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# The effects of two different recovery postures during high intensity interval training

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**The Effects of Two Different Recovery Postures During High Intensity Interval Training**

By

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Accepted in Partial Completion of the Requirements for the Degree

Master of Science

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## **MASTER'S THESIS**

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Joana Vaya Malinao Houplin

May 16<sup>th</sup> 2014

**The Effects of Two Different Recovery Postures During High Intensity Interval Training**

A Thesis  
Presented to  
The Faculty of  
Western Washington University

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of the Requirements for the Degree  
Master of Science

By  
Joana Vaya Malinao Houplin  
May 2014

### Abstract

The purpose of this study was to examine the effects of two different recovery postures, hands on head (HH) and hands on knees (HK), as a form of immediate recovery from high intensity interval training (HIIT). Furthermore, the study examined whether the two recovery postures influenced subsequent power performance in a Wingate Anaerobic Test. Twenty subjects were included and testing sessions were randomized for each subject. Each subject performed four intervals of 4 minutes of running (4X4) with three minutes of recovery between each running interval. During each three minute recovery interval, measurements recorded included: HRR for the first minute and then volume of carbon dioxide ( $V_{CO_2}$ ) and tidal volume ( $V_T$ ). After the last recovery interval, each subject performed a Wingate Anaerobic Test. The results show improved HRR ( $p < .001$ ) and greater  $V_T$  ( $p = .008$ ) with HK when compared to HH (53 versus 31 beats per minute for HRR and 1.44 versus 1.34 L/minute  $V_T$  respectively). However there was no difference in  $V_{CO_2}$  (1.13 L/min with HK and 1.03 L/min with HH) or subsequent mean power output on the Wingate Anaerobic Test (503 Watts with HK and 498 Watts with HH) between both groups. HK posture may be more beneficial than the popularly advocated HH posture as a form of immediate recovery from HIIT.

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## Chapter 1

### The Problem and Its Scope

#### Introduction

Athletes, from novice to elite, are always looking for strategies to recover faster from exercise to improve their performance. It is well known that the respiratory system is a major limiting factor in exercise performance (Boutellier & Piwko, 1992; Boutellier, Büchel, Kundert, & Spengler, 1992). Oftentimes, during high intensity exercise, the respiratory system is limited because the necessary increases in ventilation to maintain blood gas homeostasis results in an increase in work of breathing which exceeds the capacity of the respiratory musculature to produce sufficient flow and volume, resulting in a limited expiratory ability (Guenette & Sheel, 2007). The respiratory system is also a limiting factor in those who suffer from pulmonary disease such as chronic obstructive lung disease (COPD) (Spengler & Boutellier, 2000).

Researchers have investigated the effects of different postures during recovery from exercise, and the physiological responses in various the postures (Takahashi, Hayano, Okada, Saitoh, & Kamiya, 2005; Takahashi, Okada, Saitoh, Hayano, & Miyamoto, 2000; Taoutaou et al., 1996). Most of the research has focused on evaluating three positions, supine, seated, and upright. The supine position elicits greater cardiac output (CO), stroke volume (SV), and lower extremity peripheral resistance when compared to upright seated posture, leading to faster heart rate recovery (HRR) (Takahashi et al., 2000). Other forms of recovery have also been investigated, comparing active and passive strategies and their influence on intermittent sprint performance and power output (Castagna et al., 2008; Connolly, Brennan, & Lauzon, 2003; Graham, Douglas Boatwright, Hunskor, & Howell, 2003; Taoutaou et al., 1996). It was suggested that active recovery is superior to passive recovery in improving subsequent

performance (Connolly et al., 2003). Upright standing posture is more commonly used in the exercise and sports field setting (Buchheit, Al Haddad, Laursen, & Ahmaidi, 2009). However the effects of upright standing and immediate recovery between sets of exercise have not been as well examined. It is unknown whether upright standing posture with hands on head (HH), traditionally advocated by coaches in the field setting is the most advantage posture strategy for immediate recovery from exercise. The use of a non-traditional posture strategy of hands on knee (HK) may benefit the respiratory muscles to act more effectively and may also influence heart rate recovery (HRR) response. The non-traditional HK is a strategy commonly used in the clinical rehabilitation settings for individuals with COPD to help relieve feelings of dyspnea (Banzett, Topulos, Leith, & Nations, 1988; O'Neill & McCarthy, 1983).

The HK strategy focuses on thoracic spinal flexion and upper extremity support. Thoracic spine motion significantly influences rib cage mechanics (Lee, 1993) which in turn influences the position of the diaphragm (Goldman & Mead, 1973; Mead, 1979). The zone of apposition (ZOA) is the mechanical linkage between the diaphragm and rib cage (Boynton, Barnas, Dadmun, & Fredberg, 1991; Mead, 1979). The ZOA accounts for a substantial amount but variable fraction of total surface area of the rib cage. At residual volume (RV), it accounts for more than one half of the total surface and decreases to zero at total lung capacity (Mead, 1979). It has been suggested that the ZOA is less influenced by the height of the diaphragm but rather the orientation of the rib cage (Hruska, unpublished manuscript). Optimizing the ZOA is done by increasing the surface area of the diaphragm apposed to the lower rib cage thus optimizing the piston-like axial displacement of the dome to the apposed muscle fibers (De Troyer & Estenne, 1988). Maximizing the surface area of the ZOA is observed during spinal flexion and is decreased during spinal extension (Hruska, unpublished manuscript). Flexion of the thoracic

spine induces anterior rotation of the ribs so that the anterior aspect moves inferiorly and the posterior aspect moves superiorly (Lee, 1993). It has been suggested that anterior rotation of the rib cage (synonymous with internal rotation) increases ZOA whereas externally rotation of the rib (associated with thoracic spinal extension) decreases the ZOA (Hruska, unpublished manuscript).

The ZOA is anatomically important because it influences diaphragmatic tension, which can only be accomplished by proper opposition from the abdominal muscles (Hruska, 1997). The abdominal muscles which include the external oblique abdominis, internal oblique abdominis, rectus abdominus and transverse abdominus, along with the internal intercostals and triangularis sterni are active during expiration and also aid during inspiration due to the improved length-tension relationship of the diaphragm during expiration (Roussos, 1985).

The ability to buffer metabolic by-products of exercise, including hydrogen ions ( $H^+$ ) and carbon dioxide ( $CO_2$ ) is crucial in maintaining acid-base homeostasis during exercise (Stringer, Casaburi, & Wasserman, 1992). It is well known that failure to maintain acid-base homeostasis during exercise can have detrimental effects on performance (Costill, Verstappen, Kuipers, Janssen, & Fink, 1984; Powers & Howley, 2009). Pulmonary ventilation plays an essential role in maintaining acid-base homeostasis by regulating blood acidity (pH) with expiration of  $CO_2$  by the lungs (Powers & Howley, 2009; Stringer, 1992). The ability to expire additional  $CO_2$  produced from high intensity exercise suggests an increase in ventilation thereby maintaining blood pH (Stringer et al., 1992). An increase in ventilation to expire additional  $CO_2$  during the recovery phase of exercise in the different postures may show a more efficient use of respiratory musculature in the specific recovery posture.

HRR after exercise has also been used to assess autonomic function in those who suffer from cardiovascular disease. A decrease in HRR is a strong indicator of mortality (Borresen & Lambert, 2008; Cole, Blackstone, Pashkow, Snader, & Lauer, 1999). It has also been suggested to be a valuable tool in monitoring an athlete's training status and their response to certain training stresses (Buchheit, Laursen, & Ahmaidi, 2007; Buchheit, 2006; Imai et al., 1994; Javorka, Zila, Balhárek, & Javorka, 2002; Lamberts, Swart, Capostagno, Noakes, & Lambert, 2009; Yamamoto, Miyachi, Saitoh, Yoshioka, & Onodera, 2001). A faster HRR has been observed as a result of endurance training (Yamamoto et al., 2001) whereas a delayed HRR results in impaired performance related to fatigue (Lamberts et al., 2009). However, it is unknown if accelerated HRR during repeated work to rest transitions in exercise has an effect on subsequent performance in endurance trained subjects.

### **Purpose of the Study**

This study was conducted to determine the effect of using two different recovery postures of standing, one with hands on head and with hands on knees. The hands on head posture required subjects to stand erect with their hands clasped on top of their head. The hands on knees posture required subjects to be flexed at both the hip and knee joint, supporting their trunk by bracing on their thighs. Subjects were also instructed to maintain a flexed spine in this posture which was measured with inclinometers to ensure consistency of thoracic flexion between subjects and rest periods. The study focused on observing minute ventilation ( $V_E$ ), breathing frequency ( $f_b$ ), carbon dioxide elimination ( $VCO_2$ ) and heart rate recovery (HRR) during the recovery intervals of high intensity interval training (HIIT).  $V_E$  and  $f_b$  were then used to calculate tidal volume ( $V_T$ ). To further examine the effects of the different postures on subsequent

performance, a Wingate Anaerobic Test was performed after the last rest interval to measure mean anaerobic power.

### **Statement of the Null Hypothesis**

The null hypothesis states that there is no effect of the recovery postures of hands on head (HH) and hands on knees (HK) during the recovery period of high intensity interval training (HIIT) on heart rate recovery (HRR), carbon dioxide elimination ( $V_{CO_2}$ ) and tidal volume ( $V_T$ ). In addition, there is also no effect of the recovery postures on a subsequent Wingate Anaerobic Test of power.

### **Significance of the Study**

During high intensity endurance exercises, respiratory muscles may fatigue in highly trained endurance athletes (Babcock, Pegelow, Johnson, & Dempsey, 1996). The ability to effectively use those respiratory muscles during the recovery phase of exercise may improve pulmonary ventilation. An increase in ventilation is known to play a crucial role in the removal of metabolic by-products of exercise like  $CO_2$  and  $H^+$  which have negative effects on performance (Powers & Howley, 2009; Stringer, 1992). Also, the ability to improve heart rate responses by accelerating heart rate recovery (HRR) during the rest phase of exercise may positively influence subsequent performance.

Although many studies have investigated the effects of recovery postures like seated and supine positions after exercise, and also passive and active recovery on repeated sprint ability, none have looked at the effects of upright standing postures like HH or a HK posture. There have not been any studies that have examined the effects of HH or HK as a strategy for immediate recovery between repeated work bouts. The current study will determine if HH or HK posture influences respiratory efficiency and HRR during the recovery period of HIIT and subsequent

anaerobic power performance during a Wingate Anaerobic Test in Division II female soccer athletes.

Endurance sports like soccer are physiologically demanding and require energy from both aerobic and anaerobic energy systems. It has been estimated that elite players during a competitive soccer match can cover a distance of 10-12 kilometers (km) at an intensity that is 80-90% of maximum heart rate (Bangsbo, Norregaard, & Thorso, 1991; Helgerud, Engen, Wisloff, & Hoff, 2001) requiring high demand from the aerobic system (Bangsbo, 1994). It has been observed that HIIT consisting of four sets of 4 minutes of work at 90-95% of heart rate max with a three minute recovery significantly improves maximum oxygen uptake ( $VO_{2max}$ ) of soccer players (Helgerud, 2007; McMillan, Helgerud, Macdonald, & Hoff, 2005). It has also been reported that elite soccer players perform 150-250 short bouts of high intensity actions during a game (Mohr, Krstrup, & Bangsbo, 2003) which rely more on the anaerobic system (Bangsbo, 1994, and Krstrup, 2006). The ability to perform high intensity bouts of work in soccer is crucial to game performance. The current study will determine if different posture strategies have an influence on cardiorespiratory mechanisms during the recovery period of HIIT. Additionally, this study examined the effects of the recovery postures on a subsequent performance of anaerobic power in a Wingate Anaerobic Test. The results will be important in determining the benefits of HH or HK as a strategy for immediate recovery during high intensity activity and its influence on a subsequent power performance.

### **Limitations of the Study**

1. Subjects in this study were all female soccer players and results are not generalized to other subject populations.
2. It was encouraged that all subjects included in the study completed all testing measures with the best of their ability and that maximal effort was given during the Wingate Anaerobic Test.
3. Fitness levels of subjects varied. Subjects were not asked to change any training regimens during the study. This was difficult to control in all subjects.
4. The testing was performed on a treadmill. Soccer players do not usually train on treadmills so performance on a treadmill may differ from actual field performance.
5. The Wingate Anaerobic Test for power may not directly reflect actual physiological capabilities of soccer players.

### **Definition of Terms**

Body plethysmography: The measurement of intrathoracic gas volume and body volume changes (Goldman, 2005).

Dyspnea: Difficult or labored breathing (Marieb, 2004).

Expiration: The exhalation of air from the lungs. During normal, quiet breathing is considered passive and is considered active during high intensity exercise and hyperventilation (Powers & Howley, 2009).

Expiratory Muscles: Include the external oblique abdominis, internal oblique abdominis, rectus abdominus, transverse abdominus, internal intercostals, and triangularis sterni (De Troyer & Estenne, 1988; Kera & Maruyama, 2005; Hudson, 2010).



Expiratory Reserve Volume (ERV): Volume of gas that can be maximally exhaled from the end-expiratory level during tidal breathing (Wanger, 2005).

Fatigue: A loss in the capacity to develop force and/or velocity of a muscle, resulting from muscle activity under load and which is reversible by rest (NHLBI, 1990).

Functional Residual Capacity (FRC): Volume of gas present in the lung at end expiration during tidal breathing (Wanger, 2005).

Heart Rate Recovery (HRR): The immediate decrease in heart rate after exercise and can be defined as the absolute difference in heart rate immediately after exercise and 60 seconds later (Cole et al., 1999).

Inspiration: The inhalation of air into the lungs, where the respiratory diaphragm is the major muscle of inspiration (Powers & Howley, 2009).

Inspiratory Muscles: Include the respiratory diaphragm, external intercostals, scalenes and sternocleidomastoid (Guenette & Sheel, 2007; Hudson, 2010; Roussos, 1985).

Inspiratory Reserve Volume (IRV): Amount of air in the lungs after a full inhalation (Marieb, 2004).

Maximal Inspiratory Pressure (MIP): The highest atmospheric pressure developed during inspiration against an occluded airway (Marieb, 2004).

Maximum Voluntary Ventilation (MVV): Measurement of ventilatory capacity, which requires rapid, deep breathing for 15 seconds, then extrapolated and reported as liters per minute (Marieb, 2004).

Mean Anaerobic Power: Represents the average power throughout 30 seconds and metabolically correlates with the power of anaerobic glycolysis (Popadic Gacesa, Barak, & Grujic, 2009).

Minute Ventilation ( $V_E$ ): Volume of air breathed each minute (Marieb, 2004).

Ratings of Perceived Exertion (RPE): A scale used in monitoring an individual's perception of effort during physical activity (Borg, 1982).

Respiratory Muscles: Includes both inspiratory and expiratory muscles, which include the diaphragm, external intercostals, scalenes, sternocleidomastoid, internal intercostals, external oblique abdominis, internal oblique abdominis, rectus abdominus, triangularis sterni and transverse abdominus (Guenette & Sheel, 2007; Kera & Maruyama, 2005; Roussos, 1985).

Respiratory Sinus Arrhythmia (RSA): RSA is a beat to beat interval variation in heart rate which is influenced by respiration (Hayano, Yasuma, Okada, Mukai, & Fujinami, 1996; Hirsch & Bishop, 1981).

Spirometer: Measures inspired and expired lung volumes (Wanger, 2005)

Tidal Volume ( $V_T$ ): The amount of air moved during either inspiration or expiration of each cycle of breath (Marieb, 2004).

Total Lung Capacity (TLC): The amount of air in the lungs after a maximum inspiration, and is also the sum of vital capacity and residual volume (Powers & Howley, 2009).

Vital Capacity (VC): Amount of air volume moved in one breath from full inspiration to maximum expiration (Marieb, 2004).

Wingate Anaerobic Test: Measures anaerobic power and consists of a 30 second all out exhaustive ergometer test where the subject pedals against a resistance relative to subject's body weight (Zupan et al., 2009).

Zone of Apposition: Anatomical relationship between the respiratory diaphragm and the rib cage, influencing the respiratory diaphragm's mechanical action and respiratory advantage.

## Chapter II

### Review of the Literature

#### Introduction

The respiratory system is a major limiting factor in high intensity, endurance exercises in both trained and untrained subjects (Boutellier & Piwko, 1992; Boutellier, 1992). The respiratory muscles during high intensity endurance activity fatigue and cause a decrease in performance (Babcock, 1996; U. Boutellier & Piwko, 1992; U. Boutellier et al., 1992; Guenette & Sheel, 2007). Respiratory muscle fatigue is especially observed in those who suffer from pulmonary dysfunction like chronic obstructive pulmonary disease (COPD) (Banzett, 1988; Probst, 2004; Solway, Brooks, Lau, & Goldstein, 2002). Those who suffer from COPD report feelings of dyspnea during daily activities, especially activities that involve the use of upper extremities (Couser, Martinez, & Celli, 1992; Dolmage, Maestro, Avendano, & Goldstein, 1993). Several authors have reported that the use of a rollator device or bracing of arms on the knees or a solid object relieves the feeling of breathlessness or dyspnea (Banzett, 1988; Cavalheri, 2010; Kera & Maruyama, 2005; O'Neill & McCarthy, 1983; Probst, 2004; Solway, 2002). However, there is no evidence that this strategy of bracing arms on the knees will improve feelings of dyspnea or respiratory efficiency in subjects without COPD.

The purpose of this chapter is to review the literature relating the respiratory system as a limiting factor in performance and how it can be influenced by changes in body posture. The first section focuses is on the respiratory muscles and how they become fatigued during high intensity endurance exercises. It also examines the zone of apposition (ZOA) of the diaphragm to the rib cage. The next section presents ventilatory and metabolic responses to changes in body position, focusing on upper extremity elevation. The following section focus is on the effect of different

strategies to relieve dyspnea in a population who suffer from respiratory dysfunction. The next section presents the relationship between respiration and heart rate (HR) and extends to further focus on heart rate recovery (HRR) and its use as a training tool to monitor athletes. The following section then focuses on the use of high intensity interval training (HIIT) in soccer players. The last section presents the use of the Wingate Anaerobic Test in athletes, focusing on soccer players.

**Respiratory muscles and fatigue.** The diaphragm is the predominant muscle during quiet breathing and accessory muscles, including intercostals and scalenes, become more active during exercise to assist during inspiration (Guenette & Sheel, 2007; Roussos, 1985). Abdominal muscles, which include the external oblique abdominis, internal oblique abdominis, rectus abdominus and transverse abdominus, along with the internal intercostals and triangularis sterni are active during expiration also aid during inspiration due to the improved length-tension relationship of the diaphragm during expiration (De Troyer & Estenne, 1988; Roussos, 1985). The abdominal muscles store elastic energy in the chest wall during expiration which then recoil the stored energy during inspiration (Kera & Maruyama, 2005; Roussos, 1985). The abdominal muscles also play an important role in controlling rib cage position which in turn influences diaphragmatic tension and the ZOA (Hruska, 1997). Appropriate diaphragmatic tension is necessary for respiratory efficiency (De Troyer & Estenne, 1988). The ZOA is the mechanical linkage between the diaphragm and rib cage and is crucial in optimizing mechanical efficiency of the respiratory musculature (Boynton et al., 1991; Hruska, 1997; Mead, 1979). The abdominal muscles oppose the diaphragm during contraction and provide resistance for displacement and rib cage expansion (De Troyer & Estenne, 1988; Hruska, 1997). The opposition of the abdominal muscles on the diaphragm maximizes the ZOA (Hruska, 1997).

Like any other skeletal muscle, respiratory muscles are susceptible to fatigue during high intensity activity (Guenette & Sheel, 2007; Johnson, Babcock, Suman, & Dempsey, 1993; Romer & Polkey, 2008; Roussos, 1985). According to the National Heart Lung, and Blood Institute (1990), fatigue is a reduction in force-generating capacity of the muscle, resulting from muscle activity under load which is reversible by rest. Respiratory muscle fatigue is seen in highly trained subjects, normal subjects, and sedentary subjects, (Babcock, 1996; Boutellier, 1992; Johnson, 1993).

As exercise intensity increases, respiratory muscles must be able to meet the demands needed to sustain sufficient energy to produce force (Guenette & Sheel, 2007; Macklem, 1980). However, these increases in respiratory muscle demands can only be sustained depending on the amount of energy supply available (Macklem, 1980). Oftentimes, during high intensity exercise, the respiratory system is limited because the necessary increases in ventilation to maintain blood gas homeostasis results in an increase in work of breathing. Thus, the increased work capacity of the respiratory musculature (lung & chest wall) increases to produce adequate flow and volume results in limited expiratory ability (Guenette & Sheel, 2007). Limited expiratory flow is suggested to cause diaphragm fatigue. The fatigue leads to competition of blood flow to respiratory and locomotor muscles and also increasing demands from accessory respiratory muscles (Harms et al., 1997).

It is difficult to accurately assess diaphragmatic fatigue due to the muscle and motor nerves location which are to some extent inaccessible (Romer & Polkey, 2008). However, estimations of force development of the diaphragm (transdiaphragmatic pressure) are measured by taking the differences of gastric and esophageal pressures induced by stimulation of both phrenic nerves (Romer & Polkey, 2008). A similar method is used to measure abdominal muscle

force by estimating the gastric pressure response to magnetic stimulation of the thoracic nerve (Kyroussis et al., 1996). Application of these techniques has been widely used to examine respiratory fatigue in humans (Aubier, Farkas, De Troyer, Mozes, & Roussos, 1981; Kyroussis et al., 1996; Romer & Polkey, 2008).

Johnson et al. (1993) measured diaphragmatic fatigue using the technique of supramaximal bilateral phrenic nerve stimulation (BPNS) in 12 male subjects varying in fitness levels from sedentary to highly trained. Diaphragmatic twitches were measured according to contraction and relaxation time of the diaphragm. Prior to and after exercise testing, diaphragm muscle force was assessed by performing maximal inspiratory maneuvers against an obstructed airway. The subject's first exercise test was used to determine  $VO_{2max}$ ; ten subjects ran on a treadmill and two exercised on a stationary bicycle. The subjects performed a light warm up and then were quickly brought up to high work intensity of 90-95% or 80-85% of their  $VO_{2max}$  which was maintained until exhaustion. Immediately after exhausting exercise, diaphragm tests were performed again following identical protocols of pre-exercise testing. Results show an increase in ventilation rate ( $46 \pm 7\%$  at 95% work load and  $41 \pm 9\%$  at 85% work load) which was due to an increase in breathing frequency ( $f_b$ ) at a constant tidal volume ( $V_T$ ). At higher work intensities, there was a positive relationship (a larger fall in the time integral of transdiaphragm pressures during exercise, a smaller fall after exercise during stimulation) between diaphragm fatigue and changes in diaphragm work over the duration of the exercise. The variability in work intensities over the final minutes ranged from 60-112% of  $VO_{2max}$  but the prevalence of diaphragm fatigue increased when intensity surpassed 85% of  $VO_{2max}$ . During conditions of heavy endurance exercise, the diaphragm and other inspiratory muscles must be able to generate and maintain adequate rates of breathing at high percentages of available inspiratory muscle

pressure generation. Fatigue occurs when the diaphragm can no longer sustain adequate force development needed to maintain hypernea. Results of this study indicate that diaphragmatic fatigue occurs during heavy endurance exercise and can affect those of varying fitness levels.

To further investigate whether diaphragmatic fatigue is the factor limiting performance, Boutellier et al. (1992) investigated the respiratory system as an exercise limiting factor in normal endurance trained subjects. The subjects underwent 4 weeks of respiratory training five times a week for 30 minutes a day. After initial familiarization with the equipment and devices, vital capacity (VC), peak flow, forced expired volume in 1 second ( $FEV_1$ ), maximal voluntary ventilation (MVV), and breathing endurance were measured. Breathing endurance was measured by voluntary breathing with individually adjusted frequencies between 42-48  $\text{min}^{-1}$  and  $V_T$  2.50-3.25 L/min, assuring that exhaustion occurred within 10 minutes. Training was increased either by fb or  $V_T$ . Once the subject was able to follow the set ventilation for 30 minutes (determined by breathing endurance test), the increase was set for the next day in order to train the respiratory muscles hard. After 4 weeks of training, the subjects improved breathing endurance from 6.1 minutes to 40 minutes, their cycling endurance at anaerobic threshold by 38% (anaerobic threshold remained the same), and decreased minute ventilation (117 to 103  $\text{l min}^{-1}$ ) during the endurance test at any given intensity. The subjects also reported that respiratory training reduced and even eliminated the feeling of breathlessness, even during the hardest part of the tests. The increase in average speed due to reductions and elimination of breathlessness and hyperventilation after respiratory training further supports that the respiratory system is a limiting factor in exercise (Boutellier et al., 1992).

Boutellier and Piwko (1992) conducted research similar to the previous study but instead used healthy, sedentary subjects to investigate if an untrained respiratory system can limit

endurance exercise. In order to determine if the respiratory system was the limiting factor in normal sedentary subjects, the subjects performed a breathing endurance test and a cycle test to exhaustion. The breathing test was determined by subjects maintaining a respiratory rate of 45 breaths/minute at about 60-66% of their VC and was stopped when subjects could no longer maintain preset respiratory rate. The cycle test required subjects to exercise at an intensity of 80% of their physical working capacity (PWC) which is described as the mechanical power developed in cycling at 170 beats per minute. Because there is a linear relationship between heart rate and exercise intensity, PWC can be extrapolated from submaximal exercise intensities (Boutellier & Piwko, 1992). After baseline measurements were taken, subjects performed respiratory training for 4 weeks which required breathing for 30 minutes a day at 50-53% of their vital capacity five times a week, and increased  $f_b$  each week (1 breath / min). After the 4 weeks of training, breathing and cycling tests were repeated. Results showed that breathing endurance increased by 268% and cycle endurance increased by 50% after the training period. Therefore, in both studies, the observed increases in performance after training supports that the respiratory system can improve performance during exercise in both trained and sedentary individuals.

In the previous studies, normal trained and sedentary subjects were assessed and both groups were limited by their respiratory system associated with respiratory muscle fatigue. Some have debated that highly trained athletes may be less susceptible to respiratory muscle fatigue, namely diaphragmatic fatigue, due to their high intensity and volume of training (Coast, Clifford, Henrich, Stray-Gunderson, & Johnson, 1990). This idea was investigated by Babcock et al. (1996) who hypothesized that subjects with greater aerobic capacity will be protected completely or at least partially from exercise-induced diaphragm fatigue. Twenty-four subjects performed pulmonary function tests and exercise testing. A maximal treadmill test was used to split the



subjects into two groups, highly fit or fit, based off  $VO_{2max}$  values. Bilateral phrenic nerve stimulation (BPNS) was used to assess diaphragmatic fatigue throughout the exercise testing. The data showed that diaphragm force output decreased in a similar pattern in those highly fit individuals and those who were considered not as fit ( $-23.1 \pm 3.1\%$  and  $-23.8 \pm 3.8\%$ , respectively). This suggests that highly fit individuals with high aerobic capacity are not protected from exercise induced diaphragm fatigue (Babcock et al., 1996).

It was also observed that respiratory pattern of muscle recruitment during heavy endurance exercise may be affected by an early onset of diaphragm fatigue, causing increased recruitment of accessory (inspiratory and expiratory) muscles (Johnson et al., 1993). The study previously mentioned by Johnson et al (1993) reported that at rest and from the beginning of the fifth to the tenth minute of each exercise period (until volatile exhaustion) transdiaphragmatic pressures plateaued for the remaining exercise period, while ventilation and inspiratory flow rate continued to rise throughout the exercise until termination. This suggests that the relative contribution of diaphragm is less. Therefore, an increased reliance is required on accessory muscles in producing hyperventilatory responses throughout the remainder of the exercise. The findings of the study suggest that high intensity exercise greater than 85% of  $VO_{2max}$  elicits diaphragm fatigue which results in an increased reliance on accessory muscles thereby altering respiratory muscle recruitment. The consequences of respiratory muscle fatigue is a major limiting factor in high intensity exercise performance that lead to inadequate ventilation, an increased work of breathing, and altered breathing mechanics (Boutellier, 1992; Romer & Polkey, 2008).

**Ventilatory and metabolic response to arm elevation.** During high intensity exercise, the respiratory muscles may become fatigued (Babcock, 1996; Johnson, 1993). Some of these

respiratory muscles also act as postural muscles and essentially have two functional roles (Duron, 1973; Hudson, 2010). Hudson et al. (2010) investigated the interplay between the inspiratory and postural functions of the human parasternal intercostal muscles. It was hypothesized that the parasternal intercostals on the right side contract during rotation of the trunk to the right but are not active during rotation to the left. Six healthy subjects were used for the study. Surface and indwelling electromyography (EMG) was used to record activity of the parasternal muscles and neural drive during isometric rotations of the trunk in both the right and left direction. Subjects also breathed through a mouth piece connected to a pneumotachograph to show changes in lung volume. EMG activity was recorded during a period of quiet breathing in a neutral position and when instructed made an isometric contraction against a loaded cell below the clavicle. Once rotation was established, subjects were instructed to hold isometric contraction for 20-30 seconds while maintaining quiet breathing. The subjects performed a total of 120 ipsilateral rotations and 123 contralateral rotations. Motor unit activity of the parasternal intercostal muscles during breathing in the neutral position, ipsilateral and contralateral rotation, and breathing with trunk rotated, showed that 91% of these motor units were active exclusively during inspiration. Motor units that were active in both neutral and rotated breaths during ipsilateral rotation increased from 11.0 Hz to 14.3 Hz respectively. These results support the study's hypothesis which stated that the parasternal intercostals on the right side contract during rotation of the trunk to the right but are not active during rotation to the left. In addition, it was also recognized that motor units that are active during rotation of the trunk are also active in the inspiratory phase of respiration. More surprisingly, the parasternal intercostals during rotation of the trunk caused changes in muscle activation during inspiration. These observations support that the parasternal intercostals not only contribute to inspiratory functions during respiration but also

function as postural muscles. Another muscle that also contributes to both respiratory and postural function is the diaphragm. The primary function of the diaphragm is for respiration, but it also contributes to spinal stability (Hodges, Butler, McKenzie, & Gandevia, 1997; Hodges, Heijnen, & Gandevia, 2001). However, when respiratory demands increase, the postural role of the diaphragm decreases and may contribute to spinal instability during strenuous exercise (Hodges et al., 2001).

To further investigate this idea of respiratory and postural muscle interplay, a majority of the literature has focused on ventilatory response to arm elevation of patients with COPD. This special population has been observed to have low tolerance to arm activities. Their ability to tolerate arm exercises is not only determined by upper body strength or endurance but the influence of arm position itself may play an important role (Dolmage et al., 1993).

In order to determine the effects of arm positioning and ventilatory response in COPD patients, Dolmage et al. (1993) conducted a study to observe changes in arm position at rest and during leg exercise with associated changes in ventilatory response. Three different protocols were used to see the effects of arm position on lung volume, ventilation, and ventilation during exercise. Body plethysmography was used to assess vital capacity (VC) and functional residual capacity (FRC) of each subject while seated and arms either resting on their lap or elevated with hands clasped on their head. To determine the effect of arm position on ventilation, each subject sat on a straight backed chair. The control position required subjects to rest their arms on the chair arms. The unsupported arm elevation (UAE) required subjects to flex their elbow 90° at shoulder level (surrender position). The supported arm elevation (SAE) had a similar position to the UAE but was supported by a customized sling that served to counteract the effects of gravity. On a separate day, subjects underwent leg exercise on an electric cycle ergometer with the same

protocols used during resting conditions. Subjects exercised for 4 minutes at 50% of maximum workload with a 5 minute rest between arm positions. The results of arm elevation on static lung volumes demonstrated that during arm elevation there was a small but significant decrease in VC from 2.64 liters (L) with arms down to 2.44 L. With arms elevated, a small increase in FRC occurred but was not significant. When examining the effect of arm position on ventilation at rest, there was no change during SAE in  $\text{VO}_2$ , maximum carbon dioxide production ( $\text{VCO}_2$ ), and minute ventilation ( $\text{V}_E$ ) when compared to the control. There was a significant increase in  $f_b$  in breaths per minute (16.2 control, 17.9 SAE, and 17.5 UAE) and a decrease in tidal volume ( $\text{V}_T$ ) when comparing the control to SAE (579 ml control, 533 ml SAE and 694 ml UAE). However, during UAE there was a significant increase in  $\text{VO}_2$  (227 to 308  $\text{L min}^{-1}$ ),  $\text{VCO}_2$  (195 to 263  $\text{L min}^{-1}$ ),  $\text{V}_E$  (9 to 11.9  $\text{L min}^{-1}$ ), and  $f_b$  (16.2 to 17.5) when compared to the control position. The effects of arm position on ventilation during exercise illustrated similar results as during rest but with increased extent of ventilatory response.

It was also reported that subjects experienced greater shortness of breath (SOB) during UAE (4.4) when compared to SAE (2.9) and control position (3.1), measured with a Borg scale. Elevating the arms causes some muscles like the pectorals to stretch causing passive expansion of the rib cage resulting in shortening the neck accessory muscles. In doing this, there is a decreased ability of these muscles to generate force which in turn hinders their contribution to inspiratory volume (Dolmage et al., 1993). This study also included a protocol where the arms were elevated and supported by a customized sling to negate the effects of gravity that may increase metabolic activity related to arm elevation to maintain that position. The results indicate that when arms are elevated and supported,  $\text{V}_E$  was similar to the control position, however a more rapid and shallow pattern of breathing was still observed. When arms are elevated, it is

suggested that the contribution of the muscles attached to the thoracic cage that assist in respiration are dampened by the same muscles contributing to its postural maintenance. Exercise with arms elevated shortens accessory breathing muscles, decreasing their contribution to rib elevation during inhalation. In addition, there is also a passive stretching of the thoracic cage, which does not provide an advantage to meeting the increased ventilatory demands (Dolmage et al., 1993).

A similar study by Martinez, Couser, & Celli (1991) examined the respiratory response to arm elevation (AE) in patients with Chronic Airflow Obstruction (CAO). Twenty subjects were assessed and studied in the sitting position with the back supported at a 90° angle either with AE or arms down (AD) for two minutes. Pulmonary and ventilatory functions were measured with a spirometer and body plethysmography. A metabolic cart was used to study the effects of AE on metabolic cost. Gastric ( $P_g$ ) and pleural ( $P_p$ ) pressures were also taken simultaneously with two thin-walled latex balloons passed transnasally to determine effects of AE on ventilatory muscle recruitment. Electromyography (EMG) signal of the sternocleidomastoid was also assessed using two surface electrodes during the last 30 seconds of AE or AD.  $VO_2$  and  $VCO_2$  increased within 30 seconds of AE.  $VO_2$  peaked at 20% towards the end of the second minute. Similarly,  $VCO_2$  peaked at 23% with AE towards the end of the two minutes. This increase in  $VO_2$  and  $VCO_2$  remained elevated through the first minute after lowering arms. Respiratory response to AE shows that within 30 seconds of AE,  $V_E$  increased by 24% and peaked during the last 15 seconds of AE. The increase in  $V_E$  was due to an increase in  $V_T$  which is similar to the findings of Couser et al. (1992). AE also resulted in significant alterations in ventilatory pressures. Maximal inspiratory mouth pressure ( $PI_{max}$ ) dropped significantly after AE from 54 to 48 cm H<sub>2</sub>O, however both maximal expiratory mouth pressure ( $PE_{max}$ ) and  $Pdi_{max}$  did not change with AE,

73.6 (AD) to 70.9 cm H<sub>2</sub>O (AE) and 61.4 (AD) to 63.7 cm H<sub>2</sub>O (AE), respectively. Results showed significant increases in EMG activity in the sternocleidomastoid muscle when looking at amplitude with no changes in median frequency. The results of this study are similar to the findings of Couser et al. (1992) who also found marked increases in metabolic and ventilatory demands with AE in healthy subjects. The significant increases in P<sub>di</sub> and P<sub>gi</sub> during AE suggest that there was a change in ventilatory muscle recruitment, with an increased reliance on the diaphragm. Similar arm elevation protocols were used in Couser et al. (1992) which may be why similar results were observed even with the use of different subjects. In both studies, it was observed that AE caused an increase in V<sub>E</sub> which marks an increased metabolic demand thus causing an increase in respiratory muscle demand. These changes in metabolic and ventilatory parameters were associated with changes in ventilatory pressures (Couser et al., 1992). Findings from both studies reported that P<sub>di</sub> increased with AE. The difference between end inspiratory and end expiratory transdiaphragmatic pressures ( $\Delta$  P<sub>di</sub>) increased significantly and peaked at 14.1 cm H<sub>2</sub>O during the last 15 seconds of AE (Martinez et al., 1991) when arm position changed from AD to AE. Changes in  $\Delta$  P<sub>di</sub> were related to V<sub>T</sub>, and this ratio was greater with AE in both groups.

The literature has focused on upper extremity activity with those who suffer from some sort of CAO. Few studies have focused on healthy subjects and their response to AE. Couser et al. (1992) investigated respiratory response and ventilatory muscle recruitment during AE in healthy subjects. Twenty two healthy, nonsmoking subjects performed a series of pulmonary function tests and 11 subjects also underwent gastric and endoesophageal pressures. Gastric and endoesophageal pressures were determined by using two thin-walled latex balloons that went through transnasally; one was placed in the stomach and the other in the mid-esophagus.

Subjects were studied in two different seated positions, one with AE straight out in front of them and the other down at their sides (AD). Each position was held for two minutes. The results showed that there was a 16% increase in  $\text{VO}_2$  with AE when compared to AD, 289 ml/min and 336 ml/min respectively. Heart rate also increased by 16% with AE from 73 beats/min to 84 beats/min. It was also observed that during tidal breathing, there was a 24% increase from AD to AE, 9.3 L/min to 11.5 L/min respectively.  $V_E$  also increased which was due to an increased  $V_T$  from 721 ml AD to 868 ml. The changes in ventilatory and metabolic parameters during AE are suggested to be due to changes in ventilatory pressures.

End inspiratory gastric pressure ( $\text{PgI}$ ) significantly increased with AE when compared to AD. There no significant changes in end inspiratory endoesophageal pressure ( $\text{PplI}$ ), end expiratory endoesophageal ( $\text{Pple}$ ), and end expiratory gastric pressure ( $\text{PgE}$ ) (Couser et al., 1992). As a group, transdiaphragmatic ( $\text{Pdi}$ ) increased from 21.4 cm  $\text{H}_2\text{O}$  AD to 26.5 cm  $\text{H}_2\text{O}$  AE. Changes in metabolic and ventilatory parameters over the time course of 2 minutes and the subsequent 5 minutes after showed that heart rate increased significantly during AE at 3 and 4 minutes when compared to AD. The data showed that heart rate significantly increased from 73 bpm AD to 82 bpm AE at 3 minutes to 85 bpm AE at 4 minutes.  $V_E$  increased from 10.3 L/min AD to 13.0 L/min AE at 3 minutes to 12.6 L/min AE 4 minutes.  $V_T$  increased from 660 ml AD to 847 ml AE at 3 minutes to 809 ml AE at 4 minutes. The increases continued at 5 minutes for  $\text{VO}_2$ ,  $\text{VCO}_2$ ,  $V_E$ , and  $V_T$  before returning to baselines at 6 and 7 minutes. It is was also observed that  $\text{PgI}$  increased with AE but immediately decreased to baseline when arms dropped to the side, even with ventilatory and metabolic demands elevated. The results of the study demonstrate that AE in normal subjects increases metabolic demands ( $\text{VO}_2$ ,  $\text{VCO}_2$ , and heart rate) that are associated with increases in  $V_E$ , tidal breathing, end expiratory gastric and transdiaphragmatic

pressures. It was suggested that elevation of the arm changes ventilatory or postural muscle recruitment along with changes in the mechanics of the rib cage. The observed increase in ventilation was due to increase in  $V_T$ , and in order to increase  $V_T$  there must be increased activity of accessory ventilatory muscles (Couser et al., 1992). During AE, there was an increase in  $P_{di}$  through higher  $P_g$  which is unusual to observe in such a low intensity exercise. There are two possible reasons for the why this was observed according to the authors. One is some of the upper torso muscles that assist in ventilation are recruited to help support the arm thus decreasing activity from ventilatory needs, thereby shifting more work to the diaphragm. Another explanation suggested that AE retards the ability of the torso, rib cage, and abdominal wall to fully expand. An increase in diaphragmatic work to meet the associated ventilatory demands is needed with the accompanying changes in AE.

The previous studies examined the effects of arm elevation positions that were at  $90^\circ$  of shoulder flexion. McKeough, Alison, & Bye (2003) investigated the effect of arm elevation greater than  $90^\circ$  on lung volume in subjects with COPD and healthy subjects. It was hypothesized that arm elevation greater than  $90^\circ$  would alter lung volume, specifically inspiratory capacity (IC) and total lung capacity (TLC) when compared to arms below  $90^\circ$  shoulder flexion. Eighteen subjects participated in the study, half of them with COPD and the other half normal. Respiratory function tests were taken prior to testing in a seated position. The arm positions were randomized and consisted of arms below  $90^\circ$  shoulder flexion, arms at  $90^\circ$  shoulder flexion, and arms greater than  $90^\circ$  shoulder flexion. Subjects were encouraged to reach maximum attainable shoulder flexion, and ranged between  $153$ - $170^\circ$  in all subjects. In all three positions, subjects were asked to hold their cheeks and maintain position for the time it took to perform four resting breaths, five panting breathings, one maximum inspiratory maneuver, one expiratory maneuver, and one



further maximum inspiratory maneuver (total time about 40-60 seconds). There was a significant change in FRC between the different arm positions in both groups. FRC in both groups was significantly higher with the arms above 90° (4.60 L in COPD and 3.24 L in healthy) shoulder flexion when compared to below 90° (4.43 L in COPD and 2.95 L in healthy) shoulder flexion and at 90° (4.45 L in COPD and 3.01 in healthy) of shoulder flexion.

It was also observed that IC was lower with arms above 90° (1.59 L in COPD and 2.24 L in healthy) when compared to below (1.83 L in COPD and 2.70 L in healthy) and at 90° (1.73 L in COPD and 2.54 L in healthy) shoulder flexion in both groups (McKeough et al., 2003) TLC was similar in all three arm positions for the COPD subjects, however the healthy subjects had a significant change in TLC. TLC was lower with arms above 90° (6.16 L in COPD and 5.48 L in healthy) when compared to below (6.26 L in COPD and 5.65 L in healthy) and at 90° shoulder flexion (6.18 L in COPD and 5.50 L in healthy). The results show that arm positioning alters lung volumes in both COPD and healthy subjects.

Shoulder flexion above 90° in both groups significantly increased FRC and decreased IC when compared to the two other positions (McKeough et al., 2003). There was no difference in TLC, IC, and FRC with arms at 90° shoulder flexion when compared to arms below 90° shoulder flexion. Other studies examined arm positions either with arms at the side or with arms elevated to 90° (Couser et al., 1992) or clasped on the head (Dolmage et al., 1993). Both studies showed either no changes or only small changes in FRC, however, McKeough et al. (2003) found significant increases in FRC when the arms were above 90° shoulder flexion. It is suggested that when arms are above the head it causes the rib cage to be passively expanded as previously reported (Dolmage et al., 1993) placing the chest wall in an inflated position (McKeough et al., 2003). IC was also significantly reduced when arms were above 90° shoulder flexion which is in

accordance to the results found by Dolmage et al. (1993), where they also found a reduction in IC when arms were clasped on the head when compared to arms by the side. The reduction in IC in both groups when shoulder flexion was greater than 90° was said to be due to the passive stretch of the chest wall, reducing the ability of the chest wall to further expand. This reduced ability of the chest wall to expand further when arms are elevated has also been suggested to be a consequence of a tight trunk musculature (e.g latissimus dorsi) (Petta, Jenkins, & Allison 1998). The reduction in IC observed in both groups may be due to the passive stretch of the chest wall causing a tight trunk musculature thereby reducing IC in both groups.

The vast majority of the literature supports the idea that respiratory muscle recruitment is altered during various arm positions in both normal healthy subjects and those who suffer from CAO. The contribution to ventilation from the diaphragm and abdominal expiratory musculature is increased with less demand on chest wall musculature (Dolmage, 1993; Couser, 1992; Martinez, 1991; Mckeough, 2003). The strategies that both groups use in arm positions vary. Normal subjects respond by increasing reliance on diaphragm recruitment (Couser et al., 1992), whereas subjects with CAO rely more heavily on expiratory and abdominal muscles (Martinez et al., 1991). Subjects with CAO may rely more heavily on these muscles because of the associated diaphragm dysfunction and weakness that come with CAO (Martinez, 1990; Martinez, 1991).

To assess the role of the diaphragm during AE, Martinez et al. (1999) chose subjects with isolated diaphragm weakness without airflow obstruction. It was hypothesized that AE should result in similar metabolic and ventilatory demands with isolated diaphragm weakness and that respiratory muscle recruitment during AE should directly relate to the degree of diaphragm weakness. Fifteen subjects with documented isolated diaphragm weakness of varying severity were used in this study. Pulmonary function tests were taken prior to testing along with

respiratory muscle testing. A thin-walled latex balloon was passed transnasally into the midesophagus and stomach so that pleural pressure (P<sub>ple</sub>) and gastric pressure (P<sub>g</sub>) could be measured breath by breath. Arm elevation protocol was similar to that used previously by Martinez et al. (1991) and metabolic and ventilatory parameters were recorded with a metabolic cart. The findings support that simple AE in normal healthy subjects and those with isolated diaphragm weakness have similar metabolic and ventilatory responses. Subjects with isolated diaphragm weakness demonstrated a 19.9% rise in VO<sub>2</sub> and 32.2% increase in VCO<sub>2</sub> after two minutes of AE. A similar rise was observed in normal subjects. There was a significant difference in baseline P<sub>di</sub> between diaphragm weakness subjects and normal subjects (37.60 cm H<sub>2</sub>O and 138.30 cm H<sub>2</sub>O respectively), however both demonstrated comparable changes in P<sub>di</sub> during AE when compared to pre-AE. P<sub>ple</sub> also demonstrated a similar trend indicating that normal subjects and those with diaphragm weakness respond in the same way to AE in regard to P<sub>ple</sub>. The rise in P<sub>di</sub> demonstrated during AE supports the previous findings of Couser et al. (1992) and Martinez et al. (1991) who studied normal subjects and those with CAO. These findings further support that simple AE alters ventilatory muscle recruitment (VMR) which increases the demand placed on the diaphragm to meet ventilatory requirements (Couser, 1992; Martinez, 1991, 1999). The simple task of raising the arms to shoulder level and beyond in the sagittal or frontal plane may change the impedance of the torso, rib cage, and abdominal wall. Raising the arms causes a passive stretch to the thoracic musculature which may place these muscles in a less effective manner in assisting in respiration. The passive stretch places the chest wall in an already inflated position and also passively expands the rib cage accessory muscles, which may place these muscles in less optimal length, decreasing their force generating abilities (Dolmage, 1993; Criner & Celli, 1988).

### **Effect of arm bracing on pulmonary function**

The effect of body position on pulmonary function has been documented, especially in the physical therapy field. The goal of pulmonary rehabilitation program is to optimize response to exercise or activities of daily living in those with and without pulmonary dysfunction (Dean, 1985). In order to optimize lung function, alveolar ventilation and capillary blood flow (V/Q) must be matched. A mismatch of these leads to hypoxemia and hypercapnea which in turn may lead to respiratory failure, as seen with those with pulmonary dysfunction (Dean, 1985).

Body positions, including supine, prone, seated, erect, hands and knees, and forward lean position effects have been assessed on pulmonary functions (Dean, 1985). A study by Craig (1960) examined the effect of different positions on expiratory reserve volume (ERV) of the lungs. ERV is well established in the literature as being one of the most variable components of the subdivisions of TLC. Young, male, healthy adults volunteered in this study. ERV was defined as the maximal volume of air that could be expired from resting end-expiratory position. VC was taken in a seated position and also in a supine position after 10 minutes of lying supine. Five positions assessed were sitting (Sit) (erect in a chair, feet flat on the floor and hands in lap), arm supported while sitting (AS) (subjects were instructed to raise elbows to the arm chairs), AS with forward lean (ASF) where subjects were instructed to lean forward on arm chairs until comfortable. In the supine position, subjects were supine with arms at their sides, supine with weight, where subjects lied supine with the addition of 1.2 kg of weight placed in a pan that was placed on the anterior abdominal wall. Hands-knees position required subjects to be on their hands and knees (HAK), with upper and lower extremities at right angles to the trunk. ERV was reported as a percentage of the subject's VC. ERV in the sitting position was 34.2% of VC, and increased with AS (37.4%), ASF (41.3%), and HAK (44.5%). The increases in ERV when arms

were supported by the chair were attributed to the displacement of weight from the thoracic cage. It was noted that a scale was placed under the arm chair and a force of about 7-9 kg was reported. By displacing the weight to the shoulder girdle in the bracing position, it acts to assist the spring like system of the lungs and thorax. During the lean forward position (ASF), there was a further increase from AS by 4% and an even further increase by about 7% in the HAK position. The increases in ERV in the AS, ASF, and HAK positions have been suggested to be due to a displacement of the abdominal contents in a downward fashion, especially with HAK position. The downward displacement of the abdominal contents has been reported to contribute greatly to increase in FRC (Dean, 1985). ERV is a component of FRC with the addition of RV making up FRC. It could be speculated that increased ERV would therefore also increase FRC which is known to be affected by body position (Dean, 1985).

The abdominal muscles are known to play an important role during respiration (De Troyer, 1983; Hudson, 2010; Kera & Maruyama, 2005; Roussos, 1985). De Troyer (1983) reported that tonic activity of abdominal muscles is present in normal humans at rest. This activity is suggested to assist during inspiration as it may facilitate a reduction in energy costs during inspiration (De Troyer, 1983). The abdominal muscles lengthen during inspiration which prevents disproportionate shortening of the diaphragm, thus increasing its ability to produce pressure during the inspiratory phase (De Troyer, 1983). The zone of apposition (ZOA) is the mechanical linkage between the diaphragm and rib cage (Boynton et al., 1991; Mead, 1979). The ZOA is anatomically important because it influences diaphragmatic tension which is controlled by abdominal muscles (Hruska, 1997).

To further investigate the role of abdominal muscles during respiration, Kera and Maruyama (2005) examined the effects of posture on respiratory activity of the abdominal

muscles. Fifteen healthy, young male adults participated in the study. Respiratory muscle strength was expressed as maximal expiratory (PE<sub>max</sub>) and inspiratory (PI<sub>max</sub>) efforts of mouth pressures. PE<sub>max</sub> was measured at TLC and PI<sub>max</sub> was measured at RV. Lung capacities were measured with a spirometer: VC, inspiratory residual volume (IRV), expiratory residual volume (ERV), V<sub>T</sub>, and FVC. MVV was also measured for 15 seconds in all positions. An external respiratory load was used to activate abdominal muscles; the load was set to 20 cm H<sub>2</sub>O. Surface EMG was used to measure abdominal muscle activity of the external obliques (EO), internal obliques (IO), and rectus abdominis (RA). Four positions were assessed, including sitting with elbows on knees (SEK), supine, standing, and sitting. Subjects were asked to breathe spontaneously (SB) with a normal rhythm under a load of 20 cm H<sub>2</sub>O.

Lung volume results show that VC in the standing (4.63 L), sitting (4.71 L), and SEK (4.72 L) were greater than in the supine position (4.46 L). IRV was greatest in the supine position (2.60 L) but smallest when looking at ERV, where SEK (2.20 L) was the largest when compared to supine (1.33 L), sitting (1.91 L), and standing (1.78 L) (Kera & Maruyama 2005). Abdominal muscle activity during MVV showed that EO was not affected by posture, RA was higher in supine position than in standing position, and IO activity was lower in SEK position when compared to sitting or standing. During inspiration, EO muscle activity was significantly higher in SEK and standing than in supine position. IO muscle activity was higher in the sitting and standing position when compared to SEK position. During spontaneous breathing, abdominal muscle activity changed significantly with changes in posture. EO muscle activity was greater in the SEK when compare to the others. IO muscle activity was greatest in the standing position when compared to SEK or supine.

The findings of this study show that as position changed, lung volumes also changed. The relative decrease in VC and TLC in the supine position when compared to standing positions was due to a shift in blood flow from the lower extremities to the thorax. In the supine position, it has been suggested that the abdominal contents are pushed against the diaphragm causing the diaphragm to rise into the thoracic cavity, thereby reducing FRC. The authors also expected to observe similar decreases in the SEK position, because the flexed trunk would cause an increase in intra-abdominal pressures. But contrary to their expectations, the VC in SEK was similar to that in the standing position and was larger than the supine. By flexing the trunk, gravity works to pull the abdominal wall down which prevents an increase in intra-abdominal pressure. The changes in FRC are mainly determined by the pressure changes that affect the elastic recoil of the rib cage. This is affected by alterations in pressures above and below the diaphragm (Kera and Maruyama, 2005). The thorax and lungs are not the only components that contribute to respiration but the abdominal cavity and muscles around it also contribute greatly (Craig et al. (1960).

The findings of this study show that EMG activity of the abdominal muscles also showed changes with changing positions. The increases in EO activity in the SEK position during inspiration and expiration are attributed to the enhanced position of the abdominals when the trunk is flexed. In this position, contraction of the abdominal muscles are stronger, as the upper limbs are braced and pressed against the thighs. A stretch of the EO muscle during rib cage expansion in the SEK position elicited a stretch reflex, contributing to the increased EO activity during inspiration. The authors concluded that the EO muscle activity in the SEK position induced an increase expiration which also increased inspiration due to the stretch reflex, thereby reducing the feelings of dyspnea especially in those who suffer from COPD.

The literature has focused attention on dyspnea relief especially in those who have some sort of CAO and COPD. It has been well established that those who suffer from CAO and COPD have difficulty with activities of daily living (ADLs) and increased feelings of dyspnea especially during tasks that involved the upper extremity (Banzette, 1988; Couser, 1992; Dolmage, 1993). Alterations in body positions influence pulmonary function. Raising the arms to 90° of shoulder flexion or greater increases metabolic cost at any given workload and a forward lean position may improve lung capacities. Another body position that incorporates both arms and a forward lean position is bracing of the arms on the knees or a solid object. This position is often used by patients with COPD to relieve their dyspnea, however the reasons for this are unclear (Banzett et al., 1988).

To further investigate this idea, Banzett et al. (1988) examined the strategy of bracing the arms in four healthy male subjects. Maximal ventilatory capacity was measured for four minutes in subjects while seated with their arms braced on a table or unbraced. VC and peak inspiratory flows were also measured in each subject in both braced and unbraced positions. Ventilatory capacity test lasted for four minutes and subjects were asked to reach a target ventilation of 70-80% of their 15 second MVV. The braced position consisted of subjects seated on the front half of the chair and leaned forward with the elbows braced firmly on the table, while the unbraced position was similar to the braced except the arms were held above the table 1-5 cm. Subjects only performed two trials on any given day and order of trial was alternated between days. VC was greater by 6% throughout the 4 minutes and 8% in the last 30 seconds of testing in the braced position when compare to unbraced. This small improvement was attributed to improved accessory muscle function with the arms braced and supported. Two mechanisms were proposed that may explain the benefits of arm bracing in respiratory function. The muscles of the back



and shoulder that act as postural and respiratory muscles have to work twice as hard when arms are not braced causing these muscles to fatigue sooner consequently impairing respiratory performance. The second proposed mechanism is that the muscles that lift and expand the rib cage originate on the pectoral girdle and move caudally when contracted. When the arms are braced by either leaning on a table with the elbows or bracing the hands on the knees with elbows locked, the position causes the shoulder girdle to be lifted and fixed thereby maintaining its optimal muscle length.

In a more recent study, Cavalheri et al. (2010) also investigated the effects of arm bracing posture on respiratory muscle strength and pulmonary function in patients with COPD. Twenty subjects with COPD were recruited for the study. All subjects performed lung function testing with a spirometer and maximal respiratory pressures were also assessed in both braced and unbraced positions. Both positions were assessed standing up either with a support or without. The height of the support was adjusted to the level of the ulnar styloid process with a 30° trunk flexion with elbows flexed. Lung function measurements revealed higher respiratory pressures during arm bracing than without. Maximal inspiratory pressure (MIP) with arms braced was  $64 \pm 22$  cmH<sub>2</sub>O and  $54 \pm 24$  cmH<sub>2</sub>O without arms braced. Arm bracing also revealed that 85% of subjects had greater inspiratory muscle strength when compared to without braced arms. Maximal expiratory pressure (MEP) was also greater in the arm braced position showing that 90% of the subjects had greater expiratory muscle strength in the braced position ( $104 \pm 37$  cmH<sub>2</sub>O) compared to  $92 \pm 37$  cmH<sub>2</sub>O without arms braced. A similar trend in MVV values was also observed,  $42$  L min<sup>-1</sup> arms braced and  $38$  L min<sup>-1</sup> without arm braced. The findings of this study along with Banzett et al. (1988) suggest that arm bracing posture improves respiratory

function by enhancing the ability of the respiratory muscles (diaphragm, abdominals, and accessory muscles) to generate force when arms are braced.

The ability to brace and support the arms on a solid surface when compared to unsupported arms, influences pulmonary function, respiratory muscle function, and functional capacity (Banzett, 1988; Cavalheri, 2010; Craig, 1960; Kera & Maruyama, 2005). The suggested mechanisms that explain this have been related to improved diaphragm function in the forward lean position (Barach, 1974; Craig, 1960; O'Neil, 1983), improved accessory muscle function (Banzett, 1988; Cavalheri, 2010; Kera & Maruyama, 2005), and reduced metabolic and ventilatory costs with arms supported (Couser, 1992; Criner & Celli, 1988; Dolmage, 1993; Martinez, 1991, 1999; McKeough, 2003).

**Effects of respiration on heart rate.** The relationship between the respiratory and autonomic system has been well studied to observe the interaction and influences of the two systems on each other, a phenomenon called respiratory sinus arrhythmia (RSA) (Blain, 2004); Yasuma & Hayano, 2004; Hayano, Yasuma, Okada, Mukai & Fujinami, 1996; Hirsch & Bishop, 1981). RSA is a beat to beat interval variation in heart rate which is influenced by respiration (Hayano, 1996; Hirsch & Bishop, 1981). When looking at an electrocardiogram (ECG), the R-R intervals are decreased during inspiration and increased during expiration (Yasuma & Hayano, 2004). A majority of the literature has focused on RSA at rest and there is little information related to exercising conditions.

A study by Blain et al. (2004) investigated the influence of breathing on RSA in humans during exercise. This study was the first to assess the influence of ventilation on RSA, therefore the use of an original signaling processing method was produced. The study used 14 healthy sedentary male subjects. Each subject performed a graded exercise test on a cycle ergometer

where 70% of peak  $\text{VO}_2$  was referenced as their workload. The submaximal exercise testing lasted 6 minutes and was done 5 times with a 12 minute recovery between each bout where  $V_E$ , respiratory frequency ( $F_R$ ), and  $V_T$  were measured. The results of the study showed that during exercise testing, similar breathing frequencies occurred. This suggests that breathing may modulate sinus node activity that parallels to  $f_b$ .

RSA has also been suggested to positively influence pulmonary gas exchange (Hayano et al., 1996) and reduce cardiopulmonary energy expenditure by decreasing the number of heart beats during expiration (Yasuma & Hayano, 2004), though these findings are not conclusive and need further exploration. It can be suggested that there is a definite interaction between respiration and heart rate which is controlled by the autonomic nervous system (Borresen & Lambert, 2008).

During exercise, the increase in heart rate is due to increased sympathetic activation and vagal withdrawal (Imai et al., 1994). The reduction in heart rate immediately after exercise is suggested to be from sympathetic withdrawal (Savin, Davidson, & Haskell, 1982) and parasympathetic reactivation (Buchheit et al., 2007; Buchheit, 2006; Cole et al., 1999; Savin et al., 1982). There is increasing interest in the literature in the regulation of heart rate after exercise, more specifically the immediate reduction in heart rate known as heart rate recovery (HRR) (Borresen & Lambert, 2007, 2008; Buchheit et al., 2007; Buchheit, 2006; Cole et al., 1999; Imai et al., 1994; Morise, 2004). HRR is defined as the rate of immediate decrease in heart rate after moderate to heavy exercise to pre-exercise levels (Borresen & Lambert, 2008) and is controlled predominantly by parasympathetic reactivation (Buchheit et al., 2007; Buchheit, 2006; Imai et al., 1994). There are a few ways to measure HRR, but the most commonly used and

simplest method is taking the absolute difference in HR immediately after exercise and 60 seconds later (Cole et al., 1999).

A delayed HRR from exercise is shown to be a strong and independent predictor of mortality, which was defined as a delay of 12 beats or less in the first minute after termination of exercise (Cole et al., 1999). It has been suggested that measuring HRR immediately after exercise may serve as a valuable prognostic tool in assessing autonomic function and identifying patients with increased risk of cardiac failure (Borresen & Lambert, 2008; Cole et al., 1999; Imai et al., 1994). Analysis of HRR after exercise has also been suggested to be a simple and valuable tool for monitoring training status of athletes (Borresen & Lambert, 2008). Fitter subjects have faster heart rate responses at the onset of exercise and also after when compared to sedentary subjects (Bunc, Heller, & Leso, 1988; Javorika et al., 2002). It has been suggested that with training, more specifically endurance training, results in enhanced HRR immediately after exercise due to increased parasympathetic tone (Yamamoto et al., 2001). Conversely, a decrease in HRR after training is shown to negatively impair performance associated with training induced fatigue (Lamberts et al., 2009). Lambert et al. (2009) demonstrated that HRR in well trained endurance athletes responds to changes in training load. The authors suggested that a prolonged HRR after an applied training load may predict accumulated fatigue from previous training load thereby blunting future performance. It is therefore plausible to suggest that measuring HRR immediately after exercise provides valuable information for monitoring an athlete's response to training.

**High intensity interval training in soccer players.** The respiratory system is a major limiting factor during high intensity endurance exercises in both trained and untrained subjects (Boutellier & Piwko, 1991; Bouteller, 1992). It has been observed that respiratory muscles

fatigue occurs during high intensity endurance training which can cause a decrease in performance levels in trained and untrained individuals (Babcock, 1996; Boutellier, 1991, 1992; Guenette & Sheel, 2007). More specifically intensity levels greater than 85 % of  $VO_{2max}$  have been observed to elicit diaphragmatic fatigue (Johnson et al., 1993).

A mode of training that elicits this type of intensity is high intensity interval training (HIIT). It has been defined as repeated bouts of exercise close to maximal effort ( $> 90\%$  of  $VO_{2peak}$ ) separated with few minutes of rest (Gibala & McGee, 2008). HIIT significantly improves  $VO_{2max}$  and improvements in  $VO_{2max}$  are associated with improved aerobic capacity in soccer players (Helgerud et al., 2001; Helgerud et al., 2007). However soccer performance is also dependent on the ability to perform multiple bouts of high intensity runs with little recovery, which increases the demands on the anaerobic system (Bangsbo, 2006; Krstrup et al., 2006; Mohr et al., 2003). It has been suggested that HIIT improves both aerobic and anaerobic performance components in soccer players (Dupont, Akakpo, & Berthoin, 2004; Helgerud et al., 2007).

A study by Helgerud et al. (2007) compared the effects of different training methods at various intensities and its influence on  $VO_{2max}$ . The study used forty, male, college students that endurance trained three times per week prior to study. Four training methods were compared and equated for total work for each session and subjects were randomly selected for each training method. The subjects trained three times per week for 8 weeks in one of the four groups. The first group performed a long slow distance run (LSD) at 70% of HR max for 45 minutes, the second group performed a continuous run at lactate threshold (LT) (85% of HR max) for 24.25 minutes, the third group performed 47 repetitions of 15/15 intervals of 15 second runs at 90-95% HR max and a 15 second rest period at 70% HR max, and the fourth group performed 4 X 4

minute intervals at 90-95% HR max with 3 minutes of rest at 70% HR max. Results of the study showed that the high intensity training group of 15/15 and 4 X 4 significantly increased absolute  $VO_{max}$  (5.5% and 7.2% respectively) when compared to the LSD and LT groups. There was no significant difference in training response between the 15/15 and 4 X 4 groups. The authors suggested that intensity of training is a key determinant to the response of the training. Therefore, the authors concluded that HIIT is more effective in improving  $VO_{2max}$  when compared to LSD and LT and was due to the higher intensities of the training.

A study by McMillan et al. (2005) that used a similar training method (4 X 4 minutes with 3 minutes recovery) to the previous study assessed the physiological adaptations in youth soccer players. However, in contrast to Helgerud et al. (2007), the training intervention was performed twice per week over an 8 week period, plus training was performed with a soccer ball on specially designed artificial turf field. The study found similar results and showed significant increases in  $VO_{2max}$  in soccer players (63.4 ml/kg<sup>-1</sup> to 69.8 ml/kg<sup>-1</sup>) with no negative effects on strength, jumping ability, and sprint performance.

A workload of 4 sets of four minute intervals at 90-95% of HR max, with 3 minute rest periods has been popularly used in soccer training regimens to improve cardiorespiratory fitness in contrast to continuous long running at lower intensities (Helgerud et al., 2007; McMillan, 2005). Since HIIT improves cardiorespiratory fitness and is widely used in the sport of soccer, it is appropriate to use in the current study to assess the effects of different postures as a form of immediate recovery strategy.

The physical demands required in soccer rely on both aerobic and anaerobic systems for energy which can influence game performance. Physical assessments of athletes are important in determining their physical capacities and training status. A commonly used assessment of

anaerobic power is the Wingate Anaerobic Test which measures peak and mean anaerobic power (Zupan et al., 2009). The Wingate Anaerobic Test is a maximal effort test performed on an ergometer where subjects are instructed to pedal as fast as possible for 30 seconds.

There is conflicting evidence in the use of the Wingate Anaerobic Test as an assessment of anaerobic power in field sports like soccer (Karakoc, Akalan, Alemdaroglu, & Arslan, 2012; Keir, Thériault, & Serresse, 2013; Krstrup et al., 2006; Meckel, Machnai, & Eliakim, 2009). However, there is evidence of moderate relationship ( $r = -0.55$ ) between peak power and fatigue index in the Wingate Anaerobic Test and field base test of the Yo-Yo intermittent recovery in evaluating anaerobic power in soccer players (Karakoc et al., 2012). It is considered the gold standard in anaerobic power assessment, however further studies are needed to determine the applicability in field base sports. Due to conflicting findings in the use of the Wingate Anaerobic Test in field base sports, it cannot be ruled out as a reliable method for testing anaerobic power in soccer players.

### **Summary**

The respiratory system is a major limiting factor in high intensity endurance exercises in both trained and untrained subjects (Boutellier & Piwko, 1992). The respiratory muscles during high intensity endurance activity fatigue and cause a decrease in performance (Babcock, 1996; Boutellier & Piwko, 1992). Respiratory muscle fatigue is especially observed in those who suffer from pulmonary dysfunction like COPD (Banzett, 1988; O'Neil, 1983). Several authors have reported that bracing of arms on the knees or a solid object relieves the feeling of breathlessness or dyspnea (Banzett, 1988; Cavalheri, 2010; Kera & Maruyama, 2005; O'Neil, 1983). However, there is no evidence that hands on the head or hands on the knees strategy for immediate recovery from HIIT will have an effect on cardiorespiratory responses of trained subjects. If

recovery posture influences speed of recovery, we would anticipate a positive influence on subsequent performance.



## Chapter III

### Methods and Procedures

#### Introduction

The purpose of this study was to determine if hands on head (HH) and hands on knees (HK) recovery postures have an influence on performance of repeated bouts of high intensity exercise and subsequent performances of anaerobic power in collegiate female soccer players. Specifically, the study examined how the two different postures could possibly influence cardiorespiratory function during high intensity interval training (HIIT) and also subsequent performance in a Wingate Anaerobic Test. The high intensity interval exercises were performed on a treadmill over three testing sessions where heart rate recovery (HRR), carbon dioxide elimination ( $\dot{V}CO_2$ ) and tidal volume ( $V_T$ ) were recorded and calculated during the recovery phase of the testing. Immediately after high intensity interval exercise performance, a Wingate Anaerobic Test was performed where mean power was recorded.

The purpose of this chapter is to describe the methods and procedures that were used in this study. The first section focuses on subject characteristics followed by the design of the study, data collection procedures, instrumentation, measurements techniques and procedure, and finally data analysis of the study.

#### Description of Study Subjects

The study sample consisted of 20 female Division II soccer players between the ages of 18-22 ( $20.3 \pm 1.1$ ) years old. All subjects in the study have trained using HIIT protocols and were in the winter season of their training schedule. The university's Human Subjects Committee reviewed the study prior to any data collection and subjects gave their informed consent (Appendix A).

## **Design of the Study**

A multiple participant, within subject design was conducted. Subjects were randomly designated a recovery posture to perform on the first testing day. Subjects performed a total of two treadmill sessions of HIIT which consisted of four minutes of running and three minutes of recovery performed four times (4 X 4 minutes) assuming one of the two recovery postures during the recovery period. After each HIIT session, a Wingate Anaerobic Test was performed. The Wingate Anaerobic Tests were performed immediately after the last recovery interval in each session. The transition time from HIIT test to the Wingate Anaerobic Test was between 15-30 seconds. The purpose of the Wingate Anaerobic Test was to determine if there were any changes in mean anaerobic power following HIIT with the influence of different recovery postures used in the rest interval.

Anthropometric baseline measures and pulmonary function tests were recorded prior to testing, including body mass index (BMI), vital capacity (VC), forced expired volume (FEV<sub>1</sub>), FEV<sub>1</sub>/VC ratio, and maximal voluntary ventilation (MVV). A total of two testing sessions were performed by each subject, separated by one week. Each session consisted of a five minute warm up at a running speed which elicited 70% of their heart rate (HR) max at 0% grade on a treadmill followed by four running intervals at an intensity of 90-95% of HR max for four minutes, with a three-minute recovery between runs, assuming either HH or HK postures. Immediately after completion of the last three minutes of recovery in assigned posture, subjects then performed a Wingate Anaerobic Test on a cycle ergometer for 30 seconds.

## **Data Collection Procedures**

**Instrumentation.** Pulmonary measures were performed using a Parvomedics (Sandy, UT) spirometer in the Exercise Physiology Lab at Western Washington University. The

submaximal treadmill runs were performed in the laboratory on a Precor treadmill. The intensity of submaximal treadmill runs simulated game-like soccer training intensities of 90-95% of predicted HR max, derived from the  $220 - \text{age}$  equation (Fox & Haskell, 1970). Heart rate was monitored with a Polar heart (Lake Success, NY) monitor. The submaximal treadmill runs consisted of four intervals of four minutes of running with a three minute recovery assuming one of the two postures (HH or HK). HRR was measured at the beginning of the three minutes of recovery for the first minute.  $\dot{V}CO_2$ ,  $V_E$ , and  $f_b$  were recorded during the recovery phase of the testing. A Parvomedics TrueOne (Sandy, UT) Metabolic Cart was used to measure  $\dot{V}CO_2$  and  $V_T$ , which was calculated from  $V_E$ , and  $f_b$ . The Wingate Anaerobic Test for anaerobic power was performed on a Monark cycle (Sweden) ergometer, modified for electronic capture interfaced with a computer. Mean anaerobic power was measured in watts over a 30-second period. The resistance used is relative to each subject's body weight ( $0.075 \text{ kp} \cdot \text{kg}^{-1}$ ). Subjects were instructed to pedal as fast as they could for the entire 30 second period while the computer recorded the revolutions of the flywheel.

**Measurement techniques and procedures.** The researcher explained the study and the time involved to complete study. Prior to testing, subjects were informed of testing procedures and were provided with an informed consent document. The subjects were told that HH required them to stand erect with their hands clasped on top of their head, HK required them to place their hands on their knees, elbows locked and flexing their thoracic spine. HK posture required additional measurement of thoracic flexion with inclinometers to assure consistency of flexion between rest intervals (Van Blommestein, Lewis, Morrissey & MacRae, 2012). Two inclinometers were used to measure thoracic flexion at T1 and T12. It was also explained that each posture was held for the entire recovery period and recovery postures were taught and

practiced during the baseline measurement session. Each subject completed one day of baseline measurements, which included pulmonary function tests consisting of VC, FEV<sub>1</sub>, FEV<sub>1</sub>/VC, and MVV, and anthropometric measurements, which included BMI.

Familiarization with the treadmill was also performed on the same day as baseline measurements. During this time, running speed required to elicit 90-95% of HR max was determined by having subjects warm up for five minutes at a speed that elicited 70% of HR max. After the five minute warm up, speed was increased until the target HR was reached and at a speed where subjects could maintain running for the full four minutes. Subjects were encouraged to reach target HR at a comfortable speed within 1-1.5 minutes. Treadmill incline was set at 0% for all subjects during all testing sessions. Resistance and seat height for the Wingate Anaerobic Test were also measured and recorded. Familiarization with the Wingate Anaerobic Test of anaerobic power was also performed after familiarization with treadmill protocol. Following baseline measurements, two testing sessions were conducted with one week of rest between sessions. Each testing session consisted of four submaximal treadmill runs at 90-95% of HR max for four minutes with a three-minute recovery between intervals. A Wingate Anaerobic Test power test for 30 seconds was then performed immediately after the last three minute recovery interval of the HIIT.

Subjects were instructed not to participate in any high intensity activity the day before testing, so that fatigue from previous activity would not affect testing sessions. Subjects were also encouraged not to consume caffeine the day of testing and to get a minimum of seven hours of sleep the night before testing. Uniform verbal encouragement was given to all subjects during all treadmill running sessions and Wingate Anaerobic Test.

The submaximal treadmill runs were used to simulate intensities experienced in the field so that application of activity was similar to that which subjects undergo on a daily basis in training. Subjects were asked to sit on a stool placed on the treadmill for preparation of testing, which consisted of HR monitor attachment. Subjects warmed up on treadmill for five minutes at 70% HR max. After the five minute warm up, speed was increased until subjects reached the target HR (90-95% HR max) within 1-1.5 minutes, after which they were instructed to maintain speed by adjusting the treadmill speed. Subjects were instructed to keep pace with the treadmill for the full 4 minutes of running and, immediately after 4 minutes, were instructed to step to the sides of the treadmill and assume the recovery position assigned for the given testing session. At the same time, a 2-way breathing mouthpiece valve interfaced with the metabolic cart was inserted, and a nose clip was applied for data collection during the three minutes of recovery.  $\dot{V}CO_2$ ,  $\dot{V}_E$ , and  $f_b$  were measured every minute over the recovery period. These values were averaged over the three minutes for analysis. HRR is commonly defined as the difference in HR at the end of exercise and then 60 seconds later (Cole et al., 1999). Similarly, in this study, HRR was measured immediately at the end of the exercise and then one minute later. The average of first minute in each rest interval was used for analysis. The HR monitor was worn throughout the entire session, which included HIIT and the Wingate Anaerobic Test. Subjects performed a total of four 4 minute running intervals (4 X 4), and four 3 minute recovery intervals for each session. Immediately after the last 3 minute recovery interval, preparation for the Wingate Anaerobic Test was completed, which included detachment of mouth piece and nose clip. This was done as efficiently as possible to minimize transition time from testing procedures.

The Wingate Anaerobic Test was immediately performed after the last 3 minute recovery interval of the HIIT. Subjects were asked to pedal as fast as they could for the entire 30 second

trial while the computer counted the revolutions of the flywheel. Verbal encouragement was given throughout the 30 seconds.

### **Data Analysis**

Descriptive statistics were determined for each variable. Dependent t-tests were used to detect significant differences due to the two treatments using SPSS, version 21. The dependent variables analyzed included HRR,  $V_{CO_2}$ , and  $V_T$  during each recovery posture and mean anaerobic power (Watts) from the Wingate Anaerobic Test. Significance was defined as a p-value less than 0.016 with the Bonferroni correction applied for multiple tests.

## Chapter IV

### Results and Discussion

#### Introduction

The purpose of this study was to examine the effects of two postures used during the recovery interval in high intensity interval training (HIIT) and the effect on heart rate recovery (HRR), carbon dioxide elimination ( $V_{CO_2}$ ) and tidal volume ( $V_T$ ). To further examine the effects of the two postures on subsequent power performance, a Wingate Anaerobic Test was performed to measure mean anaerobic power. Results pertaining to the analysis are presented and discussed following the description of the subject's characteristics.

#### Subject Characteristics

The study population consisted of 24 female Division II soccer players between the ages of 18 and 22 years old. All subjects were in their winter training season when they began participation in the study and were instructed to not modify their training. During the duration of the study four subjects dropped out. Three subjects dropped out due to time conflicts with their scheduled testing times and one subject's data was dropped due to incomplete data collection, which resulted in a final subject pool of 20 participants. Table 1 presents subject characteristics and spirometer measures in all 24 subjects.

**Table 1: Subject characteristics and Spirometer measures**

<b>Subjects</b>	N = 24
	<b>Mean ± SD</b>
<b>Age (years)</b>	20.3 ± 1.1
<b>Weight (kg)</b>	65 ± 6.7
<b>Height (m)</b>	1.706 ± 0.1
<b>BMI (kg/m<sup>2</sup>)</b>	22.4 ± 1.8
<b>VC (L)</b>	4 ± 0.6
<b>FEV<sub>1</sub> (L)</b>	3.1 ± 0.4
<b>FEV<sub>1</sub>/VC (%)</b>	80% ± 0.1
<b>MVV (L/min)</b>	133 ± 16

### Results of the study

After completion of testing sessions, the following results were observed. Variables measured include: HRR,  $\text{VCO}_2$ ,  $\text{V}_T$  and mean anaerobic power.

Comparison of HRR data by a dependent t-test revealed a significant difference between HH and HK postures. HK posture recovered significantly greater than HH posture ( $t(19) = 11.82, p < .001$ ). Table 2 shows the average and standard deviation of HRR in both postures. A difference of 22 beats per minute (bpm) between HK and HH after the first minute of recovery. These differences can be seen in Figure 1.

$\text{VCO}_2$  was measured and recorded throughout the three minutes of recovery and was averaged over the four recovery intervals to get a mean recovery  $\text{VCO}_2$  for each posture. The dependent t-test revealed no significant effect of the postures ( $t(19) = 2.01, p = 0.04$ ) (Table 2). Figure 2 displays the differences in  $\text{VCO}_2$  between HH and HK.

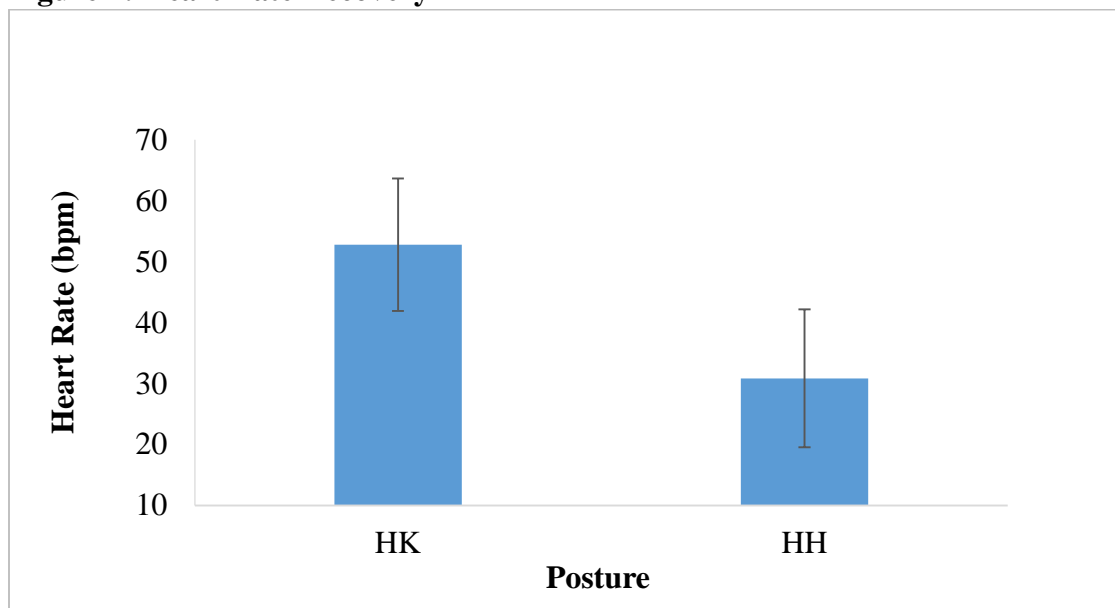


$V_T$  was calculated from minute ventilation ( $V_E$ ) and breathing frequency ( $f_b$ ), which were measured and recorded throughout the three minutes of recovery and averaged over the four recovery intervals. Comparison of the groups revealed a significant difference between HH and HK posture. HK posture  $V_T$  was significantly greater than HH posture  $V_T$  ( $t(19) = 2.93, p < .008$  (Table 2). Figure 3 reveals the difference in  $V_T$  values between the two postures.

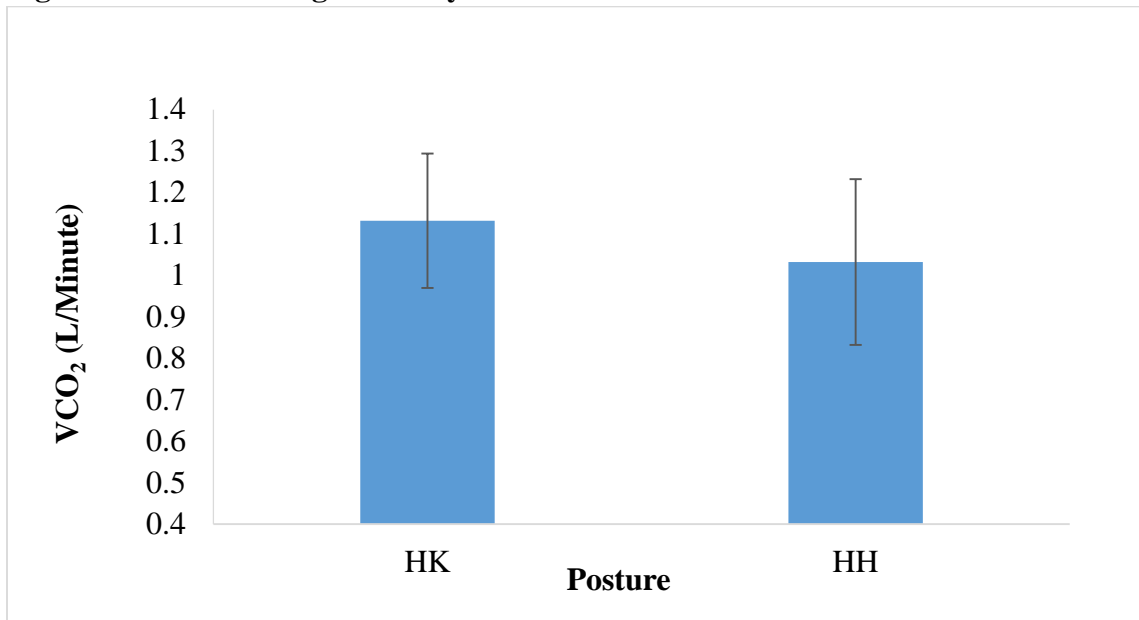
**Table 2: Heart Rate Recovery (HRR), Carbon Dioxide elimination ( $V_{CO_2}$ ), and Tidal Volume ( $V_T$ )**

	HRR (bpm)	$V_{CO_2}$ (L/min)	$V_T$ (L/min)
<b>HK</b>	53 ± 10.9	1.1 ± 0.2	1.4 ± 0.2
<b>HH</b>	31 ± 11.3	1.0 ± 0.2	1.3 ± 0.2

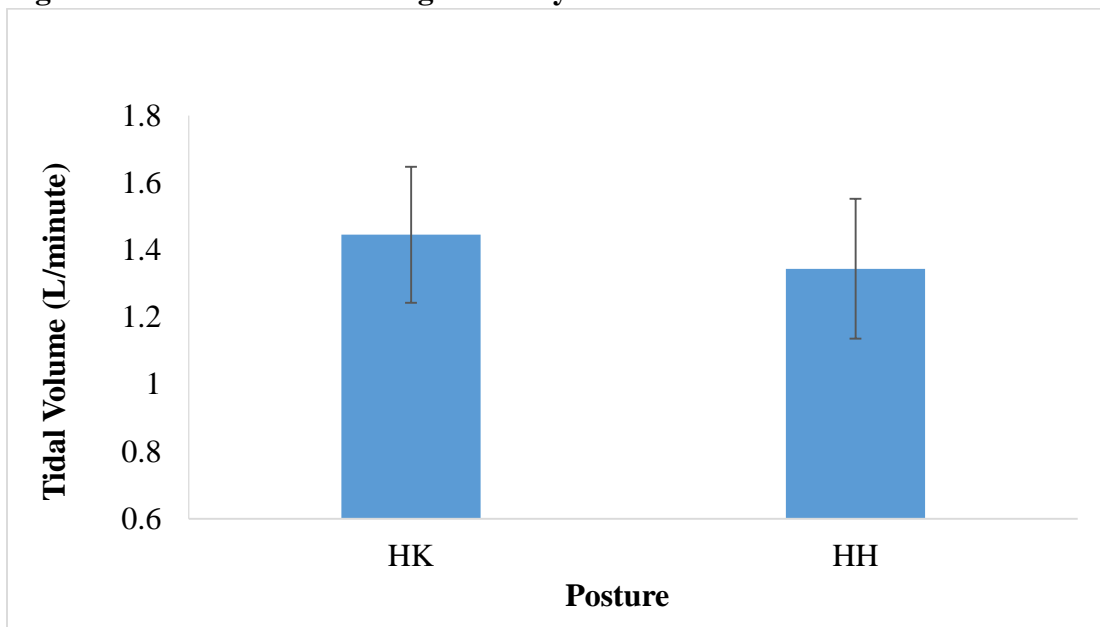
**Figure 1: Heart Rate Recovery**



HK = Hands on knees; HH = Hands on head

**Figure 2: VCO<sub>2</sub> During Recovery Intervals**

HK = Hands on knees; HH = Hands on head

**Figure 3: Tidal Volume During Recovery Intervals**

HK = Hands on knees; HH = Hands on head

HK posture required additional measurement of thoracic flexion with inclinometers during the rest interval to assure consistency of flexion between each rest interval. Table 3 shows the averages of thoracic flexion between each rest interval.

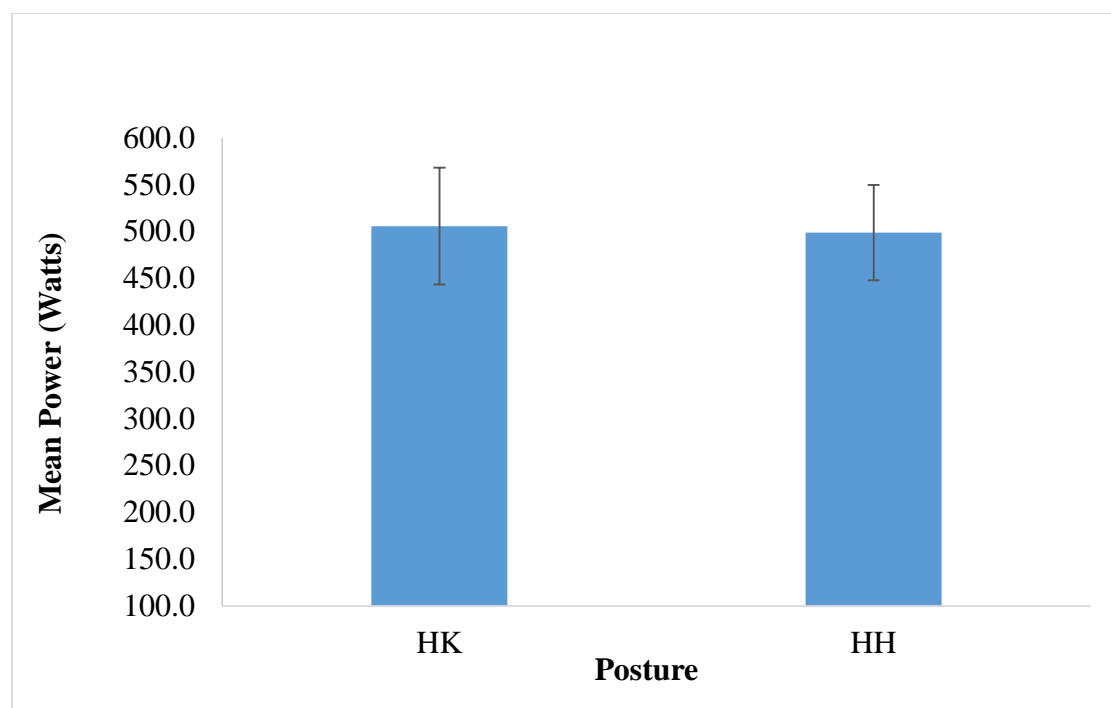
**Table 3: Mean Thoracic Flexion during hands on knees (HK)**

	<b>Mean Thoracic Flexion (°) ± SD</b>
<b>Rest 1</b>	14.6 ± 4.4
<b>Rest 2</b>	15.5 ± 7.0
<b>Rest 3</b>	17.6 ± 7.6
<b>Rest 4</b>	19.5 ± 8.2

Comparison of mean anaerobic power between HK and HH data revealed no significant difference between the two postures ( $t(19) = .536, p = .598$ ). Table 4 shows the means and standard deviation in Watts of anaerobic mean power for both groups. Figure 4 displays the differences in mean anaerobic power between the two groups.

**Table 4: Mean Power values**

	<b>Mean Anaerobic Power (Watts) ± SD</b>
<b>HK</b>	505.8 ± 62.5
<b>HH</b>	498.8 ± 50.9

**Figure 4: Mean Anaerobic Power in Watts**

HK = Hands on knees; HH = Hands on head

## Discussion

The purpose of this study was to determine if HH or HK recovery posture would have an influence on cardiorespiratory function during HIIT and also subsequent performance in a Wingate Anaerobic Test. Subjects were randomly assigned HH or HK posture for the first testing session. Each subject performed four intervals of 4 minutes of running (4X4) with three minutes of recovery between each running interval. During each three minute recovery interval, measurements recorded included: HRR for the first minute and then  $V_{CO_2}$ ,  $V_E$  and  $f_b$  were also recorded for three minutes then averaged.  $V_E$  and  $f_b$  were used to calculate  $V_T$ . After the last recovery interval, each subject performed a Wingate Anaerobic Test. This was done for both HH and HK testing sessions.

The results show improved HRR and greater  $V_T$  with HK when compared to HH. However, there was no difference in  $V_{CO_2}$  or subsequent performance in the Wingate Anaerobic

Test of power between both groups. HK posture improved HRR by 22 bpm when compared to HH. It was also shown that HK yielded greater  $V_T$  values when compared to HH (1.4 and 1.3 L/min respectively). The results indicate that HK and HH posture both influence speed of recovery during HIIT exercise but HK may be more effective. The improved cardiorespiratory response with HK is similar to what has been reported in the literature regarding reduced feelings of dyspnea in those who suffer from COPD (Banzett, 1988; Cavalheri, 2010; Kera & Maruyama, 2005; O'Neil, 1983). HK posture, which is associated with thoracic flexion and internal rotation of the rib cage, is reported in the literature to increase the zone of apposition (ZOA) of the diaphragm (Hruska, 1997; Hruska, unpublished manuscript). There is also a close association between respiration and heart rate called respiratory sinus arrhythmia (RSA) (Blain, 2004; Hayano et al., 1996; Hirsch & Bishop, 1981; Yasuma & Hayano, 2004). RSA has been shown to positively influence pulmonary gas exchange and improve cardiopulmonary energy expenditure (Hayano et al., 1996; Yasuma & Hayano, 2004). The improvement in HRR with HK may be attributed to the improved respiratory mechanics with HK posture, thereby influencing heart rate. A possible mechanism that may be attributed to the improved HRR with HK posture is the influence of the diaphragmatic vagal nerve (Bordoni & Zanier, 2013). The vagus nerve innervates the crural region of the diaphragm (Bordoni & Zanier, 2013). It has been observed that the vagus is connected to the trigeminal nerve, and stimulation of the trigeminal nerve influences cardiac arrhythmia and arterial pressure, leading to bradycardia (Bordoni & Zanier, 2013).

It is unknown whether different arm positions, such as HH and HK, influence respiratory mechanics and improve the ability to recover quickly from bouts of exercise. The majority of the literature has focused on the effect of different body positions on arm position and its influence

on respiratory mechanics, especially in those who suffer from respiratory diseases. This population is known to have a low tolerance to arm activities that is not only determined by strength or endurance but by position of the arm itself (Dolmage et al., 1993). It has been shown that arm elevation at 90° shoulder flexion and greater in those with respiratory dysfunction alters static ventilatory responses when compared to arms down and below 90° shoulder flexion (Dolmage et al., 1993; Martinez et al., 1991; McKeough et al., 2003). A study by Couser et al. (1992) examined respiratory and ventilatory muscle recruitment with arms elevated (AE) and arms down (AD) in healthy subjects. The authors reported an increase in metabolic demand ( $\text{VO}_2$ ,  $\text{VCO}_2$ , and heart rate) with AE when compared to AD associated with increases in  $\text{V}_E$ . In contrast to the present study, these subjects were seated and did not perform high intensity bouts of exercise before measurement of cardiorespiratory variables. The arm positions also differed greatly in both studies and did not follow similar protocols. In the present study,  $\text{V}_E$  was similar in HH and HK (40.4 and 39.4 L/min respectively). However  $\text{V}_T$  was significantly greater with HK (1.4 L/min) compared to HH (1.3 L/min). Couser et al. (1992) attributed the increase in  $\text{V}_E$  with AE to increased  $\text{V}_T$  and accessory muscle activity. In contrast to their findings,  $\text{V}_E$  was similar in HH and HK but  $\text{V}_T$  was greater with HK. The present study also showed greater  $\text{VCO}_2$  expiration with HK (1.1 L/min) than with HH (1.0 L/min), but results were not statistically significant. The present study suggests that HK posture placed the respiratory muscles such as the diaphragm and accessory muscles in a better position for contraction, thereby increasing the amount of air inhaled and exhaled per breath, as seen with an increase in  $\text{V}_T$ . Similarly, Couser et al. (1992) also suggested that AE increased diaphragmatic work and hindered rib cage and abdominal wall movement for full expansion during respiration. Although the present study and

previous study by Couser et al. (1992) differed in protocol and results, it can be suggested that there is a definite interaction between posture and cardiorespiratory mechanics.

HK posture is associated with thoracic flexion and internal rotation of the rib cage which increases the ZOA (Lee, 1883; Hruska, unpublished manuscript). In contrast, HH posture promotes thoracic extension which is associated with external rotation of the rib cage and reduced ZOA (Lee, 1883; Hruska, unpublished manuscript). The ZOA is the mechanical linkage between the diaphragm and ribcage (Boynton, 1991; Mead, 1979). It is suggested that the ZOA is maximized with thoracic flexion and is minimized with thoracic extension (Boyle, Olinick, & Lewis, 2010). Optimizing the ZOA allows the diaphragm to operate with maximal efficiency (Lando et al., 1999). The present study suggests that HK posture optimizes the ZOA and places the diaphragm in a better position for activation when compared to HH posture. Thoracic flexion was measured during HK posture to assure consistency with thoracic flexion between each rest interval. On average, subjects exhibited an increase in thoracic flexion with each rest interval from 14.6° in the first rest to 19.5° in the fourth rest interval. The increase in thoracic flexion with each rest interval may infer a natural increase in thoracic flexion with fatigue and exercise. An increase in thoracic flexion may further enhance the ZOA and diaphragm for contraction.

HH posture places the diaphragm in a suboptimal position, decreasing its mechanical efficiency. A decrease in the ZOA reduces the ability of the diaphragm to contract effectively, due to its poor position along its the length-tension curve (Boyle et al., 2010; Lando et al., 1999). Elevating the arms to 90° or more of shoulder flexion as observed with HH posture changes the impedance of the torso, rib cage, and abdominal wall (Couser et al., 1992; Dolmage et al., 1993; McKeough et al., 2003). Raising the arms causes a passive stretch of the thoracic wall and abdominal muscles, which may place them in a less effective position for assisting in respiration

(Couser et al., 1992; Dolmage et al., 1993; McKeough et al., 2003). HH position may cause faulty posture alignment for effective respiration, placing the abdominal muscles in an over lengthened position (Boyle et al., 2010). The abdominal muscles play an important role in respiration, especially during the exhalation phase. The abdominal muscles oppose the diaphragm during contraction and provide resistance for displacement and rib cage expansion (De Troyer & Estenne, 1988; Hruska, 1997). The opposition of the abdominal muscles on the diaphragm maximizes the ZOA (Hruska, 1997). An over lengthened abdominal region may reduce its ability to effectively oppose the diaphragm, leading to less effective respiratory mechanics.

HK posture, which assimilates the bracing posture strategy popularly used in patients with COPD to relieve their feelings of dyspnea and improve respiratory mechanics, may also benefit healthy subjects. The present study suggests that HK posture may not only improve diaphragm position but also enhance abdominal muscle function. The present study showed increased  $\dot{V}CO_2$  values with HK (1.1 L/min) when compared to HH (1.0 L/min), although not statistically significant. It is suggested that the HK posture improved exhalation ability of the abdominal muscles, leading to a slight increase in  $\dot{V}CO_2$ . Cavalheri et al. (2010) investigated the effects of arm bracing on respiratory muscle strength and pulmonary function in patients with COPD. The results showed greater maximal inspiratory and expiratory pressures with arms braced when compared to unbraced arms. The results of the previous study along with the findings of the present study suggest that bracing the arms improves respiratory function by decreasing the postural demands of these muscles (diaphragm, intercostal, abdominals, and accessory muscles) during HK.



Kera and Maruyama (2005) also supported the idea of improved force generating capabilities with a braced posture. They examined the effects of posture on respiratory activity of the abdominal muscles. The results showed increased abdominal activity with the braced position (seated, elbows on knees) and was attributed to the enhanced position of the abdominals when the trunk is flexed. The authors also suggested that the abdominals in this position elicited a greater stretch reflex during expiration, thereby increasing inspiration and, thus, reducing feelings of dyspnea in the subjects. Similarly, in the present study, there was a significant increase in  $V_T$  with HK (1.4 L/min) in comparison to HH (1.3 L/min), suggesting an increased ability to inspire and expire per breathe assuming HK posture. Subjects also reported more ease in breathing assuming the HK posture when compared to HH.

Based on the literature and the findings presented here, HK posture may improve respiratory mechanics thereby improving the ability for heart rate to recover faster from high intensity bouts of exercise. However it is still unknown whether HK or HH posture used during the recovery phase of exercise influences subsequent performance because there was no significant difference in the Wingate Anaerobic test mean power in the present study.

### **Summary**

The results of the study show that HK posture improved HRR when compared to HH by 22 bpm. Greater  $V_T$  values with HK (1.4 L/min) in comparison to HH (1.3 L/min) suggests improved respiratory function with HK posture. The positive influence of HK during the recovery interval of HIIT relate well to the literature regarding the arm bracing strategy used in patients with COPD to relieve dyspnea and improve respiratory function. The similar position of the trunk and arms when braced and with HK may be why these two postures display similar cardiorespiratory responses. In contrast, HH and arm elevation to 90° of shoulder flexion and

greater resulted in a less efficient respiratory musculature activity, which may explain the smaller HRR and lower  $V_T$  values in the present study. The differences found in the present study and literature may be due to the differences in methodology and subject characteristics. However, the results of the literature and present study show a definite connection between posture and cardiorespiratory responses during exercise and recovery.

## Chapter V

### Summary, Conclusions, and Recommendation

#### Summary

There is a lack of literature regarding the effects of different postures, such as hands on knees (HK) and hands on head (HH), used as a form of immediate recovery during bouts of high intensity interval training (HIIT). The limited research in the apparently healthy population focused on three positions, supine, seated, and upright and the physiological responses after exercise (Takahashi et al., 2000)(Takahashi et al., 2000). Other forms of recovery have also been examined such as active and passive strategies and their influence on subsequent performance (Castagna et al., 2008; Connolly, 2003; Graham, 2003; Taoutaou et al., 1996). However, upright standing postures like HK and HH are more commonly found in the exercise and sports field setting. It is clear that there is a definite connection between posture and cardiorespiratory mechanics shown in this study.

Subjects in the current study consisted of 20 Division II female soccer players recruited from the university. Each subject was familiar with HIIT which was part of their regular training. However each subject was not familiar with the Wingate Anaerobic Test protocol, which may be why there was no effect on subsequent power performance. Motivation can also play a crucial role in determining power performance (Hopkins, Schabort, & Hawley, 2001). The motivation of athletes may differ in field base settings from those in the laboratory which may have an effect on performance in tests. There is also conflicting evidence in the use of the Wingate Anaerobic Test as an assessment of anaerobic power in field sports like soccer (Karakoc et al., 2012; Keir et al., 2013; Krustup et al., 2006; Meckel et al., 2009), which may have also contributed to this study's results.

The present results suggest that HK may be more beneficial than HH as a form of immediate recovery posture. On average, subjects exhibited an increase in thoracic flexion with each rest interval from  $14.6^\circ$  in the first rest to  $19.5^\circ$  in the fourth rest interval. It could be suggested that fatigue may induce a natural increase in thoracic flexion. The ability to recover faster from a bout of exercise may influence subsequent performance, though the present study found no significant difference in mean anaerobic power. There was also no significant difference in mean carbon dioxide elimination ( $V_{CO_2}$ ) in the two postures. However, HK posture resulted in significantly improved heart rate recovery (HRR) and tidal volume ( $V_T$ ) values in comparison to HH, suggesting an improved cardiorespiratory function assuming HK posture.

### **Conclusion**

Based on the findings of this study, there were significant improvements in HRR and  $V_T$  during the recovery interval of HIIT when assuming HK posture. The subsequent power performance of a Wingate Anaerobic Test and  $V_{CO_2}$  did not display any significant difference between the two postures. Therefore, HK posture may be more beneficial than the popularly advocated HH posture as a form of immediate recovery from HIIT.

### **Recommendations**

**Future Research.** Postures like HK and HH as a form of immediate recovery from multiple bouts of exercise have not yet been fully understood, thus more research is needed in this area. The lack of research in athletes and the use of different recovery strategies between multiple intervals of exercise are needed for further development of strategies to optimize recovery.

Repeating the use of HK and HH posture in the present study with athletes in various sports may provide further insight to the applicability of these postures in a variety of sports. It is recommended that future studies implement shorter work-to-rest ratios to observe differences in recovery with varying rest and work periods. It is also recommended that subsequent performance tests to determine if HK and HH posture influences ensuing performance, resembles the movement pattern of the athlete in their sport.

**Practical Applications.** The ability to recover faster from multiple bouts of exercise may serve to improve subsequent performances. Based on the findings of this study, there was no effect of HK or HH posture on subsequent power performance. However, HK posture induced significantly improved HRR and  $V_T$  values in comparison to HH. The positive effects of HK posture on HRR and  $V_T$  may suggest improved cardiorespiratory mechanics when assuming this posture during the recovery interval of HIIT. Subjects also reported more ease in breathing assuming the HK posture when compared to HH. The results of this study imply that HK posture may be more beneficial than HH posture as a form of immediate recovery during HIIT

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Appendix A  
Informed Consent



## INFORMED CONSENT FOR EXERCISE TESTING

You are invited to participate in a research study conducted by Joana Houplin, from the Department of Physical Education, Health, and Recreation at the Western Washington University. This study involves research on immediate forms of recovery postures. Immediate recovery posture is a strategy used typically in an attempt to recover faster from multiple bouts of exercise such as high intensity interval training (HIIT). The ability to recover faster from a bout of exercise may influence subsequent performance positively. The purpose of this research is to investigate the effects of two different immediate forms of recovery postures of hands on head (HH) and hands on knees (HK) during HIIT in trained female soccer players. HH posture is generally observed in the sports field setting and is advocated by coaches to be more advantageous than HK. There is limited research on these two recovery postures in healthy, trained subjects, however the HK posture is commonly used in the clinical rehabilitation setting with people who suffer from respiratory disease to relive feelings of shortness of breath.

The two postures, HH and HK, require subjects to assume specific positions. The HH posture requires subjects to stand erect, clasping their hands on top of their head. The HK posture requires subjects to place their hands on their knees, elbows locked, and flexing their thoracic spine. Each posture will be held for the entire recovery period. Depending on the randomization of postures, hands on knees (HK) posture requires additional measurement of thoracic flexion with inclinometers at the same time of insertion of 2-way breathing mouth piece. Researchers will measure thoracic flexion while assuming HK posture to assure consistency of flexion between rest intervals. The testing session consists of four submaximal treadmill runs at 90-95% of HR max for 4 minutes with a 3 minute recovery between intervals. A workload of 4 sets of four minute intervals at 90-95% of HR max, with 3 minute recovery periods has been popularly used in soccer training regimens (Helgerud et al., 2007; McMillan, 2005). Since HIIT improves cardiorespiratory fitness and is widely used in the sport of soccer, it is appropriate to use in the current study to assess the effects of different postures as a form of immediate recovery strategy.

Given your participation, you will meet for three testing sessions at Western Washington University Exercise Physiology Laboratory. The sessions will include the following expectations.

Session one (baseline measurements and familiarization with equipment): Baseline measurements include pulmonary function tests consisting of vital capacity (VC), forced expired volume in one second ( $FEV_1$ ),  $FEV_1/VC$ , and MVV. Height and weight are also measured and used to calculate BMI. Measuring VC requires subjects to inspire maximally and then expire maximally into the spirometer as forceful and rapidly as possible. Performing this maneuver requires subjects to place their mouth around the mouthpiece, forming a tight seal with their lips. A nose clip is also attached to the subject to prevent air escaping through the nose. From this maneuver we can also measure  $FEV_1$  and calculate  $FEV_1/VC$ . Measuring MVV requires a

similar set up but subjects are asked to breathe as fast and deep as possible for 12 seconds. Familiarization of treadmill and Wingate protocol is performed and also used to determine running speed and resistance, respectively. Determination of running speed will require subjects to perform standardized warm-up which consists of jogging on the treadmill for 5 minutes at a speed that elicits 70% of HR max. After 5 minute warm up, speed is increased until target HR (90-95% HR max) is reached and at a speed that subjects can maintain for 4 minutes. Subjects are encouraged to reach target HR within 1-1.5. Determination of running speed requires subjects to perform only one interval (4 minutes of running followed by 3 minutes of recovery) followed by a Wingate test of anaerobic power. The Wingate anaerobic power test requires subjects to pedal on a cycle ergometer as fast as possible for 30 seconds with resistance. The resistance is relative to each subject's body weight and is calculated with an equation ( $0.075 \text{ kp} \cdot \text{kg}^{-1}$ ).

Session two: (randomization of hands on head or hands on knees testing): Researchers will go through testing procedures with subjects. Subjects are fitted with HR monitor followed by standardized warm-up. Speed is increased until subject reaches target HR (90-95% HR max) within 1-1.5 minutes and is instructed to stay there by adjusting the speed. Subjects are instructed to keep pace with treadmill for the full 4 minutes of running and immediately after 4 minutes are instructed to step to the sides of the treadmill and assume recovery position assigned for the testing session. At that same time, researchers will insert a 2-way breathing mouthpiece valve interfaced with the metabolic cart, and application of a nose clip. This is done for data collection of variables analyzed during the 3 minutes of recovery. You may experience some discomfort breathing such as dry mouth while wearing the mouthpiece and nose clip during the 3 minutes. This procedure is repeated four times, all subjects will perform a total of four 4 minute running intervals (4 X 4), and four 3 minute recovery intervals for each session. Immediately after the last 3 minute recovery interval, detachment of mouth piece and nose clip is done and subjects are transferred from the treadmill to the cycle ergometer for the Wingate Test. This is done as efficiently as possible to reduce transition time from testing procedures. The Wingate anaerobic power test requires subjects to pedal on a cycle ergometer as fast as possible for 30 seconds with a set resistance.

Session three: (posture not assumed in second session): The third testing session will follow the same protocol to session two except the subject will assume the recovery position not used in the second testing session.

As with any exercise, there are risks of muscle, tendon, ligament, injuries and fatigue. Some discomfort when performing HIIT and Wingate test of anaerobic power is expected, as it is asked that you give maximal effort during both sessions. Supervision of exercise and standardized warm up are done to minimize risk of injury. You may withdraw from participation in this study at any time, without penalty.

The benefits of this research are that you will know if there is an advantage in using the HH or HK posture as a strategy to recovery faster from high intensity interval exercise. Furthermore, subjects will know if assuming HH or HK posture during recovery will have an influence on high intensity anaerobic performance. This information may be used in training as a way to recover more rapidly from exercise.

Any questions you may have regarding this study procedures will be answered by the primary researchers, Joana Houplin and Dr. Lorrie Brilla who can be contacted at houplij@students.wvu.edu or 360-490-0305 and Lorrie.Brilla@wvu.edu or 360 650-3056.

Any questions about your rights as a research subject should be directed to the WWU Human Protections Administrator (HPA), 360-650-3220. If any injury or adverse effect of this research is experienced you should contact Joana Houplin, Dr. Lorrie Brilla, or the HPA.

Any and all data collected will be kept completely confidential and will be stored and analyzed by subject number only. The data will be stored on a password protected computer and will be stored separately from the consent forms in order to protect the anonymity of the participants. Only the primary researchers will have access to your records.

Your signature indicates that you have read and understand the information provided above, that you willingly agree to participate, that you may withdraw your consent at any time and discontinue participation without penalty, that you have received a copy of this form, and that you are not waiving any legal claims, rights or remedies.

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Participant Name (Printed)

Date

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Participant Signature

Appendix B  
Human Subjects Activity Review Form

## Human Subjects Activity Review

### 1. What is your research question, or the specific hypothesis?

The experimental hypothesis states that recovery postures of hands on head (HH), and hands on knees (HK) during the recovery period of high intensity interval training (HIIT) will have an effect on ventilation (VE), breathing frequency ( $f_b$ ) and heart rate recovery (HRR) in soccer players. In addition, there is also an effect of the recovery postures on a subsequent Wingate test of anaerobic power.

### 2. What are the potential benefits of the proposed research to the field?

The ability to buffer metabolic by products of exercise, including hydrogen ions ( $H^+$ ) and carbon dioxide ( $CO_2$ ) is crucial in maintaining acid-base homeostasis during exercise (Stringer, Casaburi, & Wasserman, 1992). It is well known that failure to maintain acid-base homeostasis during exercise can have detrimental effects on performance (Costill, Verstappen, Kuipers, Janssen, & Fink, 1984; Powers & Howley, 2009). Pulmonary ventilation plays an essential role in maintaining acid-base homeostasis by regulating blood acidity (pH) with expiration of  $CO_2$  by the lungs (Powers & Howley, 2009; Stringer, 1992). The ability to expire additional  $CO_2$  produced from high intensity exercise suggests an increase in ventilation thereby maintaining blood pH (Stringer et al., 1992). An increase in ventilation to expire additional  $CO_2$  during the recovery phase of submaximal HIIT in the different postures may show a more efficient respiratory musculature in the specific recovery posture.

During exercise, the increase in heart rate is due to increased sympathetic activation and vagal withdrawal (Imai et al., 1994). The reduction in heart rate immediately after exercise is suggested to be from sympathetic withdrawal (Savin, Davidson, & Haskell, 1982) and parasympathetic reactivation (Buchheit et al., 2007; Buchheit, 2006; Cole et al., 1999; Savin et al., 1982). There is increasing interest in the literature in the regulation of heart rate after exercise, more specifically the immediate reduction in heart rate known as heart rate recovery (HRR) (Borresen & Lambert, 2007, 2008; Buchheit et al., 2007; Buchheit, 2006; Cole et al., 1999; Imai et al., 1994; Morise, 2004). HRR is defined as the rate of immediate decrease in heart rate after moderate to heavy exercise to pre-exercise levels (Borresen & Lambert, 2008). There are a few ways to measure HRR, but the most commonly used and simplest method is taking the absolute difference in HR immediately after exercise and 60 seconds later (Cole et al., 1999).

It has been suggested that measuring heart rate recovery (HRR) immediately after exercise may serve as a simple and valuable tool for monitoring training status of athletes (Borresen & Lambert, 2008). Fitter subjects have quicker heart rate responses at the onset of exercise

and also after exercise when compared to sedentary subjects (Bunc, Heller, & Leso, 1988; Javorka et al., 2002). It has been suggested that training, results in enhanced HRR immediately after exercise due to increased parasympathetic tone (Yamamoto et al., 2001). Conversely, a decrease in HRR after training is shown to negatively impair performance associated with training induced fatigue (Lamberts et al., 2009). Lambert et al. (2009) demonstrated that HRR in well trained endurance athletes responds to changes in training load. The authors suggested that a prolonged HRR after an applied training load may predict accumulated fatigue from previous training load thereby blunting future performance. It is therefore plausible to suggest that measuring HRR immediately after exercise provides valuable information for monitoring an athlete's response to training.

Soccer players depend on both aerobic and anaerobic energy systems for performance. It is important to train at game like intensities to improve performance. A mode of training that is popular used in soccer is high intensity interval training (HIIT). It has been defined as repeated bouts of exercise close to maximal effort ( $> 90\%$  of  $VO_{2peak}$ ) separated with few minutes of rest (Gibala & McGee, 2008). A workload of 4 sets of 4 minute intervals at 90-95% of HR max, with 3 minute rest periods has been popularly used in soccer training regimens to improve cardiorespiratory fitness in contrast to continuous long running at lower intensities (Helgerud et al., 2007; McMillan, 2005). Since HIIT improves cardiorespiratory fitness and is popularly used in the sport of soccer, it is appropriate to use in the current study to assess the effects of different postures as a form of immediate recovery strategy.

The HH posture which has been traditionally advocated by coaches to be more effective than the non-traditional posture of HK lacks evidence for support. The majority of the literature that has examined the use of HK posture is in a population who suffer from respiratory disease like chronic obstruction pulmonary disease (COPD) (Banzett, Topulos, Leith, & Nations, 1988; O'Neill & McCarthy, 1983). It has been suggested that this posture improves respiratory function when compared to elevating arms above the shoulder (McKeough et al., 2003).

The ability to recover more rapidly in-between bouts of exercise may positively influence subsequent performance which is crucial in the sport of soccer which requires multiple bouts of high intensity runs with little recovery (Bangsbo, 2006; Krstrup et al., 2006; Mohr et al., 2003). Thus, it may help to identify a posture that has a positive influence on recovery from HIIT.

### **3. What are the potential benefits, if any, of the proposed research to the subjects?**

After completion of the study, subjects will know if there is a benefit in using the HH or HK posture as a strategy to recovery faster from high intensity endurance exercise. Furthermore, subjects will know if assuming HH or HK posture during recovery will have an influence on subsequent performance. This information may be used in training as a way to recover more rapidly from exercise.

**4. a. Describe how you will identify the subject population, and how you will contact key individuals who will allow you access to that subject population or database.**

The population sample will consist of female soccer players between the ages of eighteen and twenty-three who have experience in HIIT and at least three years of playing competitive soccer. A form requesting permission to contact the athletes will be given to and signed by the head soccer coach of the team before the researcher contacts the athletes. It is understood that all subjects participate in soccer year-around. Athletes will be instructed to continue in their normal training (including resistance training, conditioning, and sports specific practice), except during the day of testing.

**b. Describe how you will recruit a sample from your subject population, including possible use of compensation, and the number of subjects to be recruited.**

At least twenty subjects will be recruited to participate in this study. The soccer players will be recruited from Western Washington University. Inclusion for this study demands that subjects be free of any musculoskeletal or neurological impairment or injury. Athletes who participate in this study will not be compensated for their participation.

**5. Briefly describe the research methodology. Attach copies of all test instruments/questionnaires that will be used.**

**Instrumentation:** Pulmonary measures are performed using a Parvomedics (Sandy, UT) spirometer in the Exercise Physiology Lab at Western Washington University. The submaximal treadmill runs are performed in the laboratory on a Precor treadmill. The intensity of submaximal treadmill runs simulated game like soccer training intensities of 90-95% of HR max. Heart rate is monitored with a Polar heart monitor (Lake Success, NY). The submaximal treadmill testing consists of four intervals of four minutes of running with a three minute recovery assuming one of the two postures. HRR is measured at the beginning of the three minutes of recovery for the first minute. Ratings of perceived exertion (RPE) are measured using the Borg 6-20 scale during the last minute of running period and also during every minute of the three minutes of recovery along with minute ventilation ( $V_E$ ), and breathing frequency ( $f_b$ ) during the recovery phase of the testing. A Parvomedics TrueOne (Sandy, UT) Metabolic Cart is used to measure  $V_E$  and  $f_b$ . Thoracic flexion when assuming hands on knees (HK) posture will be measured with inclinometers to assure consistency of back flexion between recovery periods. The Wingate test for anaerobic power is performed on a Monark cycle (Sweden) ergometer, modified for electronic capture interfaced with a computer. Mean anaerobic power was measured in watts over a 30 second period. The

resistance used is relative to each subject's body weight ( $0.075 \text{ kp} \cdot \text{kg}^{-1}$ ). Subjects are instructed to pedal as fast as they could for the entire 30 second period while the computer records the revolutions of the flywheel.

**Measurement techniques and procedures:** The researcher explained the study and the time involved to complete study. Prior to testing, subjects are informed of testing procedures and are provided with an informed consent document. The subjects are told that hands on head (HH) requires them to stand erect with their hands clasped on top of their head; hands on knees (HK) requires them to place their hands on their knees, elbows locked and flexing their thoracic spine. Assuming the HK posture requires additional measurement of thoracic flexion using inclinometers to assure consistency of back flexion between recovery periods. It is also explained that each posture is held for the entire recovery period. Each subject completed one day of baseline measurements which included pulmonary function tests consisting of vital capacity (VC), forced expired volume in one second ( $\text{FEV}_1$ ),  $\text{FEV}_1/\text{VC}$ , maximal voluntary ventilation (MVV), and Body mass index (BMI) from height and weight.

Familiarization with the treadmill is also performed on the same day as baseline measurements. During this time determination of speed to elicit 90-95% of HR max is done by having subjects warm up for five minutes at a speed that elicits 70% of HR max. After the five minute warm up, speed is increased until target HR is reached and at a speed that subjects can maintain running for the full 4 minutes. Subjects are encouraged to reach target HR at a comfortable speed within 1-1.5 minutes. Treadmill incline is set at 0 % for all subjects during all testing sessions. Familiarization with the Wingate test of anaerobic power is also performed after familiarization with treadmill protocol. Following baseline measurements, two testing sessions are conducted with one week of rest between sessions. Each testing session consists of four submaximal treadmill runs at 90-95% of HR max for 4 minutes with a three minute recovery between intervals. A Wingate anaerobic power test for 30 seconds is then performed immediately after the last three minute recovery interval of the high intensity interval training (HIIT).

Subjects are instructed to not participate in any high intensity activity the day before testing, so that fatigue from previous activity will not affect testing session. Subjects are also encouraged to not consume caffeine the day of testing and to get a minimum of seven hours of sleep the night before testing. Verbal encouragement is made to all subjects during all treadmill testing sessions and Wingate test.

The submaximal treadmill testing is used to simulate intensities experienced in the field so that application of activity is similar to what subjects undergo on a daily basis in training. Subjects are asked to sit on a stool placed on the treadmill for preparation of testing which consists of HR monitor attachment and explanation of Borg RPE chart. Subjects warm up on the treadmill for 5 minutes at 70% HR max. After the 5 minute warm up, speed is increased until subject reaches target HR (90-95% HR max) within 1-1.5 minutes and is instructed to



stay there by adjusting the speed. Subjects are instructed to keep pace with treadmill for the full 4 minutes of running and immediately after 4 minutes are instructed to step to the sides of the treadmill and assume recovery position assigned for testing session. At the same time insertion of a 2-way breathing mouthpiece valve interfaced with the metabolic cart, and application of a nose clip is done for data collection during the 3 minutes of recovery. Hands on knees (HK) posture requires measurement of thoracic flexion with inclinometers at the same time of inserting the 2-way breathing mouth piece. During the 3 minutes of recovery  $V_E$ , fb, and RPE are measured every minute. These values are averaged over the 3 minutes for analysis. HRR is commonly defined as the difference in HR at the end of exercise and then 60 seconds later (Cole et al., 1999). Similarly, in this study, HRR is measured immediately at the beginning of the 3 minutes of recovery for the first minute, and the average of first minute in each rest interval is used for analysis. The HR monitor is worn throughout the entire session which includes HIIT and the Wingate Anaerobic Power test. Subjects perform a total of four 4 minute running intervals (4 X 4), and four 3 minute recovery intervals for each session. Immediately after the last 3 minute recovery interval, preparation for the Wingate Test is done, which includes detachment of mouth piece and nose clip. This is done as efficiently as possible to reduce transition time from treadmill testing procedures.

The Wingate Anaerobic Power test is immediately performed after the last 3 minute recovery interval of the HIIT. Prior to testing, resistance and seat height are adjusted accordingly. The resistance is relative to each subject's body weight ( $0.075\text{kp}\cdot\text{kg}^{-1}$ ). Subjects are asked to pedal as fast as possible for the entire 30 second trial while the computer counts the revolutions of the flywheel. Verbal encouragement is given throughout the 30 seconds.

**6. Give specific examples (with literature citations) for the use of your test instruments/questionnaires, or similar ones, in previous similar studies in your field.**

The effects of different immediate forms of recovery postures between bouts of exercise on respiratory mechanics, HRR, and mean anaerobic power have not been specifically measured in a similar manner to this study. However the comparisons of HK, considered a bracing posture has been compared to non-braced postures like hands on head and the ventilatory responses associated with them. This position is often used by patients with COPD to relieve their dyspnea, however the reasons for this are unclear (Banzett et al., 1988). Cavalheri et al. (2010) investigated the effects of arm bracing posture on respiratory muscle strength and pulmonary function in patients with COPD. Twenty subjects with COPD were recruited for the study. All subjects performed lung function testing with a spirometer and maximal respiratory pressures were also assessed in both braced and unbraced positions. Both positions were assessed standing up either with a support or without. The height of the support was adjusted to the level of the ulnar styloid process with a  $30^\circ$  trunk flexion with

elbows flexed. Similarly in this study a spirometer will be used to assess respiratory function since it is readily available and accurate.

**7. Describe how your study design is appropriate to examine your question or specific hypothesis. Include a description of controls used, if any.**

This study is a single group repeated measures design to analyze the effect of different postures as a form of immediate recovery from high intensity interval training (HIIT). Twenty volunteers will participate in the study. Hands on head posture will be compared to hands on knees posture during the recovery period of HIIT and ventilation, breathing frequency, and HRR will be compared in both postures. Also a Wingate test of anaerobic power to measure mean power and if the two postures have an influence on recovery and subsequent performance. The study will also employ a t-test to compare the means of hands on head and hands on knees cardiorespiratory mechanics and also mean power between the two postures.

This study design is appropriate to examine the specific hypothesis, investigating the effect of different recovery postures as a way to recover faster from a bout of work and its influence on subsequent performance. The study will examine the differences in minute ventilation, breathing frequency, and HRR during the recovery period and also mean anaerobic power after HIIT to observe the effects of posture on subsequent performance.

**8. Give specific examples (with literature citations) for the use of your study design, or similar ones, in previous similar studies in your field.**

Multiple studies have employed similar protocol using repeated measures design when investigating the effects of braced positions like hands on knees and unbraced positions like hands on head on cardiorespiratory mechanics (Banzett , 1988; Cavalheri , 2008; Kera & Maruyama, 2005). The measurement of two different postures requires comparison of the individual under both conditions, a repeated measures design is necessary.

**9. Describe the potential risks to the human subjects involved.**

As with any exercise, there are risks of muscle, tendon, ligament injuries and fatigue that will be present. Some discomfort when performing the HIIT and Wingate test of anaerobic power is expected, as it is asked that the subjects give maximal effort during both sessions.

**10. If the research involves potential risks, describe the safeguards that will be used to minimize such risks.**

In order to ensure the safety of all subjects, each exercise will be explained in detail and monitored carefully for proper form and safe mechanics. Multiple research assistants will be present during exercise testing to ensure safety during treadmill runs. The subjects are also experienced soccer players who are familiar with the training protocols in this study.

**11. Describe how you will address privacy and/or confidentiality.**

Any and all data collected will be kept completely confidential and will be stored and analyzed by subject number only. The data will be stored on a password protected computer and will be stored separately from the consent forms in order to protect the anonymity of the participants. Only the primary researchers will have access to the records.

Appendix C  
Data Collection Sheets

## Subject Info Sheet, Baseline, First Session and Second Session Collection Sheets

### Subject Info & Baseline Measurements

Data Collection	Date	Time	Subject	#:		
Baseline					Age (yrs):	
Session # 1					Height (in:)	
Session # 2					Weight (lbs):	
					BMI:	
Injury History	<i>Back injury?</i>					
Soccer experience						
Consent form	<i>Yes / No</i>	Coin Flip: Heads - (HH) Tails - (HK)			<i>Session # 1:</i>	
Explanation of Test	<i>Yes / No</i>				<i>Session # 2:</i>	
Explanation of Posture	<i>Yes / No</i>					
Equipment	<i>Towels (2)</i>					
	<i>Stop watch</i>					
	<i>Heart rate monitor</i>					
	<i>BORG RPE</i>					
	<i>Nose clip</i>					
	<i>2-way mouth piece</i>					
	<i>Athletic tape</i>					
	<i>Inclinometers &amp; Marker</i>					
Room Set up	<i>Coin</i>					
	<i>Calibration of Metabolic cart</i>					
	<i>Spirometer set up</i>					
	<i>Set computer interfaced with cycle ergometer</i>					
	<i>Seat height for Wingate</i>					
<i>Resistance for Wingate</i>						
Baseline Measurements	<b>Anthropometric</b>					
	<i>Body Mass Index (BMI)</i>					
	<b>Pulmonary Function Tests</b>					
	<i>Type</i>	<i>1</i>	<i>2</i>	<i>3</i>		

	VC			
	FEV <sub>1</sub>			
	FEV <sub>1</sub> /VC			
	MVV			

<b>High Intensity Interval Training Heart rate ranges</b>	<i>Calculated HR max (220-age)</i>
	<i>70% HR max</i>
	<i>90-95% HR max</i>
<b>Wingate Test</b>	<i>Resistance (0.075kp*kg-1):</i>
	<i>Seat height</i>
<b>Treadmill Familiarization</b>	<i>Explanation</i>
	<i>Attach HR monitor</i>
<b>Warm up</b>	<i>Jog on treadmill @ 70% HR max for 5 minutes</i>
	<i>Increase speed until target HR is reached (4 mins running)</i>
	<i>Is target HR reached with 1-1.5 minutes?</i>
	<i>Make note of speeds at 70 &amp; 90-95% HR max</i>
<b>Wingate Familiarization</b>	<i>3 minutes rest after treadmill</i>
	<i>Set calculated resistance &amp; adjust seat</i>
	<i>Explanation of test</i>
	<i>Tell subject: "This is a maximal effort test" "Pedal as fast as you can in 30 seconds while seated" "I will count down from 3..2..1...GO"</i>

## Subject Info Sheet, Baseline, First Session and Second Session Collection Sheets

### First Session

Testing Session # 1															
<b>Preparation for HIIT Treadmill session</b> <b>Preparation of cycle ergometer for Wingate (Seat height and computer)</b>			<i>Coin flip for randomization of posture: Heads (HH) Tails (HK) - KYPHOMETERS</i>												
			<i>Explanation of testing procedures</i>												
			<i>Explanation of postures</i>												
			<i>Explanation of BORG RPE chat</i>												
			<i>HR monitor attachment</i>												
			<i>Place piece of tape on nose</i>												
			<i>Palpate &amp; mark T1 &amp; T12</i>												
			<i>Cycle ergometer prep (seat height &amp; computer)</i>												
HIIT Treadmill															
<b>HIIT Protocol</b>			<i>5 min warm up at 70% HR max</i>												
			<i>Increase speed to target HR (90-95% Hrmax)</i>												
			<i>Was speed reached w/in 1-1.5 min?</i>												
			<i>Notify subject each minute</i>												
			<i>Notify subject of last minute and 30 secs</i>												
			<i>Tell subject "Step to the sides of the treadmill and assume posture"</i>												
			<i>*HK* Research assistants ready w/ Kyphometers to measure thoracic flexion immediately during 3 mins</i>												
			<i>*HK* Tell subject "Step to the sides of the treadmill and stand erect while reaseach assistants zero out kyphometers and Research assistant inserts 2-way mouth piece and nose clip <b>*DO NOT LET SUBJECT HOLD MOUTH PIECE*</b></i>												
Rest Interval # 1				Rest Interval # 2				Rest Interval # 3				Rest Interval # 4			
	1 min	2 min	3 min		1 min	2 min	3 min		1 min	2 min	3 min		1 min	2 min	3 min
Thoracic flexion				Thoracic flexion				Thoracic flexion				Thoracic flexion			
HR max:				HR max:				HR max:				HR max:			
HR min:				HR min:				HR min:				HR min:			
HRR:				HRR:				HRR:				HRR:			
RPE				RPE				RPE				RPE			
Mean:				Mean:				Mean:				Mean:			
Rest Interval Protocol															
<b>Rest Interval Protocol</b>			<i>Notify subject every minute of rest</i>												
			<i>Notify subject 30, 10, 5 seconds of last minute</i>												
			<i>Research assistants removes mouth-piece &amp; nose clip</i>												
			<i>Tell subject to immediately step on treadmill and increase speed to target HR speed</i>												
			<i>Repeat X 2</i>												
			<i>Last rest interval: notify subject at last minute to transition to cycle ergometer rapidly</i>												
			<i>Research assistant prints off Metabolic report</i>												
Wingate Test															
<b>Wingate Protocol</b>			<i>Detachment of mouth piece &amp; nose clip</i>												
			<i>Rapid transition to cycle ergometer</i>												
			<i>Tell subject to wait for command: "3..2...1 GO" (RA sets resistance as subject starts to pedal)</i>												
			<i>Provide Verbal encouragement</i>												
			<i>Check computer for saved file</i>												
			<i>Floppy disk</i>												
			<i>Subject # - Group #- Testing session #</i>												

## Subject Info Sheet, Baseline, First Session and Second Session Collection Sheets

### Second Session

Testing Session # 2															
Preparation for HIIT Treadmill session Preparation of cycle ergometer for Wingate (Seat height and computer)	<i>Coin flip for randomization of posture: Heads (HH) Tails (HK) - KYPHOMETERS</i> <i>Explanation of testing procedures</i> <i>Explanation of postures</i> <i>Explanation of BORG RPE chat</i> <i>HR monitor attachment</i> <i>Place piece of tape on nose</i> <i>Palpate &amp; mark T1 &amp; T12</i> <i>Cycle ergometer prep (seat height &amp; computer)</i>														
HIIT Treadmill															
HIIT Protocol	5 min warm up at 70% HR max Increase speed to target HR (90-95% Hrmax) Was speed reached w/in 1-1.5 min? Notify subject each minute Notify subject of last minute and 30 secs Tell subject "Step to the sides of the treadmill and assume posture" *HK* Research assistants ready w/ Kypometers to measure thoracic flexion immediately during 3 mins *HK* Tell subject "Step to the sides of the treadmill and stand erect while reaseach assistants zero out kypometers and Research assistant inserts 2-way mouth piece and nose clip <b>*DO NOT LET SUBJECT HOLD MOUTH PIECE*</b>														
Rest Interval # 1				Rest Interval # 2				Rest Interval # 3				Rest Interval # 4			
	1 min	2 min	3 min		1 min	2 min	3 min		1 min	2 min	3 min		1 min	2 min	3 min
Thoracic flexion				Thoracic flexion				Thoracic flexion				Thoracic flexion			
HR max:				HR max:				HR max:				HR max:			
HR min:				HR min:				HR min:				HR min:			
HRR:				HRR:				HRR:				HRR:			
RPE				RPE				RPE				RPE			
Mean:				Mean:				Mean:				Mean:			
Rest Interval Protocol															
Rest Interval Protocol	Notify subject every minute of rest Notify subject 30, 10, 5 seconds of last minute  Research assistants removes mouth-piece & nose clip  Tell subject to immediately step on treadmill and increase speed to target HR speed  Repeat X 2 Last rest interval: notify subject at last minute to transition to cycle ergometer rapidly  Research assistant prints off Metabolic report														
Wingate Test															
Wingate Protocol	Detachment of mouth piece & nose clip Rapid transition to cycle ergometer Tell subject to wait for command: "3..2...1 GO" (RA sets resistance as subject starts to pedal) Provide Verbal encouragement Check computer for saved file Floppy disk Subject # - Group #- Testing session #														



## Appendix D

### Randomization of testing session

## Subject Testing Data

Hands on knees (HK) and Hands on head (HH) randomization of testing

<b>Subject</b>	<b>Session_1</b>	<b>Session_2</b>
2	HK	HH
3	HK	HH
4	HK	HH
5	HH	HK
6	HH	HK
7	HK	HH
8	HH	HK
9	HH	HK
10	HH	HK
11	HH	HK
12	HH	HK
14	HK	HH
15	HK	HH
16	HH	HK
17	HK	HH
18	HK	HH
20	HH	HK
21	HH	HK
22	HK	HH
23	HK	HH
24	HH	HK

## Appendix E

### Raw Data

## Subject Characteristics

<b>Subject</b>	<b>Age (yrs)</b>	<b>Weight (kg)</b>	<b>Height (m)</b>	<b>BMI (kg/m<sup>2</sup>)</b>
2	19	61	1.727	20.6
3	20	68	1.753	22.2
4	19	68	1.575	27.3
5	19	74	1.753	24.0
6	18	58	1.626	21.8
8	19	57	1.575	22.9
9	20	59	1.702	20.2
10	21	76	1.753	24.7
11	21	73	1.753	23.7
12	22	75	1.803	23.1
14	21	60	1.702	20.6
15	22	61	1.676	21.8
16	20	70	1.778	22.0
17	20	68	1.753	22.0
18	19	62	1.753	20.3
20	21	65	1.676	23.3
21	21	59	1.702	20.2
22	22	75	1.778	23.6
23	21	62	1.753	20.1
24	21	55	1.549	22.9

Subject Test Data  
Pulmonary Function Data

Subject	VC (L)				FEV <sub>1</sub> (L)			
	1	2	3	Average	1	2	3	Average
2	4	4.2	4.3	4.2	3.3	3.3	3.1	3.2
3	4.1	4.2	4.2	4.2	3	3.5	3.5	3.3
4	3.4	3.5	3.7	3.5	2.9	3.1	3.1	3.0
5	4.3	4.4	4.4	4.4	3.3	2.5	1.7	2.5
6	3.9	3.9	3.9	3.9	2.8	3	2.9	2.9
7	4.4	4.5	4.5	4.5	3.1	3.2	3.3	3.2
8	3	3	3	3.0	2.5	2.5	2.3	2.4
9	3.3	3.5	3.5	3.4	3.1	3.2	3.2	3.2
10	4.4	4.3	4.4	4.4	3.4	3.5	3	3.3
11	3.7	3.6	3.4	3.6	2.1	2.3	2.8	2.4
12	5.4	5.4	5.3	5.4	4.5	3.6	4.4	4.2
14	4.2	4.3	4.1	4.2	2.6	2.5	3.3	2.8
15	3.9	3.7	4.1	3.9	2.7	2.5	2.7	2.6
16	4.2	4.3	4.3	4.3	3.6	3.7	3.2	3.5
17	3.7	3.8	3.8	3.8	3.3	3.3	3.3	3.3
18	4.1	4.2	4.2	4.2	3.5	3.5	3.4	3.5
20	2.4	3.6	3.5	3.2	2.1	3.2	3.3	2.9
21	3.5	3.5	3.8	3.6	2.7	2.6	3.3	2.9
22	4.8	4.5	4.8	4.7	3.7	3.1	3.4	3.4
23	4.6	4.9	4.7	4.7	3.2	3.4	2.8	3.1
24	3.3	3.4	3.4	3.4	2.6	2.1	2.6	2.4

Subject	FEV <sub>1</sub> /VC (L)				MVV (L/min)			
	1	2	3	Average	1	2	3	Average
2	0.83	0.79	0.72	0.8	160	158	153	157
3	0.73	0.83	0.83	0.8	139	151	144	145
4	0.85	0.89	0.84	0.9	119	121	114	118
5	0.77	0.57	0.39	0.6	132	135	132	133
6	0.72	0.77	0.74	0.7	133	131	133	132
7	0.70	0.71	0.73	0.7	145	133	139	139
8	0.83	0.83	0.77	0.8	125	116	120	120
9	0.94	0.91	0.91	0.9	143	134	127	135
10	0.77	0.81	0.68	0.8	136	125	129	130
11	0.57	0.64	0.82	0.7	135	128	128	130
12	0.83	0.67	0.83	0.8	144	145	134	141
14	0.62	0.58	0.80	0.7	134	149	148	144
15	0.69	0.68	0.66	0.7	132	132	122	129
16	0.86	0.86	0.74	0.8	130	125	134	130
17	0.89	0.87	0.87	0.9	142	140	140	141
18	0.85	0.83	0.81	0.8	144	147	141	144
20	0.88	0.89	0.94	0.9	123	120	123	122
21	0.77	0.74	0.87	0.8	151	149	155	152
22	0.77	0.69	0.71	0.7	156	155	160	157
23	0.70	0.69	0.60	0.7	92	84	89	88
24	0.79	0.62	0.76	0.7	114	94	117	108

## Subject Test Data

## Thoracic Flexion with Hands on knees (HK)

<b>HK Thoracic Flexion (°)</b>				
<b>Subject</b>	<b>Rest_1</b>	<b>Rest_2</b>	<b>Rest_3</b>	<b>Rest_4</b>
2	16	28	38	40
3	20	10	25	20
4	20	13	10	30
5	18	14	20	20
6	10	17	18	10
8	20	10	10	12
9	8	12	18	10
10	20	21	26	36
11	15	10	10	11
12	10	10	10	20
14	10	10	10	17
15	17	10	20	20
16	12	20	15	15
17	10	10	10	20
18	10	10	10	15
20	12	30	28	20
21	18	16	15	12
22	16	18	20	26
23	20	30	22	20
24	10	10	16	16

## Subject Testing Data

## Heart Rate Recovery (HRR) Data

<b>HRR after 1 minute</b>			
<b>Subject</b>	<b>HK</b>	<b>HH</b>	<b>Difference</b>
2	64	48	15
3	42	24	18
4	45	24	22
5	52	19	33
6	52	42	10
8	49	32	16
9	56	44	12
10	66	37	29
11	56	44	12
12	42	26	16
14	74	44	30
15	63	45	18
16	55	31	24
17	58	39	19
18	59	17	42
20	53	26	27
21	34	12	23
22	27	13	14
23	54	24	30
24	58	31	27



## Subject Testing Data

Tidal Volume ( $V_T$ ) Data

$V_T$ (L/min)		
Subject	HK	HH
2	1.27	1.32
3	1.28	1.09
4	1.42	1.28
5	1.39	1.72
6	1.41	1.42
8	1.45	1.16
9	1.36	1.19
10	1.18	1.06
11	1.40	1.18
12	1.45	1.50
14	1.65	1.41
15	1.47	1.46
16	1.44	1.29
17	1.67	1.49
18	1.99	1.63
20	1.39	1.26
21	1.31	1.36
22	1.76	1.74
23	1.52	1.33
24	1.09	0.99

## Subject Testing Data

VCO<sub>2</sub> Data

<b>VCO<sub>2</sub> (L/min)</b>		
<b>Subject</b>	<b>HK</b>	<b>HH</b>
2	0.929161	0.895098
3	0.940183	0.80675
4	1.10898	1.07927
5	1.315148	1.314694
6	1.043551	0.905784
8	1.15731	0.888735
9	1.078568	0.89538
10	1.084357	0.919852
11	1.350807	1.153145
12	1.380094	1.176748
14	0.925553	1.537504
15	0.936077	1.01467
16	1.183713	1.325485
17	1.403439	1.045582
18	1.156459	0.857788
20	1.063442	0.803105
21	0.947332	0.911619
22	1.347573	1.245385
23	1.245345	0.952981
24	1.039241	0.918368

Appendix F  
Statistical Data

## Statistical Analysis Tables

T-Test

Table 12: HRR

Paired Samples Test								
	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
Pair 1 HK_1_HRR - HH_1_HRR	21.85000	8.26709	1.84858	17.98088	25.71912	11.820	19	.000

Table 13:  $V_T$ 

Paired Samples Test								
	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
Pair 1 TV_HK - TV_HH	.10112	.15393	.03442	.02907	.17316	2.938	19	.008

Table 14:  $V_{CO_2}$ 

Paired Samples Test								
	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
Pair 1 VCO2_HK - VCO2_HH	.09942	.21175	.04735	.00032	.19852	2.100	19	.049

Table 15: Mean Anaerobic Power

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	VCO2_HK - VCO2_HH	.09942	.21175	.04735	.00032	.19852	2.100	19	.049