift is attributable to fluid loss and vasodilation where research has demonstrated a 10% increase in HR when no fluid was consumed during exercise and only a 5% increase when exogenous fluid was consumed while maintaining cardiac output (Achten & Jeukendrup, 2003; Hamilton, Gonzalez-Alonso, Montain, & Coyle, 1991).

Similarly, hydration status, with regard to dehydration, has been demonstrated to increase HR by as much as 7.5% and decrease stroke volume (Achten & Jeukendrup, 2003). Four percent dehydration was shown to increase HR by 5% and decrease stroke volume 7%. Once blood plasma volumes were restored to normal levels, stroke volume decline was offset (Achten & Jeukendrup, 2003; Gonzalez-Alonso, Mora-Rodriguez, Below, & Coyle, 1997).

Temperatures in both hot and cold environments can also significantly affect HR response to exercise (Achten & Jeukendrup, 2003). It has been shown that in hot environments, heart rate increases and is purported to be due to the rise in core body temperature. Research evaluating exercise in a 40º C environment demonstrated that when core temperature was manipulated through water emersion, at 17º, 36º C, and 40º for 30 minutes prior to exercise, the respective HR responses after 10 minutes were 140 ± 5, 166 ± 5 and 182 ± 4 beats per minute at a work rate of 60% VO2max (Gonzalez-Alonso et al., 1999). Increases in HR in hot environments overestimates exercise intensity (Achten & Jeukendrup, 2003).

The body's response to cold environments causes peripheral vasoconstriction and an increase in central blood volume, which in turn, raises blood pressure through increased afterload on the heart (Brooks et al., 2005). Studies tend to show a decrease in HR and an increase in stroke volume in cold environments, which underestimate exercise intensity (Achten & Jeukendrup, 2003; Brooks et al., 2005).

Altitude is another environmental factor that affects HR during exercise. While the O2 percentage content of air is the same at altitude as it is at sea level, the drop in barometric pressure decreases the O2 content and has the effect of lowering O2 transport capacity and increasing the perception of work (Brooks et al., 2005). At rest and during submaximal exercise, cardiac output is increased through a rise in HR (Achten & Jeukendrup, 2003; Brooks et al., 2005; Vogel, Hansen, & Harris, 1967). At an altitude of 4,300 m, after two to three days of exposure, a 15% increase in HR was displayed compared to sea level at a moderate exercise intensity (Vogel et al., 1967). Another study stated a 22% increase in HR during submaximal exercise at 3,800 m compared to sea level (Klausen, 1966). In contrast, HR at altitude during maximal effort has been shown to remain the same or marginally decrease. In the same study comparing HR response to exercise at 4,300 m, maximal HR was shown to decrease from 180 beats/min at sea level to 176 beats/min (Vogel et al., 1967).

Time-motion analysis. Time-motion analysis through the use of video, GPS, or local position measurement systems (LPM) has been widely used in field-based team sports (Spencer et al., 2005). In the sport of ice hockey, time-motion has been used to determine the amount of time spent on ice and performing various sports-specific activities: gliding, standing, low-intensity skating, moderate-intensity skating, high-intensity skating, forward accelerating, turning, backwards skating, and combative struggling with opponents (Bracko et al., 1988; Jackson et al., 2016). The process of determining these results for each player has traditionally been labor intensive and difficult to provide timely feedback for practical use. New indoor technology may enhance this capability, but it is not yet widely available (Prozone by Stats LLC, Chicago, USA; HockeyTech, Waterloo, Canada; Kinexon, Munchen, Germany; Quuppa, Espoo, Finland).

In many field-based team sports, widespread time-motion analysis has been done in real-time through GPS technology and LPM systems (Aughey, 2011; Cummins, Orr, O'Connor, & West, 2013; Spencer et al., 2005). The GPS sensors can record player field positions at 1-, 5-, and 10-Hz sampling rates with the higher rates providing greater validity (Akenhead, French, Thompson, & Hayes, 2014; Cummins et al., 2013). This information is used to quantify the displacement of athletes as they maneuver around the playing surface, determining distances traveled, accelerations, and speeds. Energy expenditure is determined by work-rate patterns that have been typically set in up to six zones, over a range of 0.0 to 36.0 km/h-1. (Cummins et al., 2013). Various descriptions have been set for each zone, such as standing, walking, jogging, striding, running, moderate-intensity running, high-intensity running, and sprinting, although there are no standards, which makes comparisons difficult (Cummins et al., 2013; Spencer et al., 2005).

GPS is easy to use and non-evasive, and its accessibility is improving. Its limitations are that it can only be used outdoors with clear line of sight and it cannot distinguish many sports-specific movements that affect overall energy expenditure (Polglaze et al., 2016), for example, movements such as struggling with opponents in confrontational situations or kicking a ball. LPM technology is now becoming accessible for indoor sports and tracks player displacements in the same manner as GPS (Polglaze et al., 2016). It also has similar limitations in distinguishing sports-specific movements that affect energy expenditure. In ice hockey, activities similar to other sports would include physical confrontations with opponents or taking a slap shot.

As reviewed by Polglaze, Dawson, and Peeling (2016), the basis of using displacement to evaluate locomotion in walking and running is that the energy cost of running is independent of

speed, when traveling on a level surface. In walking, the energy cost increases as speed increases. This contrasts with ice skating. At long distances where metabolic power is predominantly supplied through aerobic metabolism, the energy cost of skating is also independent of skating speed. However, at shorter distances (less than 10 km) the energy cost of skating increases with speed (Formenti, 2014; Formenti & Minetti, 2007). In effect, this makes the use of displacement as a measure of energy expenditure ineffective for ice hockey. Even in field-based team sports, when taking into consideration the varied and dynamic movements required, power appears to be a more appropriate variable to measure energy cost than displacement (Polglaze et al., 2016). Work in this area attempts to incorporate metabolic power through the assessment of player accelerations measuring instantaneous velocity (A. J. Coutts et al., 2015; Osgnach, Poser, Bernardini, Rinaldo, & di Prampero, 2010).

Power output. The relationship between power output and performance has been established in sporting disciplines described as steady-state activity (Bourdin, Messonnier, Hager, & Lacour, 2004; Coyle et al., 1991). In sports where the efforts are more intermittent, a novel approach to comparing power output and performance has been used in cycling, creating a power profile over various time durations in the laboratory, and comparing this with the maximal mean power (MMP) over the same time durations during mass-start cycling road races (Quod, Martin, Martin, & Laursen, 2010). The power profile established in the laboratory records maximal efforts produced over a number of time intervals from 5 to 600 seconds demonstrating the athlete's capacity. Due to the sporadic nature of efforts produced during actual cycling road races owing to tactics, drafting and terrain, only the highest individual MMP for each period was evaluated. Race data were accumulated over 10 races so that the potential for race conditions to require maximal effort for each time frame was increased. The results demonstrated the potential

to directly compare the capacity to produce power between both laboratory and the performance settings.

The intermittent efforts generated during race conditions in various cycling disciplines are similar to the irregular efforts produce during an ice hockey game. These interspersed efforts consist of coasting, accelerating, sprinting, changes in direction, physical confrontations with opponents and resting on the bench (Bracko et al., 1988; Quod et al., 2010; Van Iterson, Fitzgerald, Dietz, Snyder, & Peterson, 2017).

Triaxial accelerometers. In an attempt to monitor more sports-specific movements, the use of triaxial accelerometer is a new tool in team sports and has been shown to be reliable (Boyd, Ball, & Aughey, 2011; Cormack et al., 2013; Cormack et al., 2014; Van Iterson et al., 2017; Walker, McAinch, Sweeting, & Aughey, 2016). The accelerometer measures a composite vector magnitude from the accelerations in three orthogonal planes (anteroposterior, mediolateral, and vertical) conveyed as a G-force (Chen & Bassett, 2005; Cummins et al., 2013). This includes all forces from acceleration or deceleration in changes of direction and impacts between players or with the ground (foot strikes and falls; Cummins et al., 2013). Accelerometers use the relationship between speed and acceleration (speed equals change in position over time; acceleration equals change in speed over time; Chen & Bassett, 2005). Acceleration is proportional to the net external forces involved and better reflects the energy costs associated with physical activity making it more enhanced evaluation variable than speed (Chen & Bassett, 2005).

Accelerometers used in measuring physical activity tend to utilize piezoelectric technology in one of two common structures, beam sensors or integrated chips (Figure 1; Chen & Bassett, 2005). Both structure enclose a piezoelectric element and a seismic mass.

Accelerations displace the seismic mass, causing a deformation in the piezoelectric element, and generate a voltage signal on one side of the element that is proportional to magnitude of the acceleration (Chen & Bassett, 2005). Specific to the beam structure, the element is most sensitive to the bending the intended direction; however, there can be deformation in other orientations (Chen & Bassett, 2005). The sensitivity to deformation in other planes or directions is dependent upon stiffness, cross-sectional area, and length of the piezoelectric material, and all beam accelerometers display this type of omnidirectional deformation to some degree (Chen & Bassett, 2005).
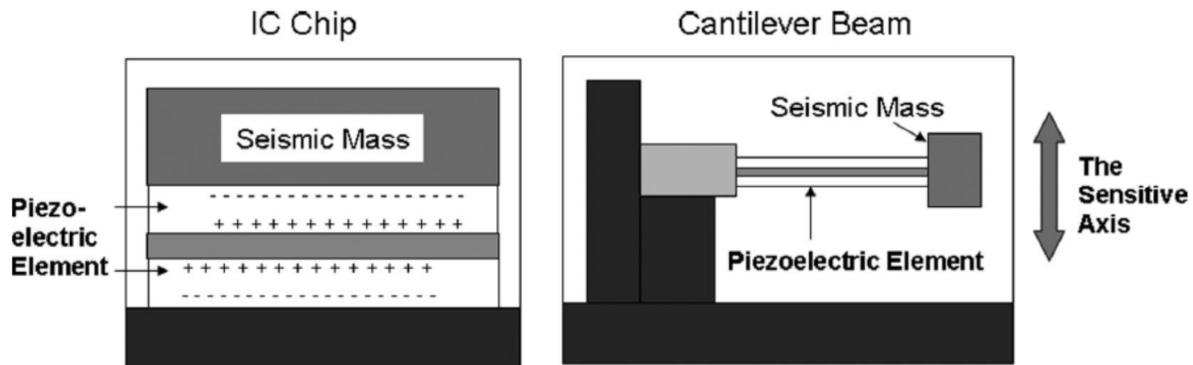
Figure 1. Common piezoelectric accelerometer configurations. Reprinted from, *The technology of accelerometry-based activity monitors: current and future,* by Chen & Bassett (2005).

The accelerometer data output is then determined by sampling frequency, filtered by bandwidth and activity counts that are defined periods of time termed (epoch; Chen & Bassett, 2005; Yang & Hsu, 2010). Sampling frequency needs to be twice the highest frequency of movement to satisfy the Nyquist criterion for the digitation of analog signals, which limits the distortion in higher frequency motions (Chen & Bassett, 2005; Oppenheim, 1983). Bandwidth filtering attempts to attenuate extreme highs and lows, increasing the linearity of output, clarifying the acceleration and reducing potential artifact (Chen & Bassett, 2005). The defined activity counts are the raw output of accelerometer signals, which are based upon the

deformation of the piezoelectric elements and can be of positive or negative voltage. The analog

voltage signal is sampled at a predetermined frequency and is then converted to a digital signal

representing the raw counts. This digital data can then be analyzed using several approaches:

counting the number of times a signal surpasses a set threshold, using an algorithm to determine

the maximum value attained over a set time frame (epoch), or using an integration algorithm to

determine the area under the curve (Chen & Bassett, 2005).

The major limitations to piezoelectric technology are in temperature-sensitive drift, low-

level output signals, and leakage of the initial charge over time. (Chen & Bassett, 2005; Yang &

Hsu, 2010). Temperature-related drift affects the signal at very low frequency (> .01 Hz) and the

use of bandwidth filtering is used to minimize this effect (Chen & Bassett, 2005). The leakage of

initial charge is dependent on the physical properties of the piezoelectric material and expresses

in a time constant (Chen & Bassett, 2005).

From a human movement standpoint, triaxial accelerometers have the ability to

distinguish various types of physical activity including lying, standing, walking, and running to

cycling, as well as duration and intensity of these activities through the use of models and

acceleration algorithms (Bonomi, Goris, Yin, & Westerterp, 2009). In evaluating more sport-

specific movements, accelerometers have been shown to be valid in accessing various activities

from hopping in place height, force during squat, and countermovement jumps to stroke patterns

in swimming and particular cross country skiing movements such as kicking and skating

(Beanland, Main, Aisbett, Gastin, & Netto, 2014; Choukou, Laffaye, & Taiar, 2014; Marsland et

al., 2012). Specific to ice hockey, the triaxial accelerometer has been shown to reliably measure

on-ice forward acceleration, backward acceleration, forward top speed, backward top speed,

repeated shift test, slap shot, bench-sitting, and coasting (Van Iterson et al., 2017).

Specific to this project proposal, the player-worn monitoring device that will be used is the Zephyr Bioharness-3 (Zephyr Technologies, MD, USA). The Bioharness-3 is a multivariable monitor (weight 18 g, 28 diam. x 7 mm) that is worn next to the skin and mounted on a chest strap (71 g; Zephry Technology, 2012). The device acts as a data logger and has a 480-hour memory with a 10-hour battery life (Johnstone, Ford, Hughes, Watson, & Garrett, 2012b). The internal triaxial accelerometer uses piezoelectric technology with a cantilever beam setup and a capacitive measurement system (Johnstone, Ford, et al., 2012b). The Bioharness-3 is designed to concurrently measure heartrate, ventilatory rate and accelerations. The device structure measures accelerations along three orthogonal axes X, Y and Z (Johnstone, Ford, et al., 2012b; Zephyr Technology, 2016). Acceleration data is measured in gravitational force units (G) with a range of +3 to -3 G along each axis or as vector magnitude units (VMU; Johnstone, Ford, et al., 2012b). Dynamic range is 16 G, with a sampling frequency of 100 Hz and bandwidth of 50 Hz (Zephry Technology, 2012).

The Bioharness-3 also accesses HR through ECG at 250 Hz (Zephyr Technology, 2016). As previously stated, monitors using chest electrodes provide the best reliability and validity for exercise. The device measures HR in beats per minute (BPM) in a range of 25 to 240 with and accuracy of ± 1 BPM (Zephry Technology, 2012).

The Bioharness-3 has been deemed reliable and valid in both laboratory and field settings (Johnstone, Ford, Hughes, Watson, & Garrett, 2012a; Johnstone, Ford, et al., 2012b; Johnstone, Ford, Hughes, Watson, Mitchell, et al., 2012). With the triaxial accelerometer showing the strongest reliability and validity in the field setting with very strong data relationships. At higher velocities, the CV remains stable; however, variability was shown to increase (Johnstone, Ford, Hughes, Watson, Mitchell, et al., 2012). HR measures showed good precision and repeatability

at velocities of 4-6 km/h-1 but diminished at higher levels. Reliability remained relatively strong until the highest velocities tested (Johnstone, Ford, Hughes, Watson, Mitchell, et al., 2012).

Since change in force equals mass times acceleration, work equals change in force divided by distance and power equals work divided by time. Through substitution we can determine the basic relationship between acceleration and power (McGinnis, 2013). Using the Zephyr Bioharness-3 technology and following the establish protocol previously stated in cycling (which evaluates peak power outputs over different time frames), the aim of this project is to evaluate peak accelerations during ice hockey competitions. Assuming that a consistent effort level between competitions occurs, a reduction in game-to-game peak accelerations would indicated a skating performance decrement due to fatigue.

Methods

Subjects

The subject group consisted of players from two elite youth ice hockey teams ($N = 33$), in two age categories (18U: $N = 17$, 16U: $N = 16$), who competed in a 3-day, Tier 1 Elite Hockey League showcase. Each team competed in four games over the three days. The 16U team played two games on Day 1, one game on Day 2 and three. The 18U team played one game on Day 1 and 2 with two games on Day 3.

Players consented to procedures approved by the Eastern Michigan University Human Subjects Review Committee (See Appendix A). Prior to the first on-ice competition, the participants were fitted with a Zephyr Bioharness-3 (Zephyr Technologies, MD, USA) player-worn sensor (PWS) and instructed to play and compete as normal throughout the event. The sensors were coded to the selected participants at this time based upon position (D1 for the first defenseman, D2 for the second, etc.). Before each on-ice session and at the conclusion of each session, staff were in attendance to turn the sensors on and off as well as to check on the athletes' well-being.

Data Collection and Analysis

For all on-ice sessions, players wore the Zephyr Bioharness-3 (Zephyr Technologies, MD, USA) PWS mounted across their chest. The PWS recorded heart rate through ECG at 250 Hz measuring HR in beats per minute (BPM) in a range of 25 to 240 with and accuracy of ± 1 BPM. Accelerations at 1 Hz are measured in gravitational force units (G) with a range of +3 to -3 G along each axis and a dynamic range of 16 G. This was done over the four games (G1 to G4) during the 3-day event. Data were saved on board the sensor for later download. The first and second games played in a day were also separately designated as M1 and M2.

After the on-ice sessions, the PWSs were retrieved and downloaded to Omnisense software (Zephyr Technologies, MD, USA). Data were then exported from Omnisense software and converted using proprietary Javascript to an Oracle database for analysis. Database queries were processed using Designer (Alteryx, CA).

In order to discriminate energy system/biochemical sources of performance changes, peak accelerations (ACC) across multiple time frames (3, 5, 10, 15, 20, 30, 40, 50, 60, 90 seconds and 2, 2.5, 3, 5, 10, 20, 30, 45 minutes) were quantified and analyzed. These time frames will be subdivided into candidate physiologically relevant categories: 3–10 sec = neuromuscular, 15–60 sec = anaerobic, and > 60 sec = aerobic energy systems. HR was also quantified and used in conjunction with ACC to determine exertion profiles for each on-ice session. The individual athletes were grouped by their respective teams, 16U team and 18U team and the two groups will be compared. If significant differences were not displayed between teams, then the entire population was evaluated $N = 33$. MANOVAs for peak ACC and HR at each time point across G1–G4 with Bonferroni *post hocs* and multiple games per day (M1, M2) for magnitude and time as main effects were performed using SPSS 23.0 (IBM, NY; $\alpha=0.05$). Effect sizes were evaluated though partial eta squared (0.010 to 0.059 small effects, 0.060 to 0.139 medium effects, $0.14 \leq$ large effects; Ellis, 2010).

The purpose of this study was to use accelerations and heart rate to estimate fatigue during a three-day competition for elite youth ice hockey players. It was hypothesized that there will not be a significant change in peak accelerations and heart rate over the course of this short-term event.

Results

Neuromuscular Time Fames

In game peak ACC for 3-, 5-, and 10-second time frames were analyzed, with no significant differences between teams or between games being displayed. During multiple games played in the same day, a small effect size (0.010 partial eta squared; η^2) was indicated at the 5-second time interval, a 2.3% decrease in mean peak ACC between M1 and M2. It should be noted that peak ACCs recorded during the neuromuscular time frames may be influence by player impacts.

HR was analyzed only at the 10-second time interval with no significant differences displayed between teams, games, or multiple games in a day. Yet small effect sizes were present between games (0.043 η^2, 4.6% decrease in means from G1 to G4) and between multiple games in a day (0.010 η^2, 2.5% decrease in means from M1 to M2).

Anaerobic Time Frames

Peak ACC in the anaerobic time frames from 15 seconds through 60 seconds showed no significant difference between teams. When peak ACCs were compared between games, small effects were present at all time segments (15-sec 0.022 η^2, 20-sec 0.023 η^2, 30-sec 0.020 η^2, 40-sec 0.028 η^2, 50-sec 0.028 η^2, 60-sec 0.045 η^2), showing decreases in means of (15-sec 0.8%, 20-sec 1.0%, 30-sec 1.2%, 40-sec 1.9%, 50-sec 2.0%, 60-sec 2.5%) between G1 and G4.

Between multiple games in a day, peak ACC was significant (0.028) at the 60-second time frame with a decrease in means of 6.2% between M1 and M2. All other time segments displayed small effect sizes (15-sec 0.028 η^2, 20-sec 0.027 η^2, 30-sec 0.027 η^2, 40-sec 0.035 η^2, 50-sec 0.049 η^2) with a decrease in means of (15 sec 3.3%, 20 sec 3.4%, 30 sec 3.6%, 40 sec 4.3 %, 50 sec 5.2%).

For HR through the anaerobic time frames, no significant differences between teams were observed. However, between games G1 and G4, there were small size effects shown at each anaerobic time frame (20-sec 0.047 η^2, 30-sec 0.048 η^2, 40-sec 0.050 η^2, 50-sec 0.051 η^2, 60-sec 0.049 η^2) with decreases in means of (20 sec 4.9%, 30 sec 4.9%, 40 sec 5.0%, 50 sec 4.9%, 60 sec 4.7%). The analysis between multiple games played in a day showed HR changes approaching significance at the 40-second (.065), 50-second (.060) and 60-second (.060) segments with small effect sizes present across each time frame (20-sec 0.037 η^2, 30-sec 0.049 η^2, 40-sec 0.054 η^2, 50-sec 0.056 η^2, 60-sec 0.056 η^2). The respective decreases in means for all time frames were (20-sec 3.3%, 30-sec 3.7%, 40-sec 3.9%, 50-sec 3.9%, 60-sec 3.9%).

Aerobic Time Frames

The aerobic time frames analyzed were 90, 120, 300, 600, 900, 1,200, 2,700, 3,600 and 5,400-seconds for both peak ACC and HR.

Peak ACC between teams showed significance ($p = 0.019$) only at the 300-second time frame and small effect sizes at 150-seconds (0.014 η^2) and 180-seconds (0.013 η^2). No other relevant differences between teams were noted. No significance was present when analyzing peak ACC between teams and game number, but small effect sizes were present across most time fames within the aerobic category (90-sec 0.037 η^2, 120-sec 0.018 η^2, 150-sec 0.014 η^2, 180-sec 0.034 η^2, 300-sec 0.027 η^2, 600-sec 0.035 η^2, 900-sec 0.031 η^2, 1,200-sec 0.014 η^2, 1,800-sec 0.019 η^2, 3,600-sec 0.012 η^2, 5,400-sec 0.010 η^2).

When comparing peak ACC for multiple games in a day, M1 to M2, significance was exhibited at 90-seconds ($p = .042$) and 180-seconds ($p = .040$) while approaching significance and displaying small or medium size effects at 120-seconds ($p = 0.051$ sig, 0.060 η^2.) and 150-seconds ($p = 0.067$ sig, 0.053 η^2; Figures 2 and 3). All other time frames showed small effect

sizes except 2,700-seconds (300-sec 0.030 η^2, 600-sec 0.025 η^2, 900-sec 0.031 η^2, 1,200-sec 0.018 η^2, 1,800-sec 0.018 η^2, 3,600-sec 0.027 η^2, 5,400-sec 0.023 η^2). The decreases in means between M1 and M2 for peak ACC were (90-sec 5.50%, 120-sec 5.21%, 150-sec 5.48%, 180-sec 6.39%, 300-sec 3.98%, 600-sec 3.47%, 900-sec 4.02%, 1,200-sec 3.18%, 1,800-sec 3.26%, 3,600-sec 4.98%, 5,400-sec 6.61%).

There was no significant differences between teams for HR across all aerobic time frames. When HR was analyzed by game number, statistical significance was attained for each time frame between 180-seconds through 2,700-seconds (180-sec, $p = 0.041$; 300-sec, $p = 0.018$; 600-sec, $p = 0.019$; 900-sec, $p = 0.006$; 1,200-sec, $p = 0.007$; 2,700-sec, $p = 0.043$) between G1 and G4 (Figures 4 to 8). In addition, significance was also reached between G1 and G3 at 180-sec ($p = 0.047$), 300-sec ($p = 0.011$), 600-sec ($p = 0.008$), 900-sec ($p = 0.003$), 1,200-sec ($p = 0.005$), 2,700-sec ($p = 0.012$). The differences in mean beats per minute over the showcase from G1 to G4 in the specified time segments displayed a decrease between a 6.06% and 8.40% (180-sec, 187.46 ± 12.5 v 176.10; 300-sec, 181.43 ± 18.1 v 166.60 ± 21.3; 600-sec, 170.41 ± 15.8 v 157.57 ± 15.5; 900-sec, 167.15 ± 15.7 v 153.28 ± 15.0; 1,200-sec, 164.25 ± 15.5 v 150.45 ± 15.5; 2,700-sec, 155.92 ± 19.0 v 142.83 ± 14.2). Small effect sizes were displayed for all other time segments (90 sec, 0.049 η^2; 120 sec, 0.051 η^2; 150 sec, 0.059 η^2; 3,600 sec, 0.052 η^2; 5,400 sec, 0.031 η^2).

When controlling for team (16 Team only) and analyzing HR by game number no significance was shown. However, medium size effects were displayed between 90 seconds and 1,200 seconds (90 sec, 0.069 η^2; 120 sec, 0.073 η^2; 150 sec, 0.074 η^2; 180 sec, 0.078 η^2; 300 sec, 0.070 η^2; 600 sec, 0.071 η^2; 900 sec, 0.074 η^2; 1,200 sec 0.073 η^2). Small size effects were exhibited for the other time frames (2,700 sec, $\eta^2 = 0.039$; 3,600 sec, $\eta^2 = 0.035$; 5,400 sec, $\eta^2 = $

0.011). In contrast, when performing the same analysis for the 18 Team only, significance was attained from the 300-second through 5,400-second time frames (300 sec, $p = 0.005$; 600 sec, $p = 0.011$; 900 sec, $p = 0.002$; 1200 sec, $p = 0.002$; 2,700 sec, $p = 0.000$; 3,600 sec, $p = 0.0101$; 5,400 sec, $p = 0.018$). For the other aerobic time frames medium effect sizes were displayed (90 sec, $0.069 \, \eta^2$; 120 sec, $0.067 \, \eta^2$; 150 sec, $0.081 \, \eta^2$; 180 sec, $0.095 \, \eta^2$).

HR analysis for multiple games in a day, for the 16 Team only, showed no significance between M1 and M2. Yet small effect sizes were present from the 90-second through 2,700-second time frames (90-sec, $0.059 \, \eta^2$; 120-sec, $0.053 \, \eta^2$; 150-sec, $0.044 \, \eta^2$; 180-sec, $0.038 \, \eta^2$; 300-sec, $0.047 \, \eta^2$; 600-sec, $0.029 \, \eta^2$; 900-sec, $0.041 \, \eta^2$; 1,200-sec, $0.043 \, \eta^2$; 2,700-sec, $0.016 \, \eta^2$). There were similar results for the 18 Team, no statistical significance was displayed for HR between M1 and M2 but medium to small effect sizes were present across all time frames (90-sec, $0.069 \, \eta^2$; 120-sec, $0.065 \, \eta^2$; 150-sec, $0.056 \, \eta^2$; 180-sec, $0.075 \, \eta^2$; 300-sec, $0.040 \, \eta^2$; 600-sec, $0.024 \, \eta^2$; 900-sec, $0.027 \, \eta^2$; 1,200-sec, $0.035 \, \eta^2$; 2,700-sec, $0.064 \, \eta^2$; 3,600-sec, $0.060 \, \eta^2$; 5,400-sec, $0.059 \, \eta^2$).

Heart Rate to Peak ACC Ratios

While no statistical significance was achieved when analyzing the HR to peak ACC ratio, between teams, small effect sizes were present in various time frames. In the neuromuscular 10-second segment, a small effect size of 0.014 was displayed. While the 20-second anaerobic time frame also exhibited a small effect size of 0.014. Through the aerobic time frames analysis approached significance at the 300-second ($p = 0.053$, $0.030 \, \eta^2$) and 600-second ($p = 0.054$, $0.030 \, \eta^2$ segments). Small effect sizes were present at 180-seconds ($0.014 \, \eta^2$) and 2,700-seconds ($0.016 \, \eta^2$).

HR to peak ACC ratios, when evaluating by game number, again showed no statistical significance but display small size effects across all physiologically related time frames (10-sec, $\eta^2 = 0.026$; 20-sec, $\eta^2 = 0.028$; 30-sec, $\eta^2 = 0.019$; 40-sec, $\eta^2 = 0.016$; 50-sec, $\eta^2 = 0.027$; 60-sec, $\eta^2 = 0.045$; 90-sec, $\eta^2 = 0.057$; 120-sec, $\eta^2 = 0.037$; 180-sec, 0.049; $\eta^2 = 300$-sec, $\eta^2 = 0.018$; 600-sec, $\eta^2 = 0.017$; 900-sec, $\eta^2 = 0.016$; 1,200-sec, $\eta^2 = 0.058$; 2,700-sec, $\eta^2 = 0.054$; 3,600-sec, $\eta^2 = 0.022$; 5,400-sec, $\eta^2 = 0.022$).

For multiple games in a day, the HR to peak ACC ratios displayed small effect sizes in the 50-second though 180-second time frames (50-sec, $\eta^2 = 0.011$; 60-sec, $\eta^2 = 0.023$; 90-sec, $\eta^2 = 0.018$; 120-sec, $\eta^2 = 0.013$; 180-sec, $\eta^2 = 0.015$) and then again at 3600-seconds (0.015) and 5400-seconds ($\eta^2 = 0.010$).

Discussion

In this study, peak ACCs and HR were measured over various physiologically relevant time frames among two elite youth ice hockey teams that competed in a 3-day, Tier 1 Elite Hockey League showcase. It was hypothesized that there would be significant changes in player exertions (peak acceleration and heart rate) between games over the course of a 3-day ice hockey showcase event as an indication of fatigue. With respect to ACC, in contrast to the hypothesis, the results did not indicate a significant drop in peak ACCs over the event timeline between day 1 and day 3 when comparing the first games completed each day. There was, however, a noted decline in peak ACCs between two competitions carried out on the same day. This suggests a potential performance decrement due fatigue, in the second contest of the day.

With respect to HR, there was a significant decrease during the event in beats per minute (BPM), between the first game, and final two games of the showcase in the aerobic time frames between 180-seconds through 2,700-seconds. The differences in mean BPM over the showcase from G1 to G4 in the specified time segments displayed a decrease between a 6.06% and 8.40%. There were no significant differences between teams for HR across all time frames; however, the third game of the competition for the U16 Team was held on their second day of competition as opposed to the third day for the U18 Team. Thus the relevant drop in HR happened in as little as 24 hours for the U16 Team. This despite the consistency of peak ACCs over the course of the event, combined with the decreased HR, may indicate a potential cardiovascular adaptation, which was not expected.

Timing of Exercise

The timing and modes of intense exercise bouts and their effects on subsequent performance have been examined in sports with varying results (M. Johnston et al., 2017; M. J.

Johnston et al., 2016; Marrier et al., 2017; Russell et al., 2016). In looking at the effects of a single exercise session of team sport training, evaluation was made by comparing two 30-meter sprints and a set of 4 repetition counter movement jumps (CMJ) done before and at the conclusion of a demanding rugby sevens practice session. While the CMJ results were unclear, a slight increase in 30-meter sprint time was noted (1.0% ± 0.7%; Marrier et al., 2017). This increase in sprint time does correspond to the decreased acceleration displayed in the current study where a small effect size (0.010 partial eta squared) 2.3% decrease in mean peak ACC between M1 and M2 was indicated at the 5-second time interval.

Comparing the effects of a single session training day and a double session training day have also been explored through assessing biochemical, endocrine, and neuromuscular responses (M. J. Johnston et al., 2016; Russell et al., 2016). In one example, academy level rugby athletes preformed two randomized protocols consisting of a speed only-session (six, 50-meter sprints with 5 minutes of recovery between bouts) or the speed-only session followed two hours later by a lower body weight training session (four sets of five back squats and Roman deadlifts at 85% one repetition maximum). Neuromuscular, endocrine and biochemical markers were tested prior to, directly after, 2 hours after and 24 hours after each protocol. What was found was that the combined speed and weight sessions significantly increased muscle soreness as opposed to the speed only session (F = 4.757, $p \leq 0.05$, effect size $\eta = 0.253$). This was without significant differences in peak power, jump height, average rate of force generation, and relative peak power as determined from CMJ, and testosterone, cortisol, and creatine kinase not being affected by the additional weight training session (M. J. Johnston et al., 2016). This is congruent with what was observed in the current study as day-to-day peak ACC showed no significant changes between the first games played.

Another study exploring two-a-day sessions, assessed the effects of various training modes performed at 8:00 am on CMJ height, reaction time and repeat sprint ability later that same day at 2:00 pm. Fifteen profession rugby athletes performed one of four morning trainings regimes, either a bench press (5 X 10 repetition at 75% 1-repetition max with 90-seconds rest between sets), cycling (6 X 6 maximum sprint with 7.5% body mass load and 54-second recovery between bouts), running (6 X 40-meter sprints with 20 second recovery intervals), or active rest as a control group. Saliva samples were taken before both morning and afternoon sessions. Each of the morning training modes had a positive effect on a minimum of one afternoon performance marker with running being the most favorable. Jump height improved after cycling (0.012 ± 0.009 m, 2.31% ± 1.76 %, $p < .001$) and running (0.020 ± 0.009 m, 3.90% ± 1.79%, $p < .001$). Sprint performance improved after weights (0.15 ± 0.19 s, 2.04% ± 2.46%, $p < .05$) and running (0.15 ± 0.17 s, 2.12% ± 2.22%, $p < .05$) while reaction time was unaffected. Saliva cortisol was unchanged; however, testosterone was greater following weights (21 ± 23 pg/mL, 17% ± 18%, $p = .002$) and running (28 ± 26 pg/mL, 22% ± 20%, $p = .001$; Russell et al., 2016). These findings seem to dispute the present study with an increase in performance noted in the second testing session of the day. While results attainted in the current project reached a significant decrease in peak ACCs at the 60-second time interval, small effects were displayed at shorter durations as well.

Exploring the order of training modes and their effect on neuromuscular, physiological, and endocrine responses over a 24-hour time period has also been examined (M. Johnston et al., 2017). In one case, athletes performed a sprint protocol, followed two hours later by a weight training session. Then on a separate occasion, the order was reversed. Ratings of perceived muscle soreness, CMJs and blood samples were drawn from the athletes before and after each

training form and then again after 24 hours. Results indicated the order of the two training mode had no effect after any of the post exercise time points, on testosterone, cortisol, creatine kinase, CMJ or perceived muscle soreness ($p > .05$). On the other hand, 10-meter sprint time was reduced (1.80 ± 0.11s vs. 1.76 ± 0.08s; $p < .05$) when the sprint session was sequenced second (M. Johnston et al., 2017). While there is a decline in power output displayed in both this and the current project, alignment is equivocal due to the relative time frames involved. Significance was only reached at the 60-second internal in the present research.

The review of these studies indicate that the varied results may be specific to the mode of exercise and timing of the sessions. Research in the case of the current study is specific to ice hockey and is competition based as opposed to having a training orientation. It was previously established that ice skating has a different energy expenditure profile than other forms of locomotion (Formenti, 2014). Where significance was reached, time frames involved with these referenced studies do not align consistently with the current project providing equivocal results.

In the current study a significant decrease in Peak ACCs was demonstrated between multiple games played in the same day, M1 to M2, and were shown in time segments of 60 seconds through 180 seconds. There was as high as a 6.39% decrease in mean ACCs at the 180-second time interval. From a playing perspective, this is along the late anaerobic and into the aerobic time frames indicating a potential for a reduced playing shift capacity. Playing shifts can extend to over 60-second and the 180-second time segment may even extend into repeated playing shifts. The decrease in ACCs at the 180-second segment may indicate reduced recovery between shifts.

Muscle Glycogen Levels

Looking at the decrease in peak ACCs between M1 and M2 there is a potentially significant change in substrate availability between games. It has been previously shown when examining glycogen levels in the vastus lateralis muscles of collegiate ice hockey players that muscle glycogen levels decline an average of 60% over the course of a hockey game (H. J. Green et al., 1978). In the first 4 hours post-exercise, muscles with a depleted level of glycogen have been shown through gluconeogenesis to synthesize glycogen at a rate of 1-2 mmol•kg wet weight of muscle -1•h-1 without CHO intake (Burke et al., 2017). With CHO ingestion, resynthesis rates have been displayed in a range of 5-10 mmol•kg wet weight of muscle -1•h-1 and a mean rate of 5-6 mmol•kg wet wt of muscle -1•h-1 (Burke et al., 2017). Based upon the potential muscle glycogen depletion in the first game M1, and what has been established as normal replenishment rates, the player's ability to restore muscle glycogen to pre-competition levels before the second game in the day, M2, is severely limited (Burke et al., 2017; H. J. Green et al., 1978). This reduced substrate level has been shown in elite Swedish ice hockey players to effect skating speed. It was demonstrated that skating speed in the third period of game play was higher for individuals with greater glycogen levels at the games conclusion than those players who were in a more depleted state (Akermark et al., 1996). This could affect player ACCs. A similar reduction in ACCs would be expected in the second game of the day especially if that game was initiated with an already reduce glycogen store. The time between the conclusion of M1 and the start of M2 for both teams evaluated was approximately 3.5 hours.

The day-to-day timing between games ranged between 15 and 22 hours including an overnight sleep. Thus over the 3-day event the possibility of greater glycogen store

replenishment was certainly enhanced compared what was possible between M1 and M2. This may be reflective of the greater stability in peak ACCs over the entire showcase.

When considering the glycogen depletion patterns in ice hockey players, histochemical analysis was used to differentiate between Type I, Type IIA and Type IIB fiber types in the vastus lateralis muscles of collegiate level players (H. J. Green et al., 1978). Prior to competition, all fiber types were classified as stained dark. The greatest depletion was reported in Type I fibers, where post competition 66% stained light, 32% intermediate, and 2% dark compared to Type IIA at 15% stained light, 65% intermediate and 20% dark. A similar pattern to Type IIA was displayed for Type IIB (H. J. Green et al., 1978). This potentially indicates a greater reliance on oxidative metabolism, corresponding to the 60-second to 180-second time frames where we see a significant decrease in peak ACCs between M1 and M2.

Potential Heart Rate Adaptation

The decline in HR over the showcase, combined with the stability displayed in the peak ACCs, suggests a potential cardiovascular adaptation. This is despite the event being held mid-season with a player population that would enter the event with a certain fitness level. In a yet to be published study, a similar drop in HR was noted (D Stojanov, 2018). The exertion profiles for 46 teenage ice hockey players were analyzed over a 5-day short-term event, using the same Zephyr Bioharness-3 technology and evaluating peak ACCs and HR thorough similar physiological relevant time frames. The 5-day event consisted seven on-ice sessions including three traditional games, three practice sessions, and a 3 vs. 3 small-sided competition. While peak ACCs did show a decline over the 5-day event, it was not as profound as expected, yet HR did decrease in the aerobic time segments (60-seconds to 1,200-seconds; $p < .05$) from the first to third practice session (D Stojanov, 2018).

Blood plasma volume. The cardiovascular adaptations to aerobic exercise have been well established (Brooks et al., 2005). At the same submaximal exertion levels, a reduction in HR is offset by an increase in stroke volume while maintaining cardiac output (Brooks et al., 2005). There are several factors can potentially contribute to this phenomena. First is an increase in blood plasma. Acute and chronic endurance exercise has been shown to increase blood plasma volume (Fellmann, 1992; Greenleaf, Sciaraffa, Shvartz, Keil, & Brock, 1981). The onset of this phenomena can happen rapidly but may take up to two days to reach a maximal expansion which can range from 9 to 25% equating to 300 ml to 700 ml (Fellmann, 1992). It has been shown that after even a single supramaximal exercise session [running 15-sec at 95% VIFT (a speed corresponding to 120% of VO2max) interspersed with 15-sec active recovery (45% VIFT) until exhaustion] a 4.8% increase was demonstrated after 48 hours in intermittent sport athletes (Buchheit, Laursen, Al Haddad, & Ahmaidi, 2009). In a second study, investigators examined the effect of multi-day endurance exercise on left ventricular function through 4 days of 3-hour race-simulated cycling (average intensity $51.8 \pm 2.8\%$ W; Oosthuyse, Avidon, Likuwa, & Woodiwiss, 2012). It was shown that on Day 5, resting end-diastolic volume increase as compared to Day 1 prior to exercise (127 ± 23 ml versus 108 ± 25 ml; Oosthuyse et al., 2012).

In a divergent study, cardiac indexes were studied in professional cyclists during the Giro d'Italia 3-week stage race (Corsetti et al., 2012). Mean plasma volume percentage increased from Day 1 to Day 12 by 1.55% and then decreased from Day 12 to Day 22 by -0.72% for a net increase of .99% over the entire race. While there was some fluctuation over the event, these changes didn't reach significance (Corsetti et al., 2012).

Where noted the potential increases in plasma volume assists in enhancing performance though improved muscle perfusion and an increased stroke volume. This offsets the reduced HR

as to maintain or even increase cardiac output through amplified use of the Frank-Starling effect during exercise (Fellmann, 1992).

Autonomic nervous system. Other factors that may contribute to this HR adaptation are changes in autonomic nervous system's response to exercise. It has been established that chronic endurance training decreases HR at submaximal exercise intensities through a reduction in sympathetic activity to the heart (Carter, Banister, & Blaber, 2003; Christensen & Galbo, 1983). The autonomic nervous systems response to an acute exercise bout increases HR with a reduction in parasympathetic activity and a corresponding sympathetic system activation. This sympathetic activation escalates from a threshold level to a maximum as exercise intensity is increased (Farrell, Joyner, Caiozzo, & American College of Sports Medicine., 2012). Though chronic endurance training, adaptation occurs with a reduction in sympathetic activity at the same relative exercise work intensity and through which various mechanisms result in a lower HR (Farrell et al., 2012). Optimal exercise training may also increase cardiac vagal activity as the parasympathetic system maintains collaboration with the sympathetic system (Earnest et al., 2004).

In a meta-analysis examining the effects of training overload on HR and HR variability (HRV), with respect to resting conditions, submaximal, and maximal exercise, it was found that in protocols under two weeks in duration resting HR increased (SMD = 0.55; p = 0.01) as well as LF/HF (SMD = 0.52; p = 0.02; Bosquet, Merkari, Arvisais, & Aubert, 2008). There was also a noted decrease in maximal HR (SMD = -0.75; p = 0.01) in this shorter-term period. Over longer duration protocols, lasting over two weeks, a small decrease in submaximal (SMD = -0.38; p = 0.006) and maximal exercise HR (SMD = -0.33; p = 0.007) were reported (Bosquet et al., 2008).

Another study on the relation between physical exertion HR/HRV was performed on professional cyclist during the tour of Spain (Earnest et al., 2004). Investigators examined resting HR and HRV on the mornings of Day 0, Day 10 (first rest day), and Day 17 (second rest day) of the race (Earnest et al., 2004). HR was also recorded continuously throughout each stage of the race and then categorized into three phases based upon previous VO2max testing (Phase I = light intensity < ventilatory threshold VT ~ 70% VO2max; Phase II = moderate intensity between VT and respiratory compensation point RCP ~ 90% VO2max; Phase III = high intensity > RCP) to determine a TRIMP for each day/stage. Total TRIMPS for Days 1 to 9 were greater than for days 10 to 15. However, TRIMPS/day were less for stages 1 to 9. While there was a trend to decline in resting HR from Day 0 (53.2 ± 1.8 BPM), Day 10 (49.0 ± 2.8 BPM), and Day 17 (48.0 ± 2.6 BPM; $p = 0.21$) no significant group mean changes were found in HR or HRV indices. What was found, was that resting HRV was inversely related to exercise volume (total TRIMPS) and intensity (TRIMPS/day). The authors noted limitations; however, they theorized that the heavy exercise attenuated HRV as HR is inherently regulated by sympathetic and parasympathetic balance (Earnest et al., 2004).

It has been shown that a consistent sympathetic system response to chronic training is in lower levels of plasma norepinephrine and epinephrine at the same absolute work intensity between the trained and untrained state in individuals (Bloom, Johnson, Park, Rennie, & Sulaiman, 1976; Farrell et al., 2012). However, studies on overtraining or overreaching are cases of extreme stress, which may throw normal compensation mechanisms out of balance. In this current case a more rapid adaptation would be needed and since research on the temporal rate of this change is lacking, the research is equivocal at this time.

Mitochondrial activity. Another potential contributing factor for the displayed reduction in HR might be attributable to an upregulation in mitochondrial activity within working muscle tissue. It has been well established that adaption to endurance training causes an increase in mitochondrial mass (Brooks et al., 2005). This does not increase the mitochondrial efficiency but can add more capacity to increase fatty acid β-oxidation and thus reduce overall stress during submaximal exercise (Brooks et al., 2005; Knuiman, Hopman, & Mensink, 2015) This increase in mitochondrial mass may enhance the O2 extraction at the muscles. An increase in maximal arterial-venous difference has been demonstrated after 3 weeks of training in both older and younger men (Murias, Kowalchuk, & Paterson, 2010). Training was performed on a cycle ergometer three times per week for 45 min at 70% VO2max˙with a resulting increase in VO2max (older 31% and younger 18%; $p < 0.05$). It was concluded that for the younger men, 56% of the increase in VO2 max was attributed to a greater Qmax and 44% to a widened a-vO2 difference with early adaptations in the first three weeks predominately relied on a widened maximal a-vO2 difference of 66%. (Murias et al., 2010). This increase in the O2 extraction would allow for faster ATP restoration and thus reduce stress on the cardiovascular system effecting HR over the exhibited 60-second to 180-second aerobic time frames. However, current research on the rate of adaptation indicates it would be difficult to assess the level of contribution of this mechanism to the HR reduction displayed in the current study.

Conclusion

The decline in HR, but not ACC across games, over the 3-day event indicates a potential cardiovascular adaptation as opposed to overt fatigue. While more research on this phenomenon is needed, a potential rapid change in blood plasma volume appears as a likely contributing factor. On the other hand, the reduced ACC from 60 to180 sec (a decline in mean ACCs as high as 6.39%) between Games 1 and 2 in the same day indicates reduced shift capacity and an overall decline in performance indicative of fatigue during the second contest. This has potential implications for youth hockey administrators and coaches who are scheduling competitions for young athletes. Understanding that the timing of games within a competitive event structure does have an impact on performance capacity of the athlete should provide some caution when including multiple games in a day. This is then a cause for concern as fatigue is generally assumed to increase the risk of injury. However the research on this connection is very limited and equivocal (McCall et al., 2015). While this study did not explore the injury aspect, it is certainly worth continued investigation.

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APPENDICES

Appendix A: IRB Approval Letter

EASTERN
MICHIGAN UNIVERSITY

Stephen McGregor <smcgregor@emich.edu>

IRBNet Board Action
1 message

Sonia Chawla <no-reply@irbnet.org>   Tue, Dec 6, 2016 at 4:31 PM
Reply-To: Sonia Chawla <schawlaw@emich.edu>
To: Steve McGregor <smcgregor@emich.edu>, Ken Martel <kenm@usahockey.org>

Please note that Eastern Michigan University Human Subjects Review Committee (UHSRC) has taken the following action on IRBNet:

Project Title: [922618-1] Quantifying Performance Decrements of USA Hockey National Camp Players
Principal Investigator: Ken Martel

Submission Type: Amendment/Modification
Date Submitted: December 1, 2016

Action: APPROVED
Effective Date: December 6, 2016
Review Type: Expedited Review

Should you have any questions you may contact Sonia Chawla at schawlaw@emich.edu.

Thank you,
The IRBNet Support Team

www.irbnet.org

Appendix B: Peak ACC for M1 vs M2

Figure 2. Peak ACC for 90 seconds time frame M1 vs M2 with shaded area 95% CI.

Figure 3. Peak ACC for 180 seconds time frames M1 vs M2 with shaded area 95% CI.

Appendix C: HR for G1 to G4.
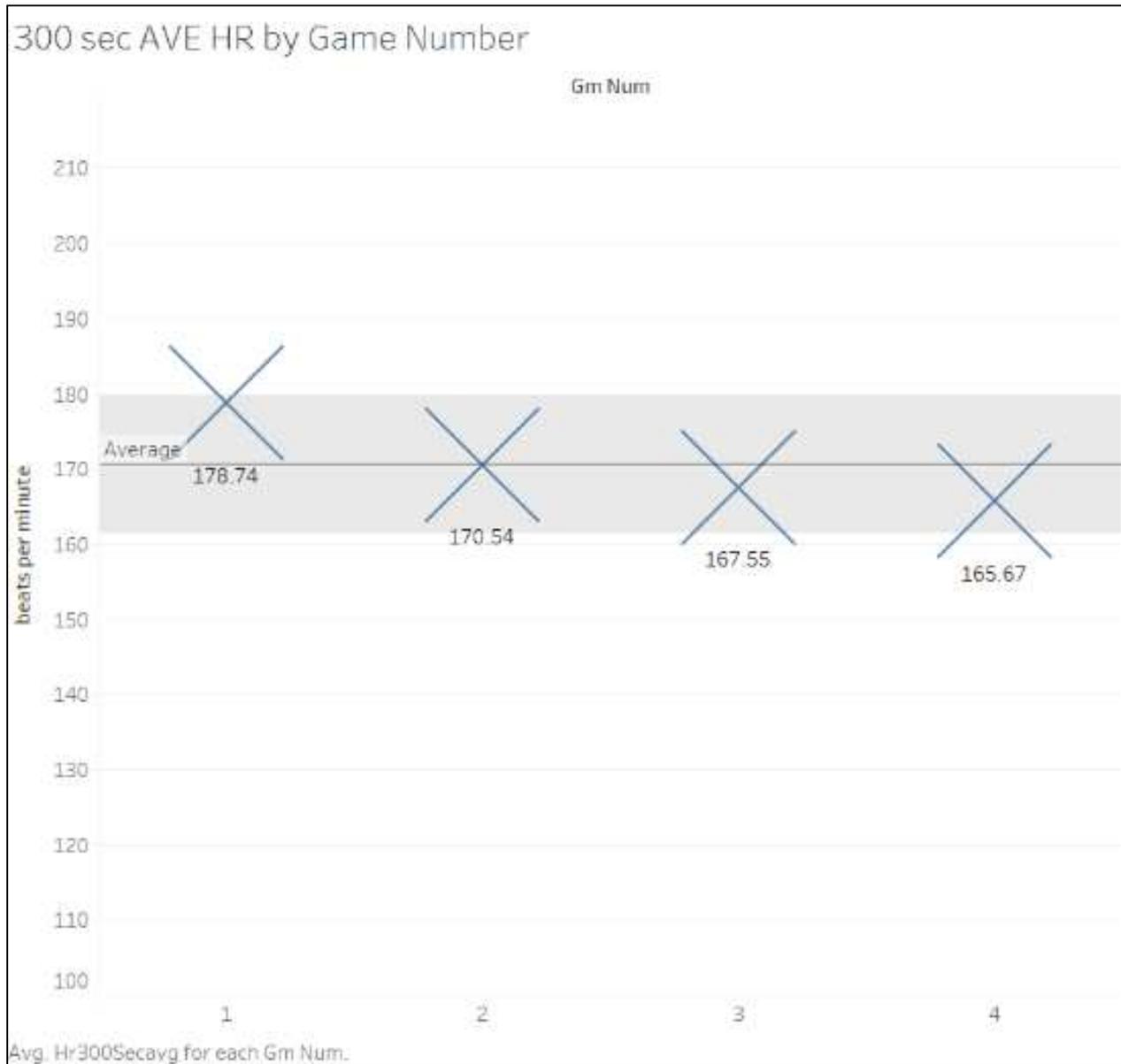
Figure 4. HR for 300 seconds time frame G1 to G4 with shaded area 95% CI.

Figure 5. HR for 600 seconds time frame G1 to G4 with shaded area 95% CI.
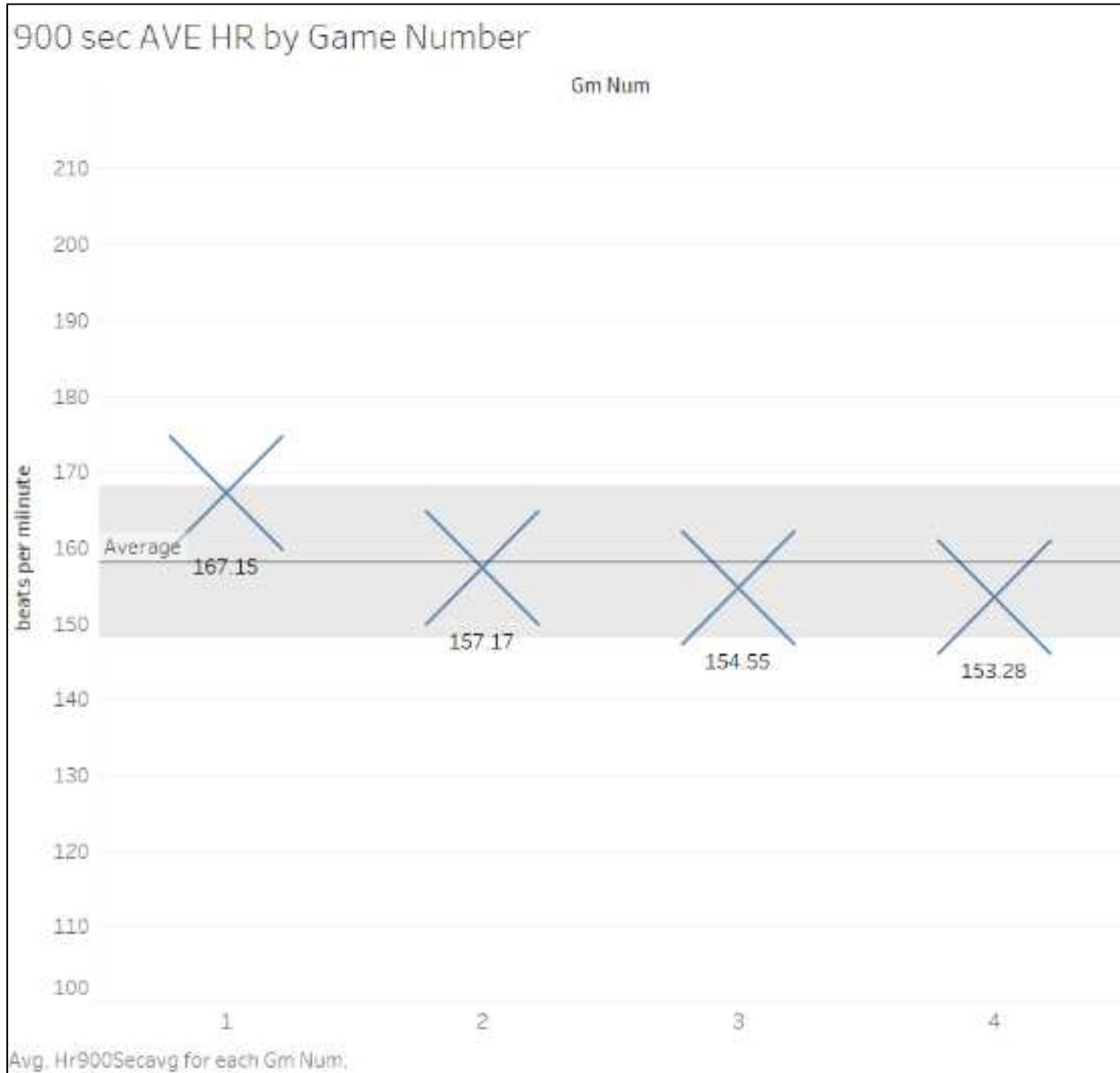
Figure 6. HR for 900 seconds time frame G1 to G4 with shaded area 95% CI.
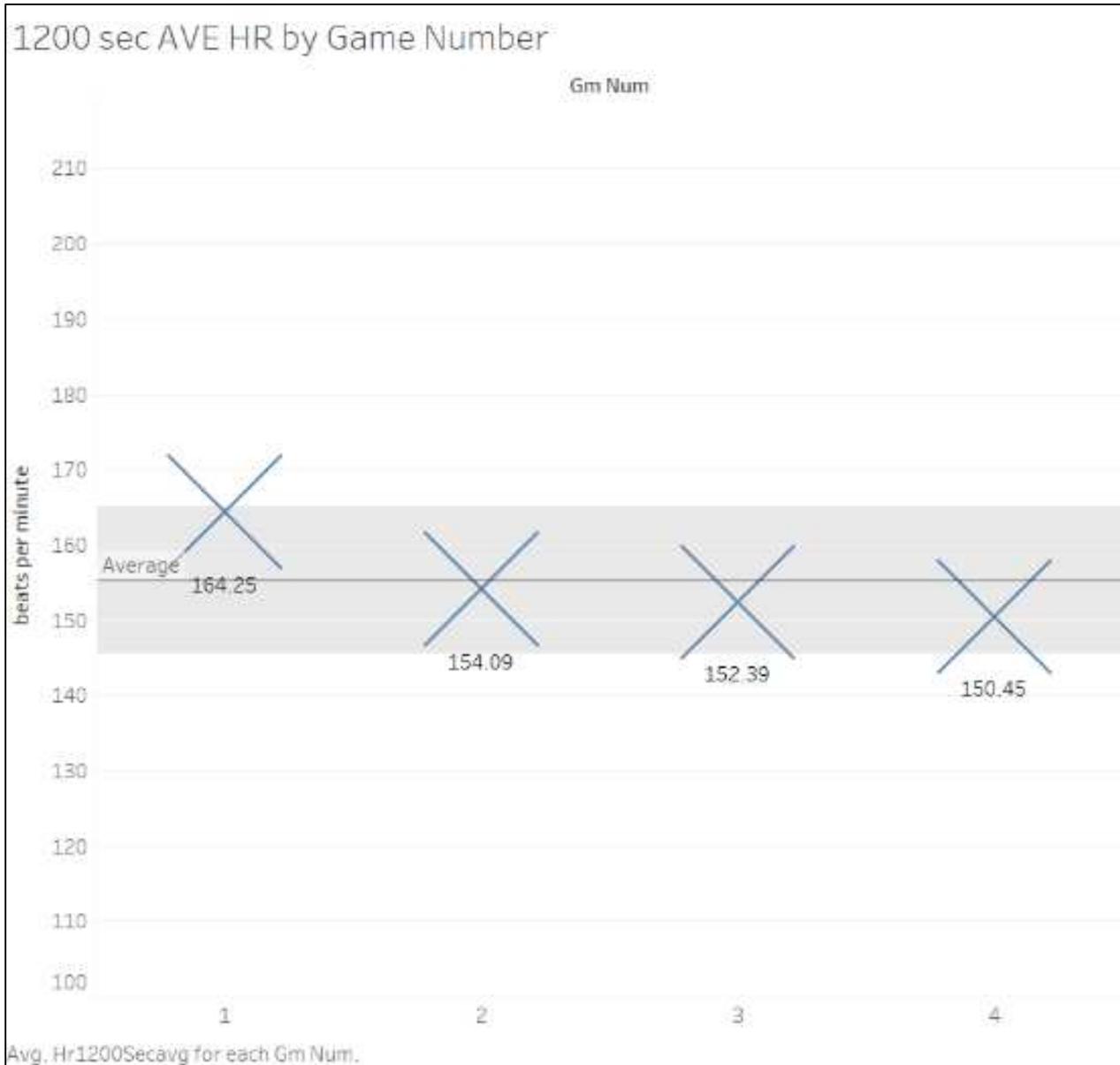
Figure 7. HR for 1,200 seconds time frame G1 to G4 with shaded area 95% CI.
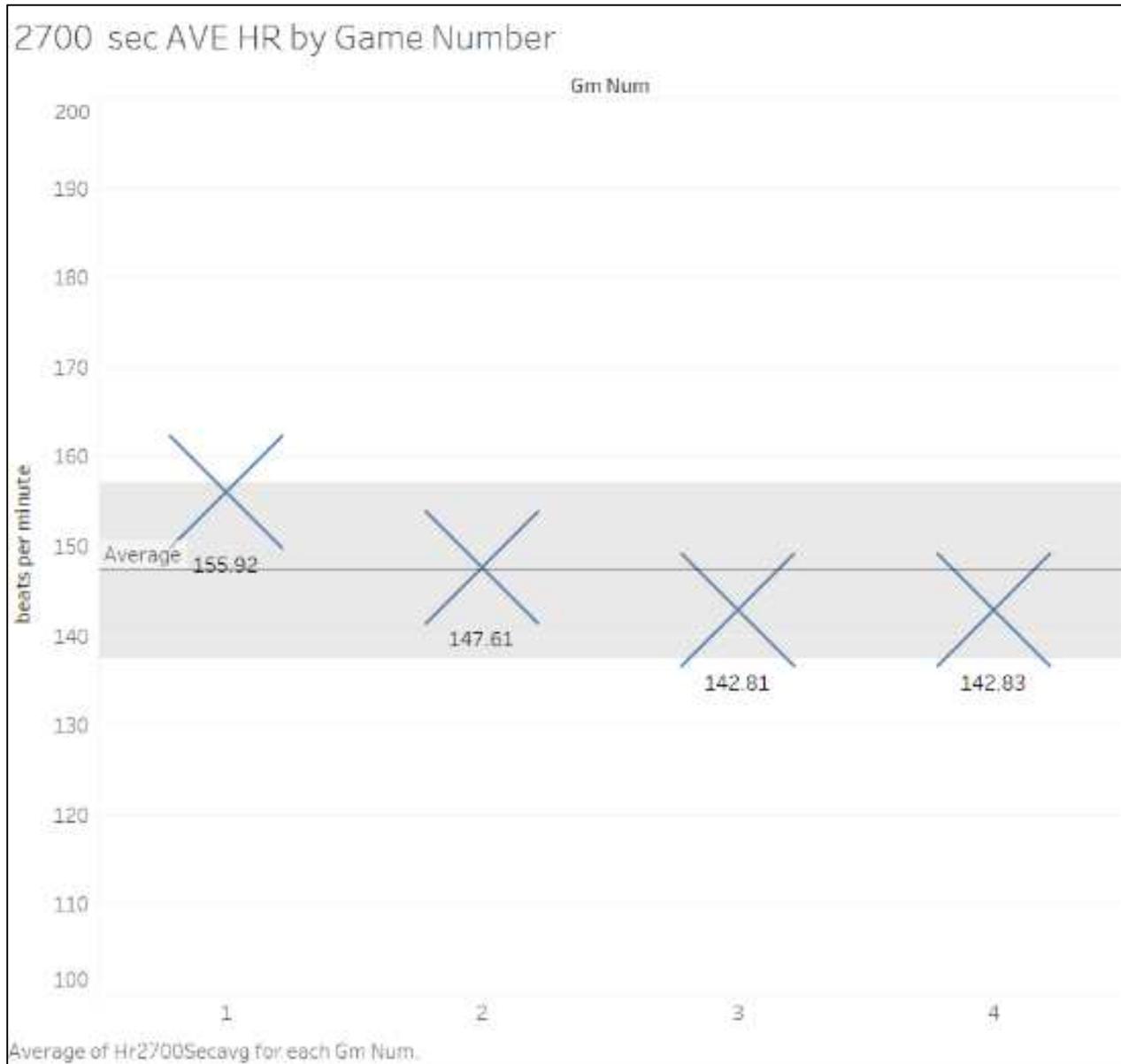
Figure 8. HR for 2,700 seconds time frame G1 to G4 with shaded area 95% CI.