The Effects Of Two Immediate Forms Of Recovery Postures On Cardiovascular Function And Subsequent Power Performance

Western Washington University, Department of Physical Education, Health and Recreation
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Dr. Gordon Chalmers
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The Problem and Its Scope

Introduction

Athletes, from novice to elite, are always looking for different 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). 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). Oftentimes, during high intense 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).

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 performance (Connolly et al., 2003). However the effects of upright standing and immediate recovery between sets of work have not been as well examined. Upright standing posture is more commonly used in the exercise and sports field setting (Buchheit, Al Haddad, Laursen, & Ahmaidi, 2009). 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 strategy of HK has been most commonly used in clinical rehabilitation settings for individuals with COPD to help with dyspnea (Banzett, Topulos, Leith, & Nations, 1988; O’Neill & McCarthy, 1983).

The ability to buffer metabolic by products of exercise, including hydrogen ions (H⁺) and carbon dioxide (CO₂) 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 CO₂ by the lungs (Powers & Howley, 2009; Stringer, 1992). The ability to expire additional CO₂ 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 CO₂ during the
recovery phase of testing in the different postures may show a more efficient 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, another with hands on knees. The hands on head posture required subjects to stand erect with their hands clasped on top of their head. Hands on knees posture required subjects to brace their hands on their knees with locked elbows, maintain a straight back, and placing most of their weight on their hands and thighs. The study focused on observing minute ventilation ($V_E$), breathing frequency ($f_b$), and heart rate recovery (HRR) during the recovery intervals of high intensity interval training (HIIT). To further examine the effects of the different postures on subsequent performance, a Wingate 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 hands on head (HH), and hands on knees (HK) during the recovery period of high intensity interval training (HIIT) on ventilation (VE), breathing frequency ($f_b$) and heart rate recovery (HRR). In addition, there is also no effect of the recovery postures on a subsequent Wingate test of anaerobic 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 looking at seated and supine positions after exercise, and also passive and active recovery on repeated sprint ability, not many have looked at the effects of upright standing postures like hands on head (HH) or a hybrid hands on knees (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 HH posture required subjects to stand erect with their hands clasped on top of their head, and the HK posture required subjects to place their hands on their knees with locked elbows while maintaining a straight back, placing most of their weight on their thighs. The current study will determine if HH or HK posture influences respiratory mechanics and heart rate
recovery (HRR) during the recovery period of high intensity interval training (HIIT) and subsequent anaerobic power performance during a Wingate test in Division II female soccer athletes.

Endurance sports like soccer are very 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, Wisløff, & Hoff, 2001) requiring a lot of demand from the aerobic system (Bangsbo, 1994). It has been observed that high intensity interval training (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$_{2\text{max}}$) 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 maintain high intensity bouts of work in soccer is crucial in training so that game performance can be improved. The current study will determine if different posture strategies have an influence on cardiorespiratory mechanisms during the recovery period of HIIT. Additionally, this study also examines the effects of the recovery postures on a subsequent performance of anaerobic power in a Wingate 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 to the best of their ability.
3. Fitness levels of subjects varied. Subjects were not asked to change any training regimens during the study. But 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 test for anaerobic 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).

Closed kinetic chain: In terms of upper extremity, defined as a combination of links in which the distal segment (the hand) is fixed and force is transmitted directly through the hand (Escamilla & Yamashiro, 2009).

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 and internal intercostals (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 diaphragm is the major muscle of inspiration (Powers & Howley, 2009).

Inspiratory Muscles: Include the diaphragm, external intercostals, scalnes 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).

Open chain kinetic: In terms of upper extremity, defined as the distal segment (the hand) moving freely with or without external resistance (Escamilla & Yamashiro, 2009).

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, 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 (TV): 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).
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 influence respiratory mechanics in normal subjects.

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 focus is on the respiratory muscles and how they become fatigued during high intensity endurance exercises. 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
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 Test of Anaerobic Power 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). However, abdominal muscles, which include the external oblique abdominis, internal oblique abdominis, rectus abdominus and transverse abdominus, along with the internal intercostals are active during expiration also aid during inspiration due to the improved length-tension relationship of the diaphragm during expiration (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). 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 ± 7% at 95% work load and 41± 9% at 85% work load) which was due to an increased in breathing frequency (f_b) at a constant tidal volume (VT). 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 min^{-1} and tidal
volumes 2.50-3.25 l, assuring that exhaustion occurred within 10 minutes. Training was increased either by \( f_b \) or VT. 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 l \( \text{min}^{-1} \)) 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). 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 / \( \text{min} \)). 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\textsubscript{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 ± 3.1% and -23.8 ± 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 diaphragm is contributing 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$_{2 \text{max}}$ 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 an 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.

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 vital capacity (VC) from 2.64 liters (L) with arms down to 2.44 L. With arms elevated, a small increase in functional residual capacity (FRC) occurred but was not significant. When examining the effect of arm position on ventilation at rest, there was no change during SAE in VO₂, maximum carbon dioxide production (VCO₂), and minute ventilation (Vₑ) when compared to the control. There was a significant increase in fₑ in breaths per minute (16.2 control, 17.9 SAE, and 17.5 UAE) and a decrease in tidal volume (Vₜ) 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 VO₂ (227 to 308 L min⁻¹), VCO₂ (195 to 263 L min⁻¹), Vₑ (9 to 11.9 L min⁻¹), and fₑ (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, $V_E$ 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. During exercise, when the arms are elevated, it affects the accessory muscles so they are less effective by shortening them. 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, Couer, & 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 (Pg) and pleural (Pp) 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. $\text{VO}_2$ and $\text{VCO}_2$ increased within 30 seconds of AE. $\text{VO}_2$ peaked at 20% towards the end of the second minute. Similarly, $\text{VCO}_2$ peaked at 23% with AE towards the end of the two minutes. This increase in $\text{VO}_2$ and $\text{VCO}_2$
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 ($P_{I_{\text{max}}}$) dropped significantly after AE from 54 to 48 cm H$_2$O, however both maximal expiratory mouth pressure ($P_{E_{\text{max}}}$) and $P_{d_{\text{max}}}$ did not change with AE, 73.6 (AD) to 70.9 cm H$_2$O (AE) and 61.4 (AD) to 63.7 cm H$_2$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 $P_{d}$ and $P_{g}$ 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 $V_E$ 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 $P_{d}$ increased with AE. The difference between end inspiratory and end expiratory transdiaphragmatic pressures ($\Delta P_{d}$) increased significantly and peaked at 14.1 cm H$_2$O during the last 15 seconds of AE (Martinez et al., 1991) when arm position changed from AD to AE. Changes in $\Delta P_{d}$ were related to $V_T$, 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 chronic airflow obstruction (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 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. Minute ventilation (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 (PgI) significantly increased with AE when compared to AD. There no significant changes in end inspiratory endosophageal pressure (PpII), end expiratory endosophageal (Pple), and end expiratory gastric pressure (PgE) (Couser et al., 1992). As a group, transdiaphragmatic (Pdi) increased from 21.4 cm H$_2$O AD to 26.5 cm H$_2$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 $VO_2$, $VCO_2$, $V_E$, and $V_T$ before returning to baselines at 6 and 7 minutes. It is was also observed that 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 ($VO_2$, $VCO_2$, 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 Pdi through higher Pg 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° of shoulder flexion. McKeough, Alison, & Bye (2003) investigated the effect of arm elevation greater than 90° on lung volume in subjects with COPD and healthy subjects. It was hypothesized that arm elevation greater than 90° would alter lung volume, specifically inspiratory capacity
(IC) and total lung capacity (TLC) when compared to arms below 90° 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° shoulder flexion, arms at 90° shoulder flexion, and arms greater than 90° shoulder flexion. Subjects were encouraged to reach maximum attainable shoulder flexion, and ranged between 153-170° 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 when compared to arms were less elevated. 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 chronic airflow obstruction (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 (Pple) and gastric (Pg) pressures 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$_2$ and 32.2% increase in VCO$_2$ after two minutes of AE. A similar rise was observed in normal subjects. There was a significant difference in baseline Pdi between diaphragm weakness subjects and normal subjects (37.60 cm H$_2$O and 138.30 cm H$_2$O respectively), however both demonstrated comparable changes in Pdi during AE when compared to pre-AE. Pple also demonstrated a similar trend indicating that normal subjects and those with diaphragm weakness respond in the same way to AE in regard to Pple. The rise in Pdi 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 the physical therapist in a 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).
As previously mentioned, the abdominal muscles are known to assist during respiration, and more importantly during expiration (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). 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 (PEmax) and inspiratory (PImax) efforts of mouth pressures. PEmax was measured at TLC and PImax was measured at RV. Lung capacities were measured with a spirometer: VC, inspiratory residual volume (IRV), expiratory residual volume (ERV), TV, 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\textsubscript{2}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 breath spontaneously (SB) with a normal rhythm under a load of 20 cm H\textsubscript{2}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 compared 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 main findings of this study found 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 chronic airflow obstruction (CAO) and chronic obstructive pulmonary disease (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 compared 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 ± 22 cmH₂O and 54 ± 24 cmH₂O 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 ± 37 cmH₂O) compared to 92 ± 37 cmH₂O without arms braced. A similar trend in MVV values was also observed, 42 L min⁻¹ arms braced and 38 L min⁻¹ 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 and accessory muscles) to generate more force when arms are braced.

A similar posture has been implemented in the manufacturing of walking aids in those with severe COPD to encourage the maintenance of physical activity (Solway, 2002; Probst, 2004). Solway et al. (2001) assessed at the short term effects of a rollator on functional exercise capacity in individuals with severe COPD. Functional exercise capacity was measured with a 6 minute walk test (6MWT). Subjects performed testing on two separate days which included two 6MWT on each day. Each subject walked with a rollator aid and then rested for one hour and walked without the aid. Randomization of test order was done on the first day and flipped on the second day of testing. 6MWT was conducted in an enclosed corridor that measured 60 m long and 3.4 m wide. Subjects were instructed to cover as much ground as possible during the test. Along with the 6MWT, a modified Borg scale was used to assess dyspnea. Cardiorespiratory function and gait were also measured which included oxygen saturation and HR which was measured with a finger probe pulse oximeter. Respiratory rate, minute volume, and the phase relationships between the rib cage and abdominal excursion were monitored with a respiratory
inductance plethysmograph. Gait analysis was assessed with a self-developed stride counter and walking speed was also calculated. Walking distance in the 6MWT increased with the use of the rollator in subjects who walked < 300 m unaided but was the same for subjects who walked > 300 m. It was also observed that rest time during 6MWT was reduced with the rollator when compared to without, 11.9 ± 5.8 seconds and 31.2 ± 8.7 seconds, respectively. There was no difference in oxygen saturation during the 6MWT with and without the rollator aid, however there was a slight decrease in HR with the rollator (102.5 beats/minute with rollator and 105.9 beats/minute without rollator). Respiratory rate, minute volume, and phase relationships between rib cage and abdominal excursion were not changed with the use of a rollator. The improvements in walking distance, rest time, and reduced feelings of dyspnea during 6MWT with a rollator aid were not associated with improved cardiorespiratory function or gait but were associated with improved accessory muscle use.

In contrast to the previous study, Probst et al. (2004) associated improved exercise capacity with the use of a rollator to increased ventilatory capacity and better walking efficiency. Unlike the previous study, Probst et al. (2004) assessed cardiorespiratory function with a portable metabolic cart which may have contributed to the differences found in both studies. The findings of Probst et al. (2004) show that walking distance improved significantly with rollator (462 m) then without (416 m) which is similar to the findings of the previous study. However, the increase in distance with the use of the rollator was attributed to higher walking speeds with the rollator (1.28 m/s) when compared to without (1.15 m/s).

These results differ from Solway et al. (2001) who found a slight decrease in walking speeds with the rollator when looking at the whole group but found no difference in those who walked < 300 m. The increase in walking speed with the rollator was also significantly related to
higher VO\textsubscript{2}, which showed a 4-10\% increase with the rollator. It was also observed that \(V_E\) and MVV increased slightly with the rollator but \(f_b\) was similar in both conditions. Even though \(V_E\) and walking distance increased with rollator, subjects reported reduced feelings of dyspnea when walking with the rollator as reported on a Borg scale.

The ability to brace and support the arms on a solid surface when compared to unsupported arms, influences pulmonary function, respiratory muscle strength, 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, Meste & Bermon, 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 ventilation ($V_E$), respiratory frequency ($F_R$), and tidal volume ($V_T$) 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. However 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). However, 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; Javorka 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$_{2\text{max}}$ 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$_{2\text{peak}}$) separated with few minutes of rest (Gibala & McGee, 2008). HIIT significantly improves VO$_{2\text{max}}$ and improvements in VO$_{2\text{max}}$ 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; Krustrup 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$_{2\text{max}}$. 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 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$_{\text{max}}$ (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$_{2\text{max}}$ 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$_{2\text{max}}$ in soccer players (63.4 ml/kg$^{-1}$ to 69.8 ml/kg$^{-1}$) 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 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.

As previously mentioned, 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 popularly used assessment of anaerobic power is the Wingate test which measures peak and mean anaerobic power (Zupan et al., 2009). The Wingate 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 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 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 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; Probst, 2004; Solway, 2002). Those who suffer from COPD report feelings of dyspnea during daily activities, especially activities that involve the use of upper extremities (Couser, 1992; Dolmage, 1993). 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; Probst, 2004; Solway; 2002). 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 there is an improved ability to recover faster from high intense bouts of exercise, there should be an influence on the 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 subsequent performances of collegiate female soccer players. Specifically, the study examined how the two different postures could possibly influence cardiorespiratory function during high intensity interval training and also subsequent performance in a Wingate Anaerobic Power test. The high intensity interval exercises were performed on a treadmill over three testing sessions where minute ventilation ($V_E$), breathing frequency ($f_b$), heart rate recovery (HRR) and a Borg rating of perceived (RPE) exertion were recorded during the recovery phase of the testing. Immediately after high intensity interval exercise performance, a Wingate 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 18-23 years old. All subjects in the study have trained using high intensity interval training (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, with the other two randomized on subsequent days. 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) assuming one of recovery postures. After each HIIT session a Wingate Test was performed. The Wingate tests were performed immediately after the last recovery interval in each session. The purpose of the Wingate 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 which included body mass index (BMI), vital capacity (VC), forced expired volume (FEV\(_1\)), FEV\(_1\)/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 speed which elicited 70% of their heart rate (HR) max at 0% grade followed by four running intervals at an intensity of 90-95% of HR max for four minutes, with a three minute recovery in-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 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 HR
max. Heart rate was monitored with a Polar heart (Lake Success, NY) monitor. The submaximal treadmill testing consisted of four intervals of four minutes of running with a three minute recovery assuming one of the two postures. HRR was measured at the beginning of the three minutes of recovery for the first minute. RPE was 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 $V_E$, and $f_b$ were recorded during the recovery phase of the testing. A Parvemedics TrueOne (Sandy, UT) Metabolic Cart was used to measure $V_E$, and $f_b$. The Wingate 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 ($0.075 \text{ kp*kg}^{-1}$). 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 hands on head (HH) required them to stand erect with their hands clasped on top of their head, and hands on knees (HK) required them to place their hands on their knees, elbows locked and maintaining neutral back. It was also explained that each posture was 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 (FEV$_1$), FEV$_1$/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 determination of speed to elicit 90-95% of HR max was done 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 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. Following baseline measurements, three 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 power test for 30 seconds was then performed immediately after the last three minute recovery interval of the high intensity interval training (HIIT).

Subjects were instructed to not participate in any high intensity activity the day before testing, so that fatigue from previous activity would not affect testing session. Subjects were 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 was made to all subjects during all treadmill testing sessions and Wingate test.

The submaximal treadmill testing was used to simulate intensities experienced in the field so that application of activity was similar to what 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 and explanation of Borg RPE chart. Subjects warmed up on treadmill for five minutes at 70% HR max. After the five minute warm up, speed was increased until subject reached target HR (90-95% HR max) within 1-1.5 minutes and was instructed to stay there by adjusting the speed. Subjects were instructed to keep pace with 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 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 was done for data collection during the three minutes of recovery. During the 3 minutes of recovery VCO$_2$, V$_E$, breathing frequency (f$_b$), and RPE were measured every minute. 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 beginning of the three minutes of recovery for the first minute, and the average of first minute in each session was used for analysis. The HR monitor was worn throughout the entire session which included HIIT and the Wingate Anaerobic Power 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 Test was made which included detachment of mouth piece and nose clip. This was done as efficiently as possible to reduce transition time from testing procedures.

The Wingate Anaerobic Power test was immediately performed after the last 3 minute recovery interval of the HIIT. Prior to testing, resistance and seat height were adjusted accordingly. The resistance used is relative to each subject’s body weight (0.075kp/kg). 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. A dependent t-test was used to analyze any differences due to the two treatments using the SPSS program. The dependent variables analyzed included HRR, V$_E$, and f$_b$ during each recovery posture and mean anaerobic
power (watts) from the Wingate Test of Anaerobic Power. Significance was defined as a p-value less than 0.05.


