RESISTIVE RESPIRATORY TRAINING AND ITS EFFECTS ON PULMONARY MECHANICS IN INDIVIDUALS DIAGNOSED WITH EXERCISE INDUCED ASTHMA

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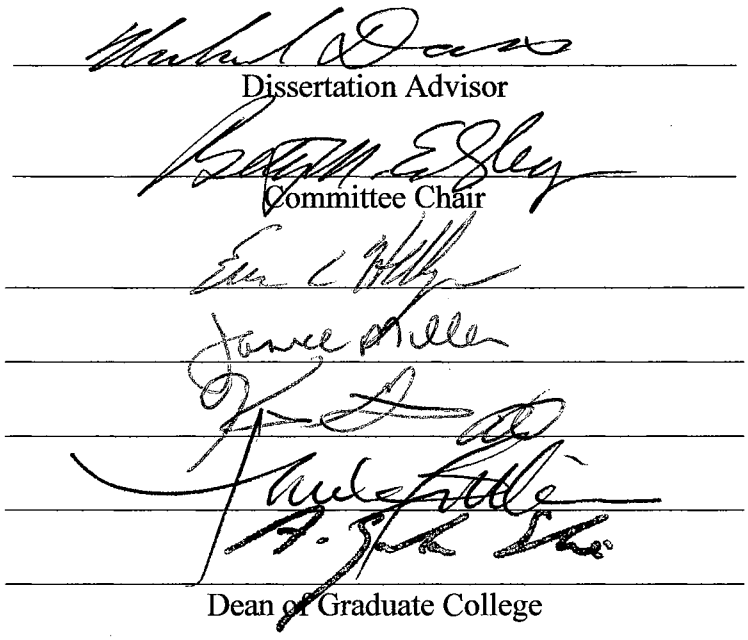

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CHAPTER 1

INTRODUCTION

Breathing is a mechanical process that depends on precise timing of neurological systems and respiratory muscle function. Most of the smooth muscle that line the respiratory tract receive efferent signals from respiratory centers in the "medullary neural networks and sensory reflex mechanisms" of the central nervous system (Rundell, Wilber, Lemanske, 2002). Accordingly, the thoracic cages' skeletal muscles responsible for inspiration include the diaphragm, external intercostals, sternocleidomastoid, anterior serrati and scalenes. Expiration is accomplished through passive relaxation of the aforementioned musculature coupled with the active contraction of the abdominal recti, the internal intercostals and other abdominal components (Guyton & Hall, 2000). These muscles, through sympathetic and parasympathetic input, must act together to efficiently move air in and out of the lungs in a normal at rest environment as well as under physically strenuous conditions. If the synergistic relationship between smooth and skeletal muscle is not harmonious, oxygen becomes depleted in the system and resultant physiological repercussions occur such as dyspnea, cyanosis and other pulmonary disorders (O'Connor, 2001).

Dynamic requirements on the breathing process that occur in sports and physical activity can become compromised, if not seriously impeded, in obstructive disorders and

pulmonary pathology (Guyton & Hall, 2000). A majority of breathing adaptation will occur without active control through parasympathetic contraction or relaxation of smooth muscle in the respiratory tract. However, to accommodate oxygen needs, skeletal muscles of respiration are recruited to inspire, opening the thorax and creating a decreased intrathoracic pressure which draws ambient air into the pleural cavity. Once the air has reached the alveoli and gas exchange has occurred, the residual air must be forcefully eliminated. Therefore, external muscles of expiration contract to create an increased pleural pressure thus forcing air out the respiratory tract (Guyton & Hall, 2000, McArdle, Katch and Katch, 1996). There are many timely and intricate actions that take place to allow for efficient breathing. If any structural or neurological pathology impedes this process, subsequent breathing disorders will arise, including inflammatory diseases and chronic obstructive disorders such as asthma (O'Connor, 2001).

Asthma is a chronic respiratory disorder "characterized by spastic contraction of the smooth muscle in the bronchioles, which causes extremely difficult breathing" (Guyton and Hall, 2000). In most cases, asthma is an inflammatory condition that reacts with heightened sensitivity to environmental triggers such as dust, pollen, chemicals, and other common allergens. In some cases, increased physical activity that causes a corresponding increase in minute ventilation and intake of ambient air may provoke bronchiolar hyperactivity and induce asthmatic constriction (Rundell, Wilber, Lemanske, 2002, Rupp, 1996).

Exercise-induced asthma (EIA), also referred to as exercise-induced bronchospasm or exercise-induced bronchoconstriction (EIB), is a physiological disorder of the respiratory system. It is typically characterized by coughing, wheezing, excessive sputum production, tightness of the chest and general dyspnea. These symptoms either occur minutes into strenuous activity, or they follow minutes after extended periods of physical exertion (Pinkowish, 2000, Lacroix, 1999, Rupp, 1996). Some authors have cited a range consisting of five to fifteen minutes following an exercise bout as the most susceptible time for symptoms to present (Houglum, 2001, Lacroix, 1999). An acute episode typically involves the release of inflammatory mediators system such as histamine, leukotrienes and prostaglandins. These mediators have local and general effects in the system such as bronchial wall edema formation, mucous plug formation, smooth muscle contraction and hypertrophy. It has been stated that long-term, aerobic-type activities such as running, cycling, and swimming are more prone to eliciting EIA symptoms, but no clear connection has been found supporting such a case (Rundell & Jenkinson, 2002). Exercise in cold dry air or air contaminated with noxious chemicals and closed environments such as ice skating arenas have been indicated as predisposing factors to EIA presentation (Tikkanen & Helenius, 1994, Strauss, et al., 1977, Gotshall, 2002, Fowler, 2001, Barfield & Michael, 2002, O'Kane & Woodford, 1999).

Although the exact etiology of EIA is unknown, many theories have been postulated as to explain this respiratory dysfunction. The two more widely accepted theories include the hyperosmolarity and thermal hyperemia theory. The hyperosmolarity theory describes a process of mucosal cell dehydration due to excessive ventilation of air that dries out the

epithelial cell lining increasing the osmolarity of these cells and thus provoking bronchoconstriction. The thermal hyperemia theory revolves around evaporation of cellular water following exposure to cold, dry air. A reflex vasodilation of the respiratory vasculature causes a transitory re-warming of the epithelial cells. The rapid flux of blood to the bronchial capillary beds initiates pro-inflammatory mediators that cause increased vascular permeability. An accumulation of edema within the structure of the bronchial walls ensues thereby limiting the diameter of the respiratory airways (Rundell, Wilber, Lemanske, 2002).

Nearly 90% of persons with asthma also suffer from EIA (Gotshall et al. 2000, Mickleborough, et al., 2003), causing a confounding mixture of symptomology and diagnosis. The difficulty in differentiating independent EIA from chronic asthma has caused many physicians to opt for extensive laboratory tests which typically involve expensive equipment and lab work. Symptoms associated with both chronic asthma and exercise-induced asthma such as bronchiolar hyperresponsiveness (BHR) tend to confound an accurate differential diagnosis causing confusion when selecting the specific treatment. Other EIA symptoms cited on medical history questionnaire that bear similarities with chronic asthma include abnormally tiring before fellow teammates, excessive drying of airways due to "mouth breathing" and lightheadness (Lacroix, 1999, Rupp, 1996). Diagnosis of exercise-induced asthma typically is conducted in a clinical setting such as a hospital or respiratory healthcare facility after a patient begins to experience the aforementioned dyspnic symptoms specifically following exercise (Hansen, 1982). Graded exercise challenges, bronchiolar provocation test using irritants

such as methacholine, and eucapnic voluntary hyperpnoea procedures have been used to identify exercise-induced asthma (ACSM, 2000, Rupp, 1996, Anderson, et al. 2001)

According to the American Thoracic Society (1994), assessment of asthmatic conditions requires baseline spirometric measures including FEV-1, Forced Vital Capacity (FVC) and Peak flow being acquired. Protocol for diagnosing EIA involves engaging the patient in an exercise challenge on an exercise modality such as a stationary bicycle or treadmill. The duration should be long enough for an appropriate assessment of pulmonary mechanics. It is important to note that presentation of symptoms are not necessary as pulmonary measures generally indicate the presence of an obstructive disorder (American Thoracic Society, 2000). During an exercise challenge, measures are periodically recorded according to a pre-established protocol and continued for approximately 20-30 minutes post exercise. If at any time during or following the exercise bout, the FEV-1 falls between 10-15% below baseline measures, the patient is typically diagnosed with EIA (ACSM, 2000, Hansen, 1996, Rundell, Wilber, Lemanske, 2002, Rupp, 1996). Much confusion remains for many investigators and medical practitioners regarding which form of asthma, if not both, an individual suffers. It is difficult to accurately differentiate EIA, a treatable transitory affliction, from chronic asthma due to their similar presentation of subjective symptoms and objective signs. Some authors (Rundell, Judelson & Williams, 2002) have stated that more accurate diagnosis of chronic asthma involves the inhalation of a bronchial irritant such as methacholine to exacerbate symptoms. Individuals who exhibit exercise-induced bronchoconstriction in response to

physical activity and no other chronic asthmatic symptoms can with assuredness be diagnosed through an exercise challenge (Rupp, 1996).

Impulse oscillometry (IOS) has been recognized as a viable diagnostic tool in the clinical community for accurately assessing lung obstruction arising from disorders such as asthma and related conditions (Klinnert, Larsen, Liu, 2003, Witt, Clark, 2002, Fischer, et al., 1999). Like other pulmonary function tests, the patient is asked to breathe normally through a mouthpiece connected to a heated pneumotach. As the patient ventilates, an impulse-generated waveform is transmitted into the airways and the reflection of these sound waves is recorded, providing a measure of the lung resistance and reactance.

The primary objective of any pharmacologic treatment of EIA is to improve bronchodilation through the decreasing inflammation and subsequent edema formation instigated by allergen or other triggers. Immediate treatment of exercise-induced asthma has traditionally centered on pharmacologic intervention with primarily short-term treatment with fast-acting bronchodilators such as beta-2 agonists including albuterol, bitolterol, pirbuterol and terbutaline (Houglum, 2001). This treatment option of a fast acting beta-2 agonist actually serves as a diagnostic tool for many physicians. EIA symptoms typically resolved quite efficiently with the short-acting agent whereas chronic asthma sufferers tend to need more elaborate treatment modalities (Rupp, 1996, LaCroix, 1999). Long-acting pharmacological agents such as salmetrol (a Beta-agonist), cromolyn sodium, Nedocromil sodium, corticosteroids, oral theophylline, and montelukast prevent the onset, shorten the duration, and/ or attenuate the symptoms associated with

bronchospasm (Lacroix, 1999, O'Kane, Woodford, 1999, Peroni, et al., 2002, Backer, 2002).

Many researchers and healthcare practitioners have recommended exercise as a potential mode of prevention for individuals suffering from exercise-induced asthma. Theoretically, increasing one's ability to reach maximal ventilatory rates and volumes decreases the impact and magnitude of EIA-related symptoms such as bronchospasm and constriction (Rupp, 1996, LaCroix, 1999, Barfield & Michael, 2002, Fowler, 2001, Carlsen & Carlsen, 2002, Sinha, 2003, Disabella & Sherman, 1998, Satta, 2000).

Respiratory muscle training has been investigated quite extensively on healthy, non-pathological subjects (Tzelepis, et al., 1994 Pardy, et al., 1998, Caine and McConnell, 1998, Inbar, et al., 2000, Chatham, et al., 1999, O'Kroy and Coast, 1993) as well as in other studies whose subjects suffered from such disorders as chronic asthma, emphysema and other forms of chronic obstructive pulmonary disorders (COPD) (Nield, 1999, Ramirez, et al., 2002). The theoretical underpinning of many of the aforementioned studies using pathological populations was to treat dyspnic, disordered breathing. By re-training neuromuscular control of the respiratory musculature, it was hypothesized that subjects could essentially return to a normal, efficient breathing rate and quality. A review of studies which targeted the respiratory- and endurance-enhancing benefits of respiratory training revealed a significant treatment effect on many pulmonary measures including an increase in general cardio-respiratory performance and endurance (Amonette and Dupler, 2001, Volianitis, et al., 2001).

Respiratory training devices have become commonplace in the endurance sport market (PowerLung®, Sportsbreather®). Most manufacturers claim that these devices restrict the amount of airflow either being inspired, expired or both. Through modulation of airflow, it is theorized that increased forces will be required to generate inspiration and expiration against specific resistance, thus eliciting the overload and progressive resistance exercise (PRE) principles. Both of these notions explain the building of skeletal muscle strength and hypertrophy through repeated increased force generation (McKardle, Katch &Katch, 1996). Use of such devices in clinical investigations (Pardy, 1998, Leith &Bradley, 1976, O'Kroy & Coast, 1993, Amonette & Dupler, 2001, Chatham, et al., 1999, Caine & McConnel, 1998) have produced significant results, prompting manufacturers to claim that it can optimize lung power, improve cardiovascular endurance, boost stamina, and decrease the subjective feeling of exhaustion (SportsBreather®, 2003).

PURPOSE OF STUDY

The purpose of this study is to determine if loading the respiratory skeletal musculature using a resistive respiratory training device will significantly decrease both subjective and objective respiratory measures arising from exercise-induced asthma. Exercise-induced asthma is a relatively common condition that affects physically active people at all levels. Although it may not be a debilitating disease like its associated chronic asthma, it has the ability to limit an individual's pulmonary function following exercise by inducing a hyperactivity of the bronchial airways. Traditionally, palliative treatment has consisted of pharmacologic intervention whereas non-pharmacologic modes of

preventative treatment have been encouraged yet seldom researched. By utilizing objective diagnostic measures, there is potential for another mode of treatment for EIA that could be used by athletes and the general population who suffer from this respiratory restrictive disorder.

HYPOTHESES

H1: There will be an increased positive effect on baseline (pre-training) pulmonary function test measures of the experimental group when compared to the control group following a five (5) week training regimen utilizing a resistive respiratory training device and protocol.

H2: There will be a positive effect in pulmonary mechanical response after exercise stress testing within the experimental group when compared to the control group following a five (5) week resistive respiratory training device and protocol.

H3: There will be a decreased sense of exertion measured by perceived modified Borg scale scores within the experimental group when compared to the control group following a five (5) week resistive respiratory training device and protocol.

H4: There will be no change in the experimental group's post-exercise stress testing measures of pulmonary resistance including magnitude of respiratory impedance (Z5), total respiratory resistance (R5), proximal respiratory

resistance(R20) and distal capacitive reactance (X5) following a five (5) week resistive respiratory training device and protocol.

H5: There will be a positive increase in the experimental group's values on post-exercise stress testing expiratory measures (FEV-1, $FEV_{25\%-75\%}$, P_{ex}) following a five (5) week resistive respiratory training device and protocol.

DELIMITATIONS

1. Subject population will consist of trained individuals as represented by their membership on a collegiate athletic team.

2. The subject pool will consist of several sports characterized generally as aerobic and anaerobic.

3. Subjects will be at different points of their respective competitive year.

4. Testing procedures will take place in a controlled environment using a treadmill.

5. All individuals will be diagnosed with EIA, but will not be differentiated between sole exercise-induced asthma and chronic asthma.

6. Gender, group, age and height will be recorded and the contributing variability will be controlled for through randomized assignment of subjects to experimental and control groups.

LIMITATIONS

1. Subjects in this study were all trained athletes. The results of this investigation cannot be applied to the general population.

2. The treatment regimen is constructed so that the individual subject must control their compliance with the protocol.

3. Treatment protocol involves the unsupervised use of the respiratory training device during the treatment period.

4. Subjects could potentially be taking long-term prescription medication for asthmatic conditions at the time of study. Long-term prescription medication regimens will not be interrupted and will be continued through duration of the study.

ASSUMPTIONS

1. Subjects will comply with resistive respiratory training with device for extent of five (5) weeks.

DEFINITION OF TERMS

AIRWAY HYPERRESPONSIVENESS: describes airways that narrow too easily or too much in response to a provoking stimulus. In asthma, airways can be hyper responsive to many different stimuli (American Medical Association, 2003).

ALLERGEN: a protein that causes one to have an allergic reaction. Examples include foods, animal dander and some drugs (Guyton & Hall, 2000).

ASTHMA: a chronic inflammatory disorder in which many cells play a role, in particular mast cells, eosinophils and T lymphocytes. In susceptible individuals this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness and cough particularly at night and/or in the early morning. These symptoms are usually associated with widespread, but variable airflow limitation that is at least partly reversible either spontaneously or with treatment. The inflammation also causes an associated increase in airway responsiveness to a variety of stimuli. (AMA, 2003)

BRONCHOCONSTRICTION: airflow limitation due to contraction of airway smooth muscle (Guyton & Hall, 2000).

BRONCHOSPASM: narrowing of bronchial tree tubes including the bronchi and bronchioles typically due to involuntary smooth muscle contraction (Guyton & Hall, 2000).

CHRONIC AIRFLOW LIMITATION: synonymous with Chronic Obstructive Pulmonary disorder (COPD). Disease state that restricts airflow entering and exiting the respiratory system (Rundell, Wilber & Lemanske, 2002).

DYSPNEA: dysfunctional or painful breathing (American College of Sports Medicine, 2000).

EDEMA: Residual collection of fluid in the capillary bed that permeates through the vessel wall into the interstitial space (Guyton & Hall, 2000).

EFFERENT: Motor neurons that carry impulse away from the brain causing an effect (McArdle, Katch and Katch, 1996).

EMPHYSEMA: A complex obstructive and destructive process causing excess air in the lung tissue (Guyton & Hall, 2000).

EXERCISE CHALLENGE TEST: graded exercise test used to assess pulmonary function and possible markers of pathology (ASCM, 2000)

FORCED VITAL CAPACITY(FVC): maximal volume of air exhaled with maximally forced effort from a position of maximal inspiration (American Thoracic Society, 2002).

FORCED EXPIRATORY VOLUME IN ONE SECOND (FEV-1): volume of air maximally exhaled in the first second of a forceful breath; typically 75% of the forced vital capacity (American Thoracic Society, 2002).

HYPEREMIA: An increased rate of blood flow to a tissue causing a cutaneous redness (Guyton & Hall, 2000).

HYPEROSMOLARITY: An increased concentration of particles (ie. ions) in solution (McArdle, Katch and Katch, 1996).

HYPERPNEA: Increased ventilation typically brought upon by exercise (McArdle, Katch and Katch, 1996).

IMPULSE OSCILLOMETRY: Diagnostic means of identifying pulmonary impedance and resistance through varying frequencies of pressure impulses (Jaeger-Tonnies, 2003).

LARYNX: Component of respiratory passageway that houses the vocal cords (Guyton & Hall, 2000).

INSPIRATORY MUSCLE TRAINING: increasing load or time of exercise on skeletal musculature controlling inspiration thus producing results of increased strength and endurance (Pardy, R.L., Reid, W.D., Belman, M.J., 1988).

FORCED EXPIRATORY VOLUME $_{25\%-75\%}$: mean forced expiratory flow during the middle half of the forced vital capacity (American Thoracic Society, 2002).

LUNG REACTANCE: the elasticity and mass inertia of the airways, lung tissue, thorax, and the inertia of the air within the bronchi (Finucane, K.E., Dawson S.V, Phelan, P.D., Mead, J., 1975).

LUNG RESISTANCE: summative restrictive forces created by the structure and function of the lung tissues airways (Finucane, K.E., Dawson S.V, Phelan, P.D., Mead, J., 1975).

MEDULLA OBLONGATA: Lower area of brain that controls respiration (Guyton & Hall, 2000).

NASOPHARYNX: cavity between nasal passage and primary air route of throat (Guyton & Hall, 2000).

PEAK EXPIRATORY FLOW: largest expiratory flow achieved with a maximally forced effort from a position of maximal inspiration. (American Thoracic Society, 2002).

PONS: component of mid-brain that aids in auto-regulatory control of breathing (Guyton & Hall, 2000).

RESIDUAL VOLUME: volume of air remaining in lungs after the most forceful exhalation (Guyton & Hall, 2000).

SPIROMETRY: medical test that measures the volume (or changes in volume of air) an individual inhales and exhales as a function of time (American Thoracic Society, 2002).

TOTAL LUNG CAPACITY: maximum volume to which the lungs can be expanded with the greatest possible effort (Guyton & Hall, 2000).

TIDAL VOLUME: total volume of air inspired or expired with each normal breath. (Guyton & Hall, 2000).

TURBINATES: small indentures in nasal cavity that produces eddying currents of passing air (Guyton & Hall, 2000).

VASODILATION: Vessel opening becoming larger allowing for increased flow (Guyton & Hall, 2000).

VITAL CAPACITY: the maximal volume of air exhaled from the point of maximal inhalation or the maximal amount of air inhaled from a point of maximal exhalation (American Thoracic Society, 2002).

CHAPTER 2

REVIEW OF THE LITERATURE

This chapter will review the anatomical and physiological relationships within the pulmonary system and the disorders, specifically exercise-induced asthma, that can adversely affect it. Content also will describe the effect of resistive respiratory muscle training through a comprehensive review of the literature.

Physiology of Breathing

Breathing is a critical, life-sustaining process both at rest and with increased physical activity (Wilmore & Costill, 1994, Powers & Howley, 2001). The mechanical process of transporting ambient air in and out of the lungs is commonly referred to as ventilation. As muscles surrounding the thorax contract and relax, the thoracic cavity which houses the lungs is constantly changing the amount of available gaseous space (Guyton & Hall, 2000). Through pressure gradients and the aforementioned muscular control, air is moved into the lungs where an exchange of gaseous oxygen and carbon dioxide occur. Partial pressures of oxygen and carbon dioxide between lung tissue and the nearby capillary beds cause a "diffusion process called respiration" (O'Connor, 1994, p. 59). Oxygen is transported through the alveolar walls and into the blood stream destined for cellular respiration while carbon dioxide is moved into the lung space for subsequent elimination from the body (Guyton & Hall, 2000).

Neural control of this process involves a cascade of impulses being sent from the medulla oblongata and pons of the midbrain initiating a contraction of inspiratory muscles. Primarily, the diaphragm and external intercostals but, when increased ventilation is required, recruit the sternocleidomastoid, anterior serrati and scalene to expand the rib cage and increase the intrathoracic space (Guyton & Hall, 2000). Interestingly, the lungs are not directly attached to the rib cage, but rather encased in pleural sacs containing fluid that aids in decreasing friction between the lungs and rib cage during respiration. These sacs are, attached to the thoracic cage so the pleural cavity does adjust and conform to the dynamic space of the chest cavity (Wilmore, Costill, 1994). The enlargement of the thoracic cavity creates a negative pressure in relation to the external atmospheric pressure and draws ambient air in through the mouth and nose into the nasopharynx. As the air is passed through the turbinates or cavities within the nose, the air is filtered, warmed and humidified as it passes into the pharynx, larynx and eventually, the trachea (Wilmore, Costill, 1994). Conditioned air is then divided into the respiratory tree of the lungs through the bilateral bronchial branches into the smaller bronchioles and terminal bronchioles eventually into the respiratory bronchioles and alveolar sacs. At this point of the respiratory zone, the one-cell-layer walls of the alveoli provide a transit system for gas exchange. The carbon dioxide that passively collects in the alveolar sacs is then conducted back through the same path as the oxygen traveled (Powers & Howley, 2001).

It is important to note that as physical activity increases, as with exercise, the ventilatory rate increases to meet demands for oxygen consumption and carbon dioxide removal. Guyton and Hall report that with, "strenuous exercise, oxygen consumption and carbon

dioxide formation can increase as much as 20-fold." (2001, p.479). However, it seems that many other contributing factors control the rate of respiration and that chemical concentrations of oxygen and carbon dioxide are the not the main determinants for increased respiratory rate (McArdle, Katch, Katch, 1996). Also, quiet breathing, which normally occurs through the nasal passage at a rate of five (5) to six (6) liters per minute, typically changes route and rate (20-30 l/min) with increased exercise and is conducted through the mouth bypassing the conditioning effects of the nasopharynx (Powers & Howley, 2001, Guyton & Hall, 2000). These factors will play a significant role in how individuals who engage in exercise accommodate increased ventilation of unconditioned air, especially in pulmonary obstructive disorders.

Diagnostic Measures of Lung Function

Traditional spirometry involves the measurement of inspired and expired air through a flow sensitive mouthpiece (Powers & Howley, 2001). Protocols suggested by many authors (Rupp, 1996, LaCroix, 1999) and organizations such as the American Thoracic Society (1994) and ACSM (2002) consist of preliminary measures followed by an exercise challenge Other modes include a pharmacological challenge utilizing bronchial irritants such as methacholine and mannitol or an eucapnic voluntary hyperpnea (EVH). The "gold standard" test as described by Hallstrand, et al., (2002) involves a seven (7) minute exercise challenge conducted on treadmill or stationary bike at 85%-90% of predicted maximal heart rate followed by serial spirometry (Hansen, 1982, Lacroix, 1999, Fowler, 2001). LaCroix (1999) further suggests that no warm up should be allowed before exercise challenge as it might blunt opportune bronchospasm. Carlsen, Engh &

Mork (2000) conducted a study of importance in regards to exercise load when they found that loads that increase heart rate to approximately 95% of predicted maximum are more provocative of EIB.

Typically, forced vital capacity (FVC), forced expiratory flow in one second (FEV1), forced expiratory volume in 25%-75% of exhalation, and peak expiratory flow are the measures used to diagnose any asthmatic condition. ACSM (2000: 131) indicates that a fifteen (15%) percent decrease in FEV-1 during the post exercise period qualifies a person as having EIA. Furthermore, Fowler (2001) and Rundell (2002) categorize the severity of EIA as a ten (10%) to twenty (20%) percent drop in FEV-1 being mild, twenty (20%) to forty (40%) percent being moderate, and a greater than 40% drop characterized as severe EIA.

Recently, the use of lung resistance during rest has been used to quantify pulmonary disorders such as asthma, chronic obstructive pulmonary disorder (COPD), chronic heart failure and cystic fibrosis with significant success (Marotta, et al., 2003, Fischer, et al., 1999, Witte, et al., 2002). Although methods such as body plethysmography have been noted to produce the most accurate measures of lung resistance, the individual being tested must sit in a thermally equilibrated chamber making an exercise challenge extremely difficult (Rundell, Wilber & Lemanske, 2002). Impulse oscillometry (IOS) represents a forced oscillation method to gauge lung resistance through normal tidal breathing. Individuals breathe normally into a mouthpiece connected to a pneumotach and a small loudspeaker that generates random impulses at a frequency of five to thirty-

five hertz (5-35 Hz). These impulses are then superimposed on the normal tidal breaths and "real-time recordings of mouth pressure and flow are used to estimate total respiratory system impedance (Z) and its two components, resistance (R) and reactance (X)" (Ortiz & Menendez, 2002, p. 532). In light of the convenience and ease of IOS measurements, some authors such as Johnson, et. al. (1999) warn that such measures taken without a maximal inspiration might distort lung capacity volumes, thereby decreasing the accuracy of such measurements. However, the authors do agree with Marotta, et al. (2003) and Fischer, et al. (1999) that at rest, forced oscillation is a reliable indicator of upper versus lower airway obstruction. Furthermore, Scmekel and Smith (1997) conducted a study using forced oscillometry to assess frank asthma after bronchial provocation through isocapnic hyperventilation of dry air. The results were significant as the sensitivity for asthma detection through forced oscillation technique was eighty-nine (89%) percent and specificity for asthma was one hundred (100%) percent.

To obtain a subjective measure of physical activity, research studies have typically enlisted the use of Borg's 6-20 category scale of rating perceived exertion (RPE). As ACSM (2002) states, the RPE, "is a valuable and reliable indicator in monitoring and individuals exercise tolerance" with consideration for, "...personal fitness level, environmental conditions and general fatigue levels." (p. 78). Borg's original study conducted in 1970 produced correlative results between subject's heart rates and their personal estimation of exertion. As the subject's heart rate increased due to increase in activity level, the sensation of more strenuous work load also increased. It was concluded through his work and subsequent work of Russell (1997) and Robertson (1998), that the

RPE was a valid instrument for subjective estimation of perceived exertion. Borg (1982), further clarifies that the modified category version (appendix) of the RPE scale would be appropriate for determining other subjective symptoms such as breathing difficulties.

Interestingly, Suzuki, et al. found no significant correlation between perceived exertion scores during exercise within subjects trained with a resistive inspiratory training device after a four (4) week period. However, several studies have utilized Borg's RPE scale or a modification thereof, (Chatham, et al, 1999, Caine & McConnell, 1998, Volianitis, 2001) to gauge how a subject perceived an exercise challenge to be physically strenuous with success.

Asthma and Effect on Lung Function

Although many authors characterize asthma as a disease (McArdle, Katch, Katch, 1996, Wilmore & Costill, 1994, Powers & Howley, 2001), Lacroix (1999) explains that, "because people who have asthma differ with respect to provocation factors, clinical manifestations, disease progression, and treatment responsiveness, asthma is often viewed as a syndrome, rather than a unique disease entity." (p. 76). Hay fever and allergic rhinitis also have been closely associated with the disease (McArdle, Katch, Katch, 1996, O'Kane and Woodford, 1999). O'Connor defines asthma as, "an abnormal autonomic regulation of bronchial muscles *(that)* produces bronchospasm, partial airway obstruction, chronic bronchial inflammation and bronchial edema." (2001, p. 68). LaCroix (1999) attributes this hyperresponsiveness to an abundance of stimuli including pollen, cigarette smoke, air pollutants, cold air, viral infection, and physical exertion.

O'Kane and Woodford (1999) describe asthma as a reversible pathology with the elimination of such triggers or through immediate or long-term treatment.

This chronic inflammatory airway disease presents itself through the involuntary constriction of respiratory smooth muscle initiated by some allergic or hyperactive response to a trigger. Many authors cite commonly reported symptoms of asthma as chest constriction, pain, respiratory fatigue and dyspnea where signs observed include wheezing, shortness of breath, productive or non-productive cough, and inability to take a complete breath (O'Kane & Woodford, 1999, LaCroix, 1999, O'Connor, 2001).
It has been traditionally diagnosed through a comprehensive medical history indicating the aforementioned symptoms or more objectively through the use of spirometry (Houglum, 2001, ASCM, 2001). According to accepted protocols (American Thoracic Society, 1994), an individual's best forced expiratory volume in the first second (FEV-1) and peak expiratory flow (PEF) readings are measured when symptom free and then used as base-lines for follow up assessment. If a drop in the FEV1 of 10% -15% occurs, they are diagnosed with asthma (O'Kane, Woodford, 1999, Houglum, 2001, Backer, 2002). Treatment for asthma range from emergency-rescue bronchodilators such as albuterol to long-term control therapy that typically involves inhaled and oral medications like corticosteroids and leukotriene inhibitors (Houglum, 2001).

The incidence and prevalence of asthma varies in regards to populations surveyed. The National Center for Health (2003) statistics reports that in 2001, 13.3 million people have reported being diagnosed with asthma within their lifetime. Kovan and Mackowiak

speculate that nearly seven (7) percent of the American population suffers from the disease (2001). Sixty (60%) percent of those indicated having experienced an acute asthmatic attack within the previous year. In 2000, 10.4 million outpatients' visits to hospitals, clinics and private doctors offices were recorded for complications arising from asthmatic attacks and a total of 4,487 deaths were attributed to asthma.

Exercise-induced bronchoconstriction (EIB) and exercise-induced bronchospasm is a related pathology, but as Rundell, Wilber & Lemanske (2002) contend, asthma tends to be multifactorial, whereas the latter two terms tend to be resultant reactions to the condition. They cite Weilers' definition of EIA being, "an intermittent narrowing of the airways, accompanied by a decrease in some measure of airflow that the individual experiences as wheezing, chest tightness, coughing, and/or dyspnea that is triggered by exercise" (2002, p. 40). Several researchers have extended this definition to encompass exposure to cold, dry air while exercising as one of the primary factors in EIA symptom manifestation (Kovan & Mackowiak, 2001, Wilber, 2002, Tikkanen & Helenius, 1994, Straus, et al., 1977, Sinha & David, 2003, Carlsen & Carlsen, 2002, Anderson, 2002, Gotshall, 2002, Folwer, 2001, Rupp, 1996, LaCroix, 1999, ACSM, 2000). As shown in Weiler's definition, symptoms of EIA are remarkably similar to those of asthma, but generally present five (5) to fifteen (15) minutes following the completion of exercise and typically subside after sixty (60) to ninety (90) minutes (Wilber, 2002). Due to a similar presentation of symptoms and disease manifestation, Wilber (2002) has delineated asthmatics into three categories; individuals who have both chronic asthma and EIA

(90% of surveyed population), individuals who have chronic asthma, but not EIA (10%) and those who have EIA but not chronic asthma or allergic rhinitis (3-10%) (p.41).

The term "refractory period" has been used to describe the period of time following an acute attack of EIA symptoms (Rupp, 1996, LaCroix, 1999, ACSM, 2000, Anderson & Holier, 2002). In the several hours that ensue, an individual might experience a weaker (by approximately 50% or more) attack or enter into a term of remission from another attack due to a decreased sensitivity of the already inflamed bronchial cells. Although many researchers claim this buffering phenomenon to be real, Rundell, et. al. (2003) studied cross-country skiers during a prolonged exercise challenge and found that only one (1) of eighteen (18) athletes experienced a refractory period as determined by spirometric measurements. Hence, it is difficult to objectively determine such a phenomenon but clinicians and researchers continue to suggest eliciting the refractory period through a warm-up period prior to exercise as a preventative technique (Houglum, 1999, Rupp, 1996, LaCroix, 1999, ACSM, 2000, Anderson & Holzer, 2002).

Many theories have been proposed as to the origin of EIA (Anderson & Holzer, 2002), but two have become the more thoroughly researched and accepted. Both host as its primary tenets the energy-transfer effect of ventilation as air is passed over the epithelial layers of the bronchial tract. It has been postulated that as ambient air is warmed and humidified, both cellular heat and water are lost within the protective layers of the respiratory passageway. This pattern of thinking has led to the hyperosmolarity and thermal hyperemia theories.

Inspired environmental air passes by the airway mucosa in the nasal passages where it is warmed to 37° C and humidified with approximately 44 mg H20/L through convective air flow. This process of conditioning the air removes water from the mucosal layers into the airway lumen, thereby increasing the osmolarity of the cells. This dehydration creates hyperosmolar cells that promote mast cell degranulation, which is a precursor of inflammatory mediator release. It is thought that the release of these pro inflammatory agents leads to bronchoconstriction and general symptoms of EIA (Kanazawa, et al., 2002, Storms & Joyner, 1997, Anderson & Holzer, 2000). However, in a study using eight (8) atopic asthmatic subjects, McFadden, et al. (1999) found that, "the removal of water from the lower respiratory tract, and by inference, the development of a hyperosmolar periciliary fluid, do not appear to be the primary causes of thermally induced asthma." (p. 223). They further contend that although water loss contributing to an increased osmolarity of the mucosal cells may not be the major influential factor in the initiation of EIA, the rapid evaporation of water does lead to loss of heat, provoking another form of the theory.

Inspiration of cold dry air significantly induces evaporation of water from the epithelial cells within the mucosa to condition the air for transfer into the lower respiratory tract. This theory has been strongly supported through study of winter sport athletes such as skiing and running where air is typically dry and frigid (Tikkanen & Helenius 1994, Strauss,et al., 1977, McDonald, et al., 1997, Rundell & Jenkinson, 2002). The evaporation and energy transfer has a transitory cooling effect on the mucosa, initiating a reflexive increase in the capillary blood flow of the airway vasculature. This

engorgement of blood flow in the capillary network is thought to be the body's attempt to re-warm the bronchial tissue back to homeostatic temperature. It has been hypothesized that this reflexive warming actually promotes pericapillary leakage and the formation of airway edema thus impinging on the lumen of the airway (Anderson & Holzer, 2000, Rupp, 1996, McKardle, Katch & Katch, 1996).

Other factors that have been studied regarding the severity of EIA disease have included the levels of nitric oxide in the respiratory epithelium (Kanazawa, et al., 2002), and the presence of vascular endothelial growth factor in asthmatic airways (Kanazawa, et al., 2002). Mickleborough and Gotshall (2003) have reported that "dietary excess of salt, omega-6 fatty acids, and a dietary deficiency of anti-oxidant vitamins and omega-3 fatty acid, can modify the severity of EIA" (p. 671). In fact, while studying the effects of dietary salts on the magnitude of the symptoms associated with EIA, separate investigations found that diets low in dietary salt intake showed marked improvements on pulmonary function tests as compared to baseline assessment. Consequently, high salt diets actually worsened measures (Gotshall, et. al. 2000, Mickleborough, 2001).

The prevalence of exercise-induced asthma is somewhat confounding due to the multitude of factors that can affect its etiology and presentation. EIA has been reported to affect approximately 90% of those diagnosed with chronic asthma and 40% of those diagnosed with allergic rhinitis or atopic dermatitis (Wilber, 2002, LaCroix, 1999). The American College of Sports Medicine (2000) state the prevalence of EIA to be approximately ten (10%) to fifty (50%) percent depending on the sport studied, the

exercise protocol, diagnostic tool and environmental conditions. This large range has since been attenuated through other research studies quoting the general publics likelihood of being diagnosed with EIA to be around twelve (12%) to fifteen (15%) percent while active individual rates range from three (3%) to more than twelve (12%) percent (Rupp, 1996). Wilber (2002) points out that 11.2% of the United States athletes competing in the 1984 Winter Olympics were diagnosed with EIA while a staggering 50% of cross country skiers in the 1998 Winter Olympics reported the disease. Interestingly, over forty medals were awarded to 1994 Olympic team members who happened to also be EIA sufferers supporting the notion that individuals diagnosed with this condition can continue competitive function.

Differentiating EIA from chronic asthma can be best described by Storms and Joyner's comments (1997): "The distinction between solitary EIA and persistent asthma is best made by pulmonary function testing at rest. In a patient with solitary EIA, the results will be normal; in the results of a patient with chronic persistent asthma, pulmonary function will be lower than normal." (p. 3). Diagnosis of exercise-induced asthma is usually accomplished by conducting a thorough medical history to rule out other pathologies such as cystic fibrosis, chronic bronchitis, pneumonia or paradoxical vocal cord dysfunction (LaCroix, 1999, Koester & Amundson, 2002, Brugman & Simons, 1998). However, differentiating between chronic frank asthma and exercise-induced asthma will present any physician with a quandary as the same symptoms are seen in both pathologies. Coughing, wheezing, chest tightness, dyspnea, shortness of breath, excess mucous and respiratory fatigues are common complaints from both asthmatics and

victims of EIA (Rundell, Judelson, & Williams, 2002). Therefore, history questions should assess respiratory symptoms associated with asthma and primarily target the period of time directly following cessation of exercise (Anderson, 2002). A physical examination will essentially reveal nothing remarkable in conditions of EIA. Some have documented the presence of polyps in the nasal passage, inflamed mucosal layers, or drainage into the pharynx possibly due to concomitant conditions such as chronic asthma (LaCroix, 1999, Rundell, Judelson, & Williams, 2002). Most physicians will opt for pulmonary function tests that include spirometry and, more recently, impulse oscillometry (Rupp, 1996, Marotta, et al., 2003).

Treatment for Exercise Induced Asthma

Historically, exercise-induced asthma has been treated through pharmacological and non-pharmacological means. In reference to the acute bronchoconstriction commonly associated with the period following an exercise bout, fast-acting rescue bronchodilators have actually been used to ameliorate the dyspnic symptoms. In diagnostic medicine, these agents have been used to differentiate between EIA and chronic asthma. It has been found that dyspnic symptoms following exercise typically cease when treated with a B- agonist inhaler whereas frank asthma tends to require more comprehensive pharmaceutical treatment (LaCroix, 1999, Anderson, 2002). These inhaled medications, or beta-2 agonists, act on the beta adrenergic receptors of the smooth muscle cells within the bronchial tract to induce bronchodilation. Medicinal effects usually occur within five (5) minutes of being administered and typically last for four (4) to eight (8) hours. When used to prevent symptoms of EIB, beta-2 agonists host effects for only half of that time,

or two (2) to four (4) hours post administration (Houglum, 2001). Although a limit has not been established on how many standard canisters one should use to treat episodes of bronchoconstriction per day, Anis, et al. (2001) reported that when investigating current trends in asthma therapy, four (4) canisters per day would not be excessive. However, this repeated usage poses another problem, that of desensitization to the drug. Hancox, et al. (2002) found that when studying eight (8) subjects diagnosed with EIB, "regular B-agonist treatment leads to increased exercise induced bronchoconstriction and a suboptimal bronchodilator response to B-agonist." (p. 1068). This is further supported by Anderson (2002) who states that, "B2- agonists are not the most suitable therapy for preventing EIA if they need to be used on a daily basis."(p. S61).

Long-term therapeutic agents ranging from inhaled corticosteroids (National Asthma Education and Prevention Program, 2002), inhaled heparin (Tahir, et al., 1993), oral montelukast (Peroni, 2002), salmeterol (Nelson, et al., 1998), sodium cromglycate and Nedocromil sodium (Backer, 2002) have had significant effects when treating EIA. These pharmaceuticals are meant to be taken on a strict treatment regimen to prevent an acute attack and should not be viewed as rescue agents (Houglum, 2001, O'Kane & Woodford, 1999).

Although contemporary medicine has provided for such advances in pharmacological intervention for the treatment of EIA, studies have shown that patient education and prescription drug adherence is less than desired. Interestingly, patient compliance with long-term therapies has not been good with some authors quoting less than fifty (50%)

percent compliance (NAEPP, 2002). Anis, et al. (2001) attributes this poor compliance rate to improper medication use and poor patient education, a common fault in many asthma control programs (NAEPP, 2002).

In addition to eliciting the refractory period with a well timed warm up period (ACSM, 2001), many authors have suggested physical conditioning to prevent or attenuate future EIA episodes (Carlsen & Carlson, 2002, Fowler, 2001, Barfield & Michael, 2002, LaCroix, 1999, Satta, 2000, Diabella & Sherman, 1998). It is hypothesized that increased aerobic fitness will increase an individual's ability to work at a "lower vital capacity, decreasing the cooling and drying stimuli, resulting in less bronchospasm" (Sinha, 2003, p. 770). Rupp (1996) lends further support for this mode of treatment stating that, "Management should begin with efforts to increase physical conditioning, which can reduce the requirements for pharmacotherapy and decrease the incidence of asthma attacks." (p. 5).

Respiratory muscle training

It is a standing physiological feat that skeletal muscle hypertrophies and becomes more neurologically trained when engaged in periodic bouts of increased resistance exercise. This natural causal model presents the fundamental underpinnings of strength training as it stands today (McArdle, Katch and Katch, 1996). In many instances, skeletal muscle strengthening is used to increase muscle mass and function for improved performance. Some individuals might employ resistive training to compensate for a debilitating injury or pathological deficiency where an individual might have to re-train the neuromuscular

pathways and re-establish pre-injury muscle tone. Either way, the overload and the Progressive Resistance Exercise (PRE) principles govern the notion of muscle training and its subsequent hypertrophy. Both of these accepted theories incorporate placing above normal stresses on muscle tissue which will in turn produce gains in muscle fiber strength and diameter (McArdle, Katch and Katch, 1996).

A majority of the studies conducted thus far on respiratory muscle training have concentrated on inspiratory muscle training on those groups afflicted with some pulmonary pathology that somehow restricts airflow. The common factors in these studies tend to focus on muscular strength and endurance gain through resistive inspiratory breathing. Lotters et al. (2002) support these points when they performed a meta-analysis of several studies conducted on inspiratory muscle training for patients diagnosed with COPD. They summarize their findings by reporting that, "both inspiratory muscle training alone and inspiratory muscle training as adjunct to general exercise reconditioning significantly increase inspiratory muscle strength and endurance." (p. 570).

Interestingly, a number of investigations performed with a pathological population utilized a methodology consisting of a moderately small (typically less than 30) subject population and a training period that ranged from weeks to months.
In two separate studies Lisboa, et al. (1994), investigated the effects of inspiratory muscle training (IMT) in patients diagnosed with chronic airflow limitation (CAL), characterized as a deficit of $\geq$30% in forced expiratory volume in one second. In the first, Lisboa, et al.

(1994), found that while studying twenty (20) subjects, the experimental group (n=10) that was trained using an inspiratory load equal to thirty (30%) percent restriction of flow showed a greater increase in markers of inspiratory muscle strength and exercise endurance compared to a control group that used a respiratory device that restricted twelve (12%) percent inspiratory flow. Subjective measures of dyspnea also decreased over the five (5) week training period in the experimental group. Both groups trained on their respective devices for thirty minutes per day for a total five (5) weeks. In the second study, inspiratory training devices hosting a thirty (30%) percent resistance in peak inspiratory flow resulted in improved measures of dyspnea, increased exercise capacity and reduced the metabolic cost of exercise. Subjects were trained for thirty (30) minutes per day, six (6) days per week for ten (10) weeks (Lisboa, et al., 1997).

In a series of studies investigating the effects of inspiratory muscle training on patients diagnosed with chronic obstructive pulmonary disorder, subject populations ranged from four (4) (Nield, 1999), eleven (11) (Goldstein, et al., 1989), thirteen (13) (Chen, Dukes, Martin, 1985), fourteen (14) (Ramirez, et al., 2002), and thirty (30) (Scherer, et al., 2000). Treatment for inspiratory training consisted of one (1) (Nield, 1999, Ramirez, et al., 2002) to two (2) (Goldstein, et al., 1989, Scherer, et al., 2000, Chen, Dukes, Martin, 1985) separate sessions per day for variable number of days (five to seven) within four (4) (Goldstein, et al., 1989), four (4) (Chen, Dukes, Martin, 1985), five(5) (Ramirez, et al., 2002), six (6) (Nield, 1999), and eight (8) (Scherer, et al., 2000) week periods. Goldstein et. al. (1989), Chen, Dukes & Martin (1985), and Scherer et al., all found that IMT increased inspiratory muscle endurance and decreased sensation of dyspnea after

treatment with a device capable of thirty (30%) percent or more restriction. Additionally, Nield (1999) found that IMT increased muscular strength, as measured through inspiratory force generated, as well as decreased dyspnic scores of COPD.

A noteworthy study conducted by Weiner, et al. (1998) involved patients undergoing coronary artery bypass surgery. Historically, this surgery left patients with depressed respiratory function that extended the time of hospitalization post surgery. In the study, forty-two (42) patients underwent prophylactic IMT for two (2) weeks one month prior to surgery. When compared to a control group (n=42) of similar patients, the experimental group maintained significant respiratory function post operation as indicated by pulmonary function tests, respiratory muscle function and gas exchange measures.

Although no studies were found specifically focusing on exercise induced asthma, several investigations composed of frank asthma patients were reviewed. In two separate studies, Weiner, et al. (1992) found that specific inspiratory muscle training conducted for six (6) months on mild asthmatics resulted in decreased dyspnic scores and decreased beta (2) agonist consumption. Weiner further supported these findings in two other independent studies that spanned over 3 (Weiner et al., 2000) and 4 months (Weiner et al., 2002) using IMT.

Resistive respiratory muscle training has not been reserved to treatment of the clinical population, but also has been used with marked success in healthy subjects as well. Pardy, Reid, & Belman (1988) indicate in a review of studies conducted on healthy

individuals, respiratory muscle training does offer marked improvement in muscle strength and endurance. They did conclude that although improvements were observed, methodological shortcomings such as subject population number and training method were present and should be corrected for in future studies. Improvements in respiratory function credited to inspiratory muscle training (IMT) typically include change in lung capacity, pressure generation, and rate of ventilation. However, Sheel (2002) agrees with Pardy, Reid & Belman, stating that studies conducted thus far, on these highly specific measures alone, provide a tenuous argument in correlating respiratory muscle training to improve exercise performance. Methodological shortcomings including small population sizes and the use of an inappropriate control group were the main areas of contention for study design inadequacy. Coincidentally, the author explained that possible reasons for the improved measures observed in exercise performance within these reviewed studies include a decrease in improvement in breathing perception, delayed fatigue of respiratory musculature and improved ventilatory efficiency. Chatham, et al. (1999) reason that these logistical faults in study execution has pre-empted the inclusion of respiratory muscle training in everyday clinical practice.

In 1976, Leith and Bradley were quoted as saying, "Many observations suggest that normal human ventilatory muscle capacities can be increased by approximate stimuli. But we found no adequate studies of ventilatory muscle training which employed direct measures of strength and endurance..." "...nor studies which applied accepted principles of strength and endurance training specifically to the ventilatory muscles, either in normal humans or in persons with respiratory disease." (p. 508). The study they

subsequently conducted involved three treatment groups (strength training, endurance training and a control group) that underwent a training period of five (5) weeks. It was found that the strength training group increased their ability to generate maximal inspiratory pressure by fifty-five (55%) percent. Likewise, endurance training improved sustained ventilation as measured by maximal voluntary ventilation (MVV) by more than fifteen (15%) percent. It was concluded from this pioneer study that individuals can increase specific ventilatory measures that correspond with respiratory muscle strength and endurance.

O'Kroy & Coast (1993) and Amonette & Dupler (2001) concur with these observed results in similar studies. Following a four (4) week training period, O'Kroy and Coast found that endurance training improved both flow and resistive markers as measured through spirometry while strength training affected strength and resistive test measures. Likewise, Amonette and Dupler used a respiratory training device that restricted airflow by more than thirty (30%) percent normal ventilation rates over a four-week training period. Specific percentages were subjectively selected according to perceived flow exertion. During this treatment, experimental group members performed thirty (30) inhalation and exhalation maneuvers on the device two (2) times per day. Investigators found that when compared to the control group, resistive respiratory training improved maximal and sub maximal ventilation, peak exhalation flow test and maximal tidal volume. Although no significant changes were seen in pulmonary mechanics such as FVC, FEV-1, peak expiratory flow, the authors suggest that because pulmonary muscles

adapt to aerobic exercise, as in the case with the specific study population, smaller (less significant) changes and differences would be seen after respiratory training.

Several studies have since focused their respective methodologies and investigated the benefits of specific inspiratory training on pulmonary function. While studying twenty-two (22) subjects using an incremental inspiratory muscle training program, Chatham, et al. (1999) discovered that the experimental group improved respiratory muscle strength and endurance, metabolic cost of ventilation and predicted maximal oxygen consumption (VO2 maximum) over a ten (10) week treatment period. Additionally, Suzuki, et al. (1993) and Caine & McConnell (1998) also found that experimental groups training with an inspiratory training device that restricted a percentage (range of 30%-40%) of inspiratory flow, improved maximal inspiratory pressure. These results indicated a significant training effect on the diaphragm and its ability to decrease intrathoracic pressure. Inbar et al. (2000) also found that while investigating twenty well conditioned athletes, the experimental group (n=10) increased inspiratory muscle strength and endurance after a ten (10) week treatment program.

Interestingly, a study conducted to determine the effectiveness of inspiratory muscle training on rowing performance revealed that along with a significant increase in inspiratory pressure, experimental protocol also improved the conductance of a sport specific exercise challenge (six minute rowing test) (Volianitis et al., 2001). These findings challenge the earlier statement by Sheel (2002) who questioned the transferability of respiratory training findings to performance. Again, the protocol used in this study resembled the methods used in a majority of these aforementioned studies.

Experimental subjects were trained for five (5) minutes (approximately 30 breathes) per day for eleven (11) weeks. At the conclusion of the investigation, subjects improved inspiratory measures and decreased perceived exertion due to training (Volianitis et al., 2001).

The comparison has been made that, "Respiratory muscles are skeletal muscle that are functionally similar to locomotor muscles. Their primary task is to act on the chest wall to move gas in and out of the lungs..." (Powers & Howley, 2001, p. 189). This fact has led many researchers to train the respiratory muscles with some form of a resistive load. In doing so, they hope to elicit neurological and morphological adaptations to exercise. In reference to the overload principle, McArdle, Katch and Katch state that, "Exercising at a level of intensity higher than is normally performed can induce a variety of highly specific training adaptations that enable the body to function more efficiently." (1996). A concept attributed to DeLorme that expanded on the overload principle was that of the progressive resistance exercise principle, which proposed that movements against increased resistance over consecutive repetitions would show significant muscular strength improvements. Chatham extrapolates on this idea of incremental loading by suggesting that respiratory muscle training should introduce an appropriate resistance to the ventilatory musculature and occur three (3) times per week for an excess of four (4) weeks. Chatham (1999) further cites Smith et al. (1992), who suggest "that if breathing pattern is controlled and substantial pressure generated during respiratory muscle training, appreciable increase in respiratory muscle strength and endurance may occur." (p. 2)

Several respiratory training devices are currently on the market (PowerBreathe®, TrainAir®, PowerLung®, UltraBreathe®, and Lung Exerciser®, PFlex®, Threshold® devices by TreatCOPD.com). The device chosen for this study is the Sports Breather® (donated by Dream Distribution, Inc.), a restrictive airway device that can control the amount of ambient air entering and exiting the device via the mouth. Graded levels of airway restriction can be manually changed on the device through the ventilation hub. Inspiratory grades 1-5 correspond with (-4, -5, -6, -13, -31 cm H2O while tested at 15 liters/minute flow rate) pressures while expiratory resistances 1-4 coincide with (+6 cmH20 at 28 liters/ minute, +14 cmH20 at 24 l/m, +24 cm H20 at 20 l/m and +43 cmH20 at 31 l/m) pressures (Fountain, C., 1997).

Summary of Literature Review

The elaborate organization of the pulmonary system is made of airways and lung tissue that serve as a vital transit area for the exchange between gases of the circulatory system and ambient air. The body's internal mechanism for regulating ventilation allows for a movement of air inside the body to accommodate specific oxygen needs. However, this intricate system can become compromised through a variety of structural and functional pathologies that can affect the timing, efficiency and quality of inspiration and expiration.

One such condition is that of asthma, a chronic obstructive disorder that affects many within the general population as evidenced by health census organizations and pharmaceutical distributors. This disease varies in severity, but is often characterized by episodes of coughing, wheezing, and a subjective feeling of a tight chest. Traditional

treatments have involved short-term, fast-acting bronchodilators as well as longer acting agents taken on as strict regiment. Although in most cases asthma is not a debilitating condition, it can disrupt the quality of life many experience while conducting activities of daily living or when engaging in physical activity.

Exercise-induced asthma is a variation of this more chronic obstructive disease that is classically defined by the bronchoconstriction brought upon by the water and heat lost due to excessive ventilation. Although common treatment modalities include fast-acting beta-2 agonists, it has been suggested that a way to attenuate the effects of this airway hyperresponsiveness could include the specific conditioning of the respiratory system, specifically the muscles of respiration.

Resistive training of the respiratory muscles using resistive respiratory training devices has been experimentally used to treat the effectiveness of respiration in obstructive disorders such as asthma, COPD and cystic fibrosis with variable success. However, a significant portion of literature exists indicating the usefulness of this resistive muscle training to increase the healthy body's ability to draw ambient air into the lungs and just as effectively push it out. Unfortunately, no research exists regarding the use of respiratory training on exercise-induced asthma. This study will build upon the theoretical concepts and statistical findings of related lines of research to build a stable foundation for inquiry.

CHAPTER 3

METHODOLOGY

This chapter will introduce the study design and specific methods and procedures used to research the effectiveness of resistive respiratory muscle training on exercise-induced asthma and pulmonary mechanics.

Study Design

A single-blind study using randomized control and experimental groups was conducted. Dependent variables or measures that did or did not change in response to the training were assessed at the beginning and end of the 5-week training period. The training period consisted of the five-(5) week period during which volunteer subjects used the resistive respiratory training device according to the study instructions. Pre-exercise stress testing was conducted prior to the five (5) week training period and represented the baseline values for each subject. Post-exercise stress testing values recorded both before and after training represented the response to acute exercise stress. Results of post-exercise stress testing were expressed as a percentage of pre-exercise stress testing values to remain consistent with related scientific research (appendix 2-A). Dependent variables derived from spirometry were forced vital capacity (FVC), forced expiratory volume in one second (FEV-1), forced expiratory volume between 25-75% of exhalation (FEV 25-75%) and peak expiratory flow (Pex). Dependent variables derived from oscillometry

were magnitude of respiratory impedance at five (5) hertz (Z5), total respiratory resistance at five (5) hertz (R5), proximal respiratory resistance at twenty (20) hertz (R20), distal capacitive reactance at five (5) hertz (X5). Modified Borg RPE scores were recorded during all exercise stress tests.

Subjects

Twenty-two (22) subjects originally volunteered to participate in the study's methods. During the course of baseline, pre-training testing, two (2) subjects became ill and opted out of the study, while eight (8) other potential volunteers did not choose to participate due to personal reasons unbeknownst to the investigation team. Follow-up telephone calls were not returned. Twelve (12) volunteer subjects (3 male/ 9 female) ranging in age (male 21.3±0.6 years; female 21.2±0.9 years), height (male 72.67±0.6 inches; female 63.33±3.4 inches), weight (male 181.33±12.7 pounds; female 142±17.7 pounds), from Oklahoma State University- Stillwater served as the subject population.

Group	N
Control	4
Experimental	8

Subjects were solicited through announcements in the Department of Athletics. Interested parties were directed to contact Matthew O'Brien, primary investigator for further information regarding the study. Upon contact, the primary investigator advised each individual about the purpose and requirements of the study and presented them with an informed consent form (appendix 1-A. All participating subjects were required to complete an "Asthma and Allergy" questionnaire to determine if they do in fact suffer from symptoms related to exercise-induced asthma. Subject approval required a cleared physical on file, a signed informed consent form and a questionnaire indicating an

unremarkable previous medial history and absence of any precluding factors to physical activity.

Testing Facility

The testing facility was located in the Seretean Wellness center at the Oklahoma State University- Stillwater campus. The room used was cleared of visible allergens (dust, plant-life, etc.) and was maintained at a temperature of (22-24° C degrees). The facility housed a Jaeger-Toennies Impulse Oscillometry Measuring Station, capable of collecting spirometric measurements and impulse oscillometric determinations of airway resistance and reactance. A Quinton Q50 treadmill capable of 0.6-6.0 mph (0.96-9.6 km/h) belt speed, a digital stop watch, and a telephone were placed in the laboratory room. Polar® heart monitors were used to monitor subject heart rate during exercise. Individual subjects were instructed to bring any prescribed (by their own physician) fast-acting bronchodilating agents with them to all appointments. The primary investigator had access to a CPR mask and bag valve mask during the course of testing.

Heart Rate Monitors

Polar® heart monitors were fastened around the subject's chest area and the heart rate watch was held by the primary investigator so that the subjects' heart rate was accurately monitored. This device assured investigators when the subject has attained 85% maximal heart rate.

Jaeger-Toennies Impulse Oscillometry Measuring Station®

This device utilizes a non-invasive procedure to determine pulmonary obstruction and location of repiratory impedance. Specifically, this study will target the amount of respiratory obstruction of the lower-level bronchioles and terminal branches leading into the alveolar sacs. This station is composed of a mainframe computer linked with a pnuemotach, "Y" frame lead, and a face attachment for measurements. Signals are generated at the pnuemotach and "superimposed on the respiratory tidal breathing waveform of the patient in the Y-adapter while the patient simply inhales ambient air via mouthpiece, pneumotach and terminating resistance (< 0.1 kPa/l/s)" (Jaeger-Toennies®). Signals are then monitored at the MasterScreen IOS® station differentiating respiratory impedance at specific pulmonary locales.

Preliminary Procedures

Each subject was informed by the informed consent form and the primary investigator as to what their participation entailed. They were informed of any potential benefits and risks due to participation. Participation involved two separate testing appointments and an intervening five-(5) week training period. At the conclusion of the study, all volunteers were given the option of keeping the respiratory training device. Potential risks of the study did not exceed the same level of risk incurred through participation in physical activity, namely running. After signing a letter of informed consent (appendix1-aa) indicating their understanding of the study purpose and requirements, subjects completed a subjective history assessment targeting respiratory dysfunction after exercise (appendix 1- bb). Five (5) days were scheduled at both the beginning and end of the five-

(5) week training period to collect preliminary and post-training data. Subjects were assigned one hour appointment times on any one of the five potential testing days. When scheduling the post-training appointment, subjects typically returned on the same day of the week so that all experienced the full five (5) weeks of training with the resistive respiratory training device. Subjects were instructed on the informed consent form to not take any short-acting prescription drugs treating their symptoms of exercise induced asthma or complete any vigorous exercise on the day of testing. No other long acting drug therapy was interrupted. They were required to bring any fast-acting aerosol bronchodilators that had been prescribed to them for EIA symptoms for safety. Subjects were instructed to wear standard exercise clothing (shorts and t-shirts) including a pair of athletic shoes equipped for running. The primary investigator, trained in cardio-pulmonary resuscitation for the professional rescuer, was present at all times during diagnostic assessment.

Grouping

Subject names and identification numbers were entered into a statistical package software program (Statistical Package for the Social Sciences, Chicago, IL.) where two lists were randomly generated using the random generator program. These lists were then coded as experimental (1) and control (0) group subjects. All names and records of subject participation were kept completely confidential and secured in the primary investigators locked office. At the conclusion of the study, this information was destroyed or deleted.

Emergency Action Procedures

The primary investigator trained in advanced CPR and life saving techniques was present at all times with a CPR and a bag valve mask able to intervene at any point during the testing protocol. The medical supervisor, a licensed physician, was on campus within one half mile (0.5 mile) of the testing facility located at the Oklahoma State University Health Services Building and was able to intervene upon contact. Emergency Medical Services was also noted as the secondary contact in case of emergency.

If at any point during this exercise testing, the athlete experienced strong presentation of symptoms such as angina, light-headedness, sudden pallor, or cyanosis (Hansen, 1982), the testing run would have been ceased and a bronchodilator prescribed to that individual would have been self-administered. If at any point of the exercise testing, the athlete lost consciousness, immediate first aid and CPR procedures would have been initiated including contacting emergency medical services and the study's medical supervisor.

Procedures

Individual subjects reported at their assigned testing times to the Seretean Wellness Center where height and weight was measured. Resting heart rate through palpation of the radial pulse was also measured while the individual was seated in a standard chair.

Spirometric and forced oscillation measurements using the Impulse Oscillometry Measuring Station® (IOS, Jaeger-Toennies) was taken while the subject was seated in a standard chair allowing ninety degrees (90°) of knee and hip flexion. The subject was

seated so that their cervical spine was slightly extended to prevent premature glottic closure. The primary investigator adjusted the extension arm of the IOS measuring station so that the oral measuring device was proximal to the subject's head. The subject was then instructed to breathe normally into the mouth piece for approximately thirty (30) seconds until three valid measurements were recorded. An average for each measure was calculated. The preliminary measures included forced expiratory volume in one second (FEV-1), forced expiratory volume between 25-75% of exhalation (FEV 25-75%), forced vital capacity (FVC), peak expiratory flow (Pex), magnitude of respiratory impedance (Z5), total respiratory resistance (R5), proximal respiratory resistance (R20) and distal capacitive reactance (X5) using the IOS station. These values served as a baseline, pre-exercise stress testing measure catalog.

A calculation of work rate of the individual subjects at eighty-five percent (85%) of the maximal heart rate (American Thoracic Society, 1994) was then calculated. The subjects age was subtracted from two hundred and twenty (220) and multiplied by 0.85 (85%) to obtain predicted maximal heart rate. The treadmill belt speed was advanced so that the subject would reach this maximal heart rate while causing no cardiovascular complications.

Before the initial exercise-stress test began, the investigator explained the use of the modified Borg scale of perceived exertion or breathlessness (appendix 1-C). The subject assumed the treadmill placing both hands on the supportive railings with both feet to each side of the treadmill belt. The investigator began the treadmill at a moderate speed (2

miles/ hour) and instructed the subject to engage walking on the treadmill belt. The investigator then gradually increased the treadmill speed by increments of 1 (one) mile per hour. Once the speed of the treadmill belt had increased the heart rate to the predicted percentage of maximal values, the subject continued at this pace for 7 (seven) consecutive minutes (Hallstrand, 2002, ASCM, 2000, ATS, 1994). The investigator asked the subject to rate their perceived level of exertion or breathlessness at minute 2, 3, 4, 5, 6, and 7 of the total exercise stress test. At the end of the seven-minute period (85% of maximal heart rate) exercise, the investigator gradually decreased the treadmill speed to a rate of one (1) mph and the challenge was concluded. The subject then dismounted the treadmill and all respiratory post-exercise stress testing measures (excluding height and weight) were assessed in same manner as the pre- exercise stress testing measures.

Spirometry including FVC, FEV-1, FEV $_{25\text{-}75\%}$ and Pex were measured immediately after exercise challenge and at 5-, 15- and 30-minute intervals for the proceeding 30 minutes (Kukafka DS, et al., 1998, Hansen, J.E., 1982). A total of four (4) spirometric measures were recorded. The maximum deviation of the four measures was selected indicating the most dramatic response to the exercise stimulus. This investigation focused on response to a stimulus and intervention and therefore only the maximal responses to exercise-stress testing were included in the data pool. If at any time, subjects FEV-1 dropped below 25% of their predicted, measurements would be ceased and bronchodilators would be self administered. No data was deleted from the study for this reason.

Impulse oscillometry measures including magnitude of respiratory impedance (Z5), total respiratory resistance (R5), proximal respiratory resistance (R20) and distal capacitive reactance (X5) were recorded within the intervening time between spirometric scores. Measures were recorded at the 4 (four)- and 20 (twenty)-minute markers to prevent confounding measures due to increased lung volume caused by forced maneuver spirometric measurements.

Training method

All subjects were naïve to respiratory muscle training. At the conclusion of their exercise stress test on day one, each individual was given a SportsBreather® respiratory training device and verbally instructed on the proper use of the instrument. Experimental group subjects received a complete, manufactured Sports Breather® device whereas the control group received a Sports Breather® which had the internal bladder removed (a placebo device). These modified devices represented a form of placebo training. Written instructions (appendix 1-D) were also given to each subject so that proper use and standard protocol were assured throughout the training period.

Initial instructions (appendix 1-D) for both groups included placing the lips around the mouthpiece without clenching their teeth. While both resistance settings are set to one (1) or minimal resistance, the subject began by inhaling deeply for three to four (3-4) seconds, pausing for one to two (1-2) seconds and then slowly exhaling for approximately five (5) seconds. The subject would then engage in resistive respiratory training at level 1 (one) (inspiratory and expiratory) for the first week. At the beginning

of the second week, the athlete would then increase both resistance levels to two and engage in training protocols for the full week. This mode of increasing resistance settings continued for all five (5) weeks. Due to the independent resistance settings, the inhale resistance setting on the hub was set to 4 (four) (maximum allowed) for weeks four (4) and five (5). If at any time during the training with the resistive respiratory device, the subject felt faint, tightness in the chest or excessively tired, they were instructed to discontinue training for that day and inform the primary investigator immediately.

Subjects performed breathing techniques according to a modified technique #1 suggested by the manufacturer (SportsBreather®) and defined above. The subject engaged in training by forcefully breathing into the device using the above technique for thirty (30) breathes or approximately six (6) breathes per minute for an approximate training time of five (5) minutes. This protocol was conducted two (2) times per day and repeated every day of a calendar week for five (5) weeks.

The primary investigator performed periodic assessment of protocol adherence in the form of telephone or personal communication on the Friday of each week. Any technical or problems with the respiratory muscle training device were resolved at these times unless study reliability was threatened. In cases of device malfunction, a new one was issued to the individual. Only one additional device was given to a subject who had lost their original one.

STATISTICAL PROCEDURES

Subject data was compiled and analyzed using the Statistical Package for the Social Sciences (SPSS®, Chicago, IL). Pre-training randomization was checked using student's t-tests on all dependent variables (appendix 2-B).

The effect of training with the resistive respiratory device was determined using a two-way repeated-measures analysis of variance (ANOVA) using subject identification numbers (social security numbers) as a repeating variable, group (experimental/ control) and training status (pre and post-training) as the independent variables. The following groups of respiratory measurements were statistically analyzed; (hypothesis 1) experimental versus control groups' pre-exercise stress testing FVC, FEV1, FEV1/FVC, PEF and FEV 25/75 prior to and following a five (5) week training period; (hypothesis 2) experimental versus control groups' post-exercise stress testing FVC, FEV1, FEV1/FVC, PEF and FEV 25/75 prior to and following a five (5) week training period; (hypothesis 3) data relating to perception of dyspnea as represented using the modified Borg scale of RPE (breathlessness) was plotted using a 2 X 7 X 2 repeated measures analysis of variance feature to graph the means of each minute of the exercise stress test for both groups (appendix 2-E) and was analyzed using group means for each minute of the seven (7) minute exercise stress test before and after training; average change in RPE for each group was also analyzed using group and average difference between pre-and post-training (2 X 2); (hypothesis 4) experimental versus control groups' post-exercise stress testing magnitude of respiratory impedance (Z5), total respiratory resistance (R5), proximal respiratory resistance (R20) and distal capacitive reactance (X5) prior to and

following a five (5) week training period. In addition, the effect of resistive respiratory training on spirometry was assessed in the experimental group using paired t-tests of their pre- and post-training values.

CHAPTER 4

RESULTS

Issues regarding data collection included review of individual subject spirometric values indicating a recording malfunction producing data that was obviously misrepresentative of actual physiology. After each forced expiratory maneuver was conducted, data was analyzed by the IOS software relative to body size and baseline measures and saved to subjects file. However, on random occasion the Master IOS station software would errantly analyze data according to a standard unknown to this investigation team and store data. Attempts to contact the device manufacturer and correct software malfunction proved unsuccessful therefore, all data were included due to inability to find and correct individual subject data. After review of the maximal values of all the associated factors included for statistical analysis, none of the measurement anomalies were included. Additionally, following the five (5) week training session, all individuals reported having upper respiratory symptoms such as nasal drainage, sinus blockage or other related symptoms possibly increasing oscillometric measures recorded.

Physical data for group (mean ± standard deviation) are shown in table 4-1:

Table 4-1

Sex	N	Age (mean±SD)	Height (inches) (mean±SD)	Weight (lb) (mean±SD)
Male	3	21.3±0.58	72.67±0.57	181.33±12.66
Female	9	21.2 ±0.87	63.33±3.39	142±17.70

Analysis of baseline spirometry and oscillometry in table 4-2 revealed no statistical difference between groups on pulmonary mechanics measures except for FVC (p=.046) indicating that although there was a statistical difference between the experimental group (5.347± 1.29 L) and control group (4.115 ± .63 L), randomization of subjects was essentially accomplished (appendix 2-B).

Table 4-2: Baseline Group Statistics

	Control			Experimental		
	N	Mean	Std. Deviation	N	Mean	Std. Deviation
Z_5HZ	4	4.3375	2.69092	8	3.9125	1.55073
R_5HZ	4	4.1000	2.42866	8	3.7038	1.52748
R_20HZ	4	3.4250	1.55886	8	3.1675	1.02112
X_5HZ	4	-1.3700	1.23925	8	-1.2175	.45663
FVC	4	5.3475	1.29611	8	4.1150	.62753
FEV_1	4	4.2350	1.29536	8	3.3363	.80631
FEV1FVC	4	78.0200	7.82847	8	80.4638	11.09776
PEF	4	8.0175	4.16044	8	6.1113	2.43383
FEF2575	4	3.7700	1.43520	8	3.4763	1.63067

Hypothesis 1

Review of subject data in table 4-3 showed no effect of resistive respiratory training on pre-exercise stress testing spirometry (appendix 2-C). Five two-way ANOVA analyses yielded FVC (p=.890), FEV1 (p= .994), FEV1 percentage of FVC (p= .788), PEF (p= .281) and FEV 25/75 (p= .820).

Table 4-3

	GROUP	Mean ±SD
Pre-training FVC	Control	5.3475±1.29611
	Exp	4.1150±.62753
Post-training FVC	Control	4.7050±2.55771
	Exp	3.5788±1.48289

	GROUP	Mean ±SD
Pre-training FEV_1	Control	4.2350±1.29536
	Exp	3.3363±.80631
Post-training FEV_1	Control	3.7575±1.98753
	Exp	2.8537±1.36352

	GROUP	Mean ±SD
Pre-training FEV1/FVC	Control	78.0200±7.82847
	Exp	80.4638±11.09776
Post-training FEV1/FVC	Control	78.5325±5.71843
	Exp	78.3750±12.41913

	GROUP	Mean ±SD
Pre-training PEF	Control	8.0175±4.16044
	Exp	6.1113±2.43383
Post-training PEF	Control	5.6175±2.78585
	Exp	5.4863±2.75380

	GROUP	Mean ±SD
Pre-training FEF2575	Control	3.7700±1.43520
	Exp	3.4763±1.63067
Post-training FEF2575	Control	3.4675±2.11262
	Exp	2.9150±1.88667

Hypothesis 2

There was no effect of resistive respiratory training on post-exercise stress testing (appendix 2-D). The hypothesis data in table 4-4 did not reach statistical significance for FVC (p=.858), FEV1 (p= .770), percentage of FEV1/ FVC (p= .083), PEF (p= .912) and FEV 25/75 (p= .497).

Table 4-4

	GROUP	Mean ±SD
Pre-training FVC Response	Control	101.12±3.66
	Exp	102.10±6.07
Post-training FVC Response	Control	155.37±104.94
	Exp	145.91±88.33

	GROUP	Mean ±SD
Pre-training FEV_1 Response	Control	105.18±15.70
	Exp	97.48±14.42
Post-training FEV_1 Response	Control	150.59±101.84
	Exp	162.80±113.37

	GROUP	Mean ±SD
Pre-training FEV1/FVC Response	Control	105.10±11.32
	Exp	97.23±13.29
Post-training FEV1/FVC Response	Control	98.50±3.18
	Exp	108.72±11.93

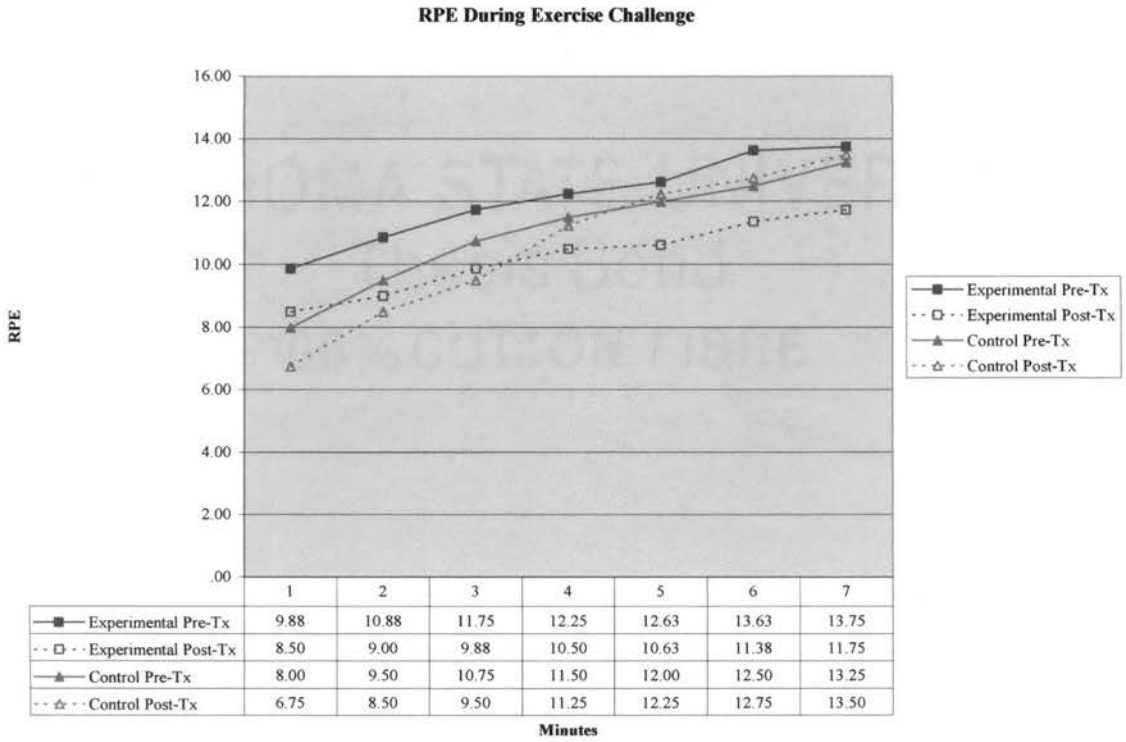
	GROUP	Mean ±SD
Pre-training PEF Response	Control	111.41±49.43
	Exp	104.36±28.79
Post-training PEF Response	Control	167.57±92.90
	Exp	153.57±105.41

	GROUP	Mean ±SD
Pre-training FEF2575	Control	115.47±28.68
	Exp	96.48±29.91
Post-training FEF2575	Control	151.80±120.74
	Exp	216.70±216.26

Hypothesis 3

Graph 4-5 shows the control and experimental groups reported mean RPEs in relation to breathing or dyspnea both before and following the five (5) week training session. There was a significant interaction between time and group (p=.011) indicating that the experimental and control group values were different at any given time point. The interaction of interest between group, training and time did not reach significance (p=.091). Other main effects for RPE (breathlessness) include: RPE (p=.161) and time (p=.000). Analysis for the remaining interaction revealed for RPE and group (p= .364) (Appendix 2-E).

Graph 4-5



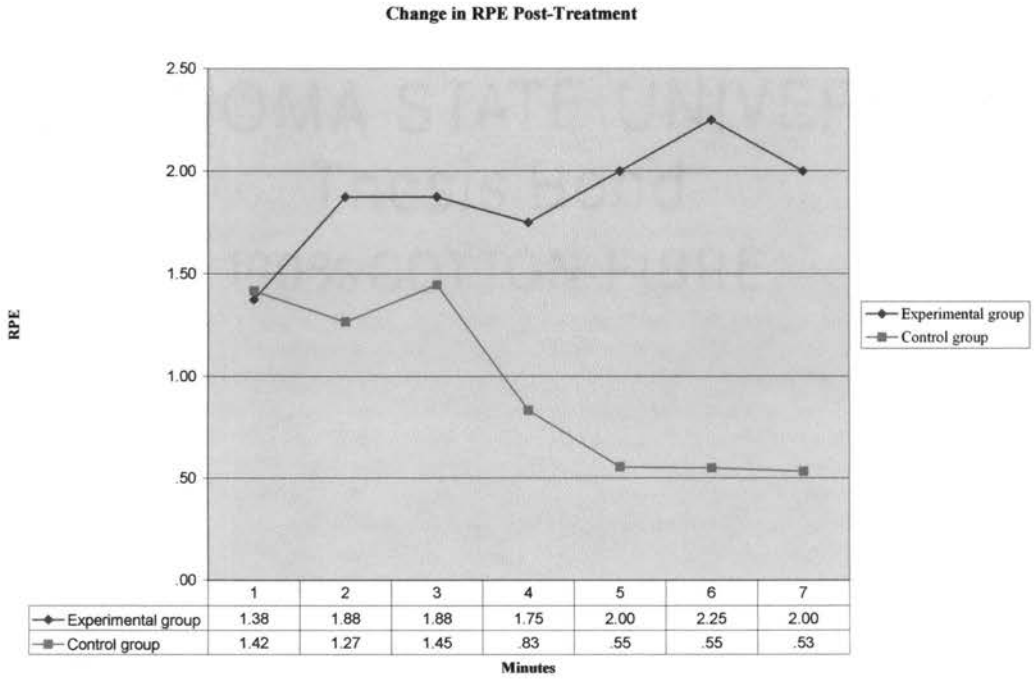
Change in RPE

The change in RPE (Δ RPE) scores found in figure 4-6 were calculated by subtracting the post-training means following the five (5)- week resistive respiratory training session from the pre-training means of both groups at each time point:

post-training mean – pre-training mean= change in RPE

The resultant values for both groups are shown in the graph below. The Δ RPE for the two groups following the five (5)-week resistive respiratory training session begins to deviate after minute one (1) where the experimental group Δ RPE was 1.38 at minute two (2) of the exercise stress test and gradually increased to a Δ RPE of 2 at minute seven (7). The control group Δ RPE exhibited an opposite trend moving from a 1.42 at minute one (1) to a .53 at minute seven (7). However, the differences in Δ RPE between experimental and control was not statistically different. Post-treatment differences between the interaction of group and RPE were (p= .091) (appendix 2-F).

Figure 4-6



	1	2	3	4	5	6	7
Experimental group	1.38	1.88	1.88	1.75	2.00	2.25	2.00
Control group	1.42	1.27	1.45	.83	.55	.55	.53

Other information that was collected, but unplanned for during the study included subjective data relating to RPE scores. Subjects enlisted in the experimental group did report to the primary investigator on the second exercise-stress testing session following the five (5) week training session such responses as it was "easier to breath during and after running", "My chest was not as tight when I was done running..." and other similar anecdotal evidence.

Hypothesis 4

There was no significant difference detected among post-exercise stress testing impulse oscillometry values found in table 4-7. Analyses included respiratory impedance measured at 5Hz (p=.186), total respiratory resistance at 5Hz (p=.105) and at 20Hz (p=.916) as well as distal capacitive reactance at 5 hertz (p=.485) for both groups (appendix 2-G). **Table 4-7**

	GROUP	Mean ±SD
Pre-training Z at5Hz Response	Control	107.79±14.92
	Exp	115.03±18.99
Post-training Z at 5Hz Response	Control	133.96±34.14
	Exp	113.63±15.17

	GROUP	Mean ±SD
Pre-training R at 5Hz Response	Control	109.28±14.30
	Exp	123.01±21.40
Post-training R at 5Hz Response	Control	168.18±90.68
	Exp	116.88±15.41

	GROUP	Mean ±SD
Pre-training R at 20Hz Response	Control	109.25±14.96
	Exp	115.96±19.66
Post-training R at 20Hz Response	Control	113.70±17.14
	Exp	121.95±15.13

	GROUP	Mean ±SD
Pre-training X at 5Hz Response	Control	106.77±34.84
	Exp	143.88±98.71
Post-training X at 5Hz Response	Control	104.70±23.23
	Exp	105.38±18.33

HYPOTHESIS 5

The tables in appendix 2-H show the percent change in post-exercise stress testing measures from pre-exercise stress testing of the experimental groups spirometry scores prior to (baseline) and following a five (5) week training period (response). Independent sample t-tests were run analyzing the experimental groups' post-exercise stress testing to training and found no significant increase along each of the following parameters, FVC, FEV1, FEV1/FVC, PEF, and FEV 25/75. Analyses for FVC ($t = -1.394$; $p=.206$), FEV1 ($t= -1.588$; $p=.156$), FEV1/FVC ($t= -1.948$; $p=.092$), PEF ($t= -1.203$; $p=.268$) and FEV25/27 ($t= -1.159$; $p=.173$).

DISCUSSION OF RESULTS

A majority of the hypotheses tested were not supported by statistical evidence. Many factors contributing to this lack of remarkable findings will be explored in the following narrative. First and foremost, solicitation and retention of the subject population created the most critical pitfalls of the study's methods. The subject population drastically decreased since the initial invitation into the study from twenty-one potential subjects down to twelve volunteer subjects (eight in the experimental and four in the control). As the subject population decreased so did power of the study statistics.

Additionally, review of the FEV-1 values during post-exercise stress testing for each individual also created a concern regarding the target population used during this study. A fifteen (15%) percent decrease in pre-exercise FEV1 following exercise is the standard diagnostic criterion for EIA. Only one subject actually exhibited this decrease of their

resting FEV1 following the seven (7) minute sub-maximal run on the treadmill. This finding raises an interesting question as to how many individuals are misdiagnosed with exercise-induced asthma according to self-reported symptoms. Similar studies investigating the relationship between self-reported symptoms and confirmed exercise-induced asthma and bronchoconstriction also have found a disparity between the number of self-reported cases and confirmed through laboratory testing (Capao-Filipe, et al, 2003, Rice, S., et al, 1985, Rundell, K.W., et al, 2001, Rundell, K.W., et al, 2000, Holzer, K., Anderson, S.D., and Douglas, J., 2002), though not of the magnitude in our study population. The subjects enlisted in this study had all been diagnosed through symptoms associated with EIA and no prior clinical tests had been conducted to determine any spirometric deficiencies in forced expiratory volume. Subsequently, all of the subjects had been prescribed some form of a fast-acting bronchodilator to treat the reported symptoms. However, post-exercise spirometric data in this study was not indicative of treatment prescribed by a physician as all short-term bronchodilating medication was prohibited on the day of testing.

Rundell and Spiering (2003) found that when studying the transient condition of exercise-induced bronchospasm in a large population (370) of Olympic caliber athletes, several of the athletes (9 of the 111 total diagnosed with EIB) actually exhibited a less severe condition called inspiratory stridor. The authors found that most athletes are diagnosed through self reported symptomology as was done within the present study. Koester and Amundenson (2002) also raised an interesting condition called paradoxical vocal-cord dysfunction (PVCD) that is often confused with exercise-induced asthma and could be a

possible explanation for the subject population's pulmonary function tests. The authors report that although pulmonary function tests conducted pre- and post-exercise may be normal, those who suffer from PVCD suffer from a transient closing of the vocal cords causing asthma-like symptoms during exercise. The study's investigation team suggested that any exercise-limiting pathology such as EIB should be diagnosed through spirometric measuring means, thus preventing the misdiagnosis of EIB. None of the volunteer subjects in this study were tested for or reported having been previously diagnosed with inspiratory stridor or PVCD. Based on our inability to confirm EIA in all but one study participant, our data are more correctly interpreted as the effects of respiratory training on normal, trained subjects.

The hypotheses investigating the values of pulmonary mechanics measured through spirometry following an exercise stress test collectively revealed no statistically significant findings, although FEV1/FVC did show a potential trend ($p<0.01$) toward hypothesis confirmation. Additionally, post-exercise stress testing measures of the experimental group using the intact resistive respiratory training device compared to the control group showed an interesting trend whereby the means of the experimental group for all spirometry tests increased while the control group means decreased.

These findings support the previous work done by Chatham, et al. (1999) and Volianitis, et al. (2001) who established minor increases in expiratory measures and performance in healthy subjects after a specified training period and regimen. In these studies, however, larger sample populations ($N>12$), longer training periods (>5 weeks) and more intense

training programs (>30 breathes, 2 times / day) were used, possibly leading to statistically significant results. It is possible that the five (5)-week training period, training protocol and small sample population used in the present study decreased the power to identify such a potential small difference. Sheel (2002) further explains that in many respiratory training studies such as this, small population size typically impact the significance of the results.

The use of an appropriate testing modality and intensity also could have provided for a more powerful look into the potential differences between the training groups. If these individuals were trained, would a treadmill producing a speed fast enough for each subject to reach eighty-five percent (85%) of their maximal heart rate during a seven-minute exercise-stress test be intense enough to truly elicit an EIA response? Although these are the typical conditions used in many diagnostic protocols (ATS, ACSM), Storms (1999) reports that most symptoms of EIA could take as long as fifteen to thirty minutes following the start of exercise to present in the well-conditioned athlete with a testing intensity of up to ninety-five percent (95%) of maximal heart rate. However, the ATS (2001) does stipulate that during these stress tests, it is not necessary to observe actual signs of EIA as respiratory mechanics such as a decrease of FEV1 are usually detected by spirometry.

Subjects in the current study were trained through both inspiratory and expiratory muscle training and then measured through forced expiratory maneuvers. Increased means for spirometric measures involving muscular strength and power (peak expiratory flow,

FEV1, FEV1/FVC) in the experimental group could be explained by adaptive changes in the respiratory muscles through training against an increased resistance. Studies (Lisboa, et al, 1994, 1997, Weiner, et al, 2000, 2002, Inbar, et al, 2000) focusing primarily on inspiratory muscle training for clinical populations reported significant increases in inspiratory muscle strength measured by peak inspiratory force and its' component of speed of inspiration. These studies, however, investigated more subjects per training group and trained individuals for longer periods of time potentially creating a distinguishable difference between groups.

The spirometric data obtained in this present study also could be a result of true physiological response to resistive respiratory training. By not reaching significance, this investigation could support the work of Amonette and Dupler (2001) and Boutellier et al (1992) who found no significant changes in FVC and FEV1 between conditioned athletes randomly separated into a control and experimental group conducting inspiratory and expiratory training. Hanel and Sechler (1991) also found no significant change in FEV1, FVC, FEV1/FVC and Pex. These studies were similar in population and methods thereby raising the question as to whether resistive respiratory training does, in fact, positively affect spirometry of trained individuals or are the testing and training methods too different. Amonette and Dupler (2001) believed that while training on a resistive device, the lungs adapt to the specified resistance and when testing spirometry there is no similar resistance, thereby producing invalid values of spirometry.

The investigation team concedes that the data in this study could be interpreted as a valid indication of respiratory muscle physiology after training for five (5) weeks, however, when viewed relative to previous literature, one must realize the potential for variability due to methods and population.

To our knowledge, this study is the first to investigate resistive respiratory training and its effect on airway resistance through impulse oscillometry. We found no significant change between groups both pre- and post-training, indicating that respiratory airways reacted similarly thought the course of the study regardless of the group training status. Subjects within the experimental group exhibited a consistent albeit insignificant, decrease in all measures of pulmonary resistance when compared to the control group subjects. Interestingly, the second testing session following the five (5) week training period occurred in early April, a spring month in Oklahoma. The spring season also brought air-borne allergens aggravating seasonal allergies that commonly affect asthmatics and in this case, all subjects of this study. Logic would indicate that an increase in the amount of airborne allergens would increase pulmonary resistance measures in both groups equally, however, that was not the case in the experimental group. The control group, on the contrary, saw an increase in all pulmonary resistance measures following the exercise stress test. The experimental groups' chronic use of the resistive respiratory training device possibly cleared and maintained the pulmonary airways even during times of bronchoconstriction.

A significant problem highlighted throughout the data collection is the recording anomalies found while testing several of the volunteer subjects. The Master IOS station software was calibrated prior to each day of testing, however, the equilibration software seemed to misread subject data and therefore report measurement aberrations that were clearly non-physiological in nature. Due to the sensitive and timely nature of each measure collection, forced spirometric maneuvers were only conducted once more if a recording mistake was observed initially.

The rationale for this decision was based primarily in the theory that once the elastic properties of the smooth muscle airways and parenchymal fibers in the lung were stretched from their original "resting" position, any subsequent tidal breathing and forced maneuvers would be registering the newly "stretched" range (Scichilone, et al, 2000, Scichilone, Permutt & Togias, 2001, Skloot, G., Permutt, S., Togias, A. 1995). This phenomenon would result in a false indication of tissue resistance, compliance and the severity of the underlying pulmonary pathology.

The perception of breathlessness by the experimental group subjects as opposed to the control group did not reach statistical significance ($p=0.091$), however, upon a closer review of the reported RPE through the post-training exercise stress test, the subjects within the experimental group reported less of a deviation from the RPE scores of the pre-training exercise stress test. This subjective phenomenon could again be explained by the resistive respiratory training's ability to decrease the sense of breathing exertion. As experimental group members became increasingly trained to forcefully breathe against an

artificial resistance, respiratory musculature developed a more efficient ability to move air in and out of the airways during episodes of perceived bronchoconstriction. These trained individuals would decrease the overall effort used to breath during and after exercise, thereby decreasing their sense of exertion and breathlessness.

This effect is best seen in figure 4-6, which shows a growing disparity between the experimental and control groups. As shown on the chart, the experimental group average progression of change in RPE from pre-training to post-training consistently increased throughout the seven (7) minute exercise stress test whereas the control group posted a smaller rate of change. Statistical analysis between both groups average change in RPE over time showed $p< 0.1$ ($p=0.091$), a trend towards hypothesis confirmation that should be noted, particularly with the low statistical power of our study.

Our data coincided with other research (Wiener, et al, 2000, 2002, Suzuki, et al, Volianitis, et al, 2000) showing that resistive respiratory training could possibly attenuate the sensation of dyspnea involved with strenuous exercise. If drawing and expiring a breath against an increased resistance develops neural adaptations providing for more efficient movement of air, we agree with other research that less energy expended and less fatigue induced through strenuous exercise will yield a decreased sense of exertion and breathlessness.

CHAPTER 5

CONCLUSIONS

Upon review of the data represented in the previous chapter, it is evident that the study methods were severely impacted by the small subject population. Subsequently, definitive statements regarding each hypothesis are difficult to pose and support in light of the small subject population and equipment recording reliability. The reader must consider these points when evaluating the data and associated conclusions.

Although none of the research hypotheses reached statistical significance there are several areas of interest raised when evaluating the data for the experimental groups' pulmonary mechanics scores.

When reviewing the data of the experimental group's post-exercise stress testing measures, there did seem to be a slight increase in all relevant forced expiratory maneuvers. This phenomenon could be due to an actual training effect as hypothesized or due to erroneous results. The increase could potentially be due to an increase in expiratory muscle strength brought upon by breathing into the resistive respiratory training device on a progressive five (5)-week cycle.

Oscillometry data showed that although no significant changes were recorded pre- and post-training, resistive respiratory training did not increase nor decrease respiratory resistance after a five (5) week training period.

Perception of exertion or breathlessness could potentially be improved through resistive respiratory training over a five (5) week period. Our data are consistent with other research that training with some resistance has a greater effect on perception of exertion and breathlessness than training without.

RECOMMENDATIONS

It is clear that future research investigating this area of resistive respiratory training for the treatment of athletes diagnosed with exercise-induced asthma should correct the methodological problems experienced within this study. Increasing volunteer subject population would prevent many of the problems of statistical analysis and the subsequent ability to generalize to the greater population allowing the proper interpretation of future research data. Many of the intriguing albeit insignificant findings of the present data could be further investigated.

Maintaining reliable diagnostic pulmonary equipment capable of recording a time series of measures would prevent data collection aberrations and contribute to the data validity. Although the device used in this study was calibrated daily and maintained acceptable measure validity, the progressively scheduled manner in which data samples were collected created much of the measurement error observed. By following the study's data collection methods and maintaining the timed structure of the data collection, it was

difficult not to contribute to the measurement error. Communication with device manufacturers did not correct the collection problems. Future research should seek and certify that the data collection equipment used will have the ability to accurately collect series of samples without re-calibrating the unit in between samples.

Future research should concentrate on the differentiation of exercise-induced asthma and exercise-induced bronchospasm to elucidate the actual underlying pathology and stimuli of each. The realization that only one of twelve of the subjects tested actually suffered from EIA is disconcerting and highlights the problem of identifying a truly affected population. Targeting inflammatory markers of a true asthmatic condition such as an elevated white blood cell count and increased release of histamine versus a temporary narrowing of the smooth muscle airways without any associated inflammation seen with EIB would improve population identification (Hermansen, 2004). The ability to differentially diagnose exercise-triggered asthma from transient EIB through clinical testing would allow future research to develop more accurate hypotheses and subsequent conclusions based on pathology.

Along with a decreased sense of exertion evidenced by decreased RPE scores, an area of interest that developed throughout the study was that of pharmacological treatment patterns. Although no actual record of fast-acting bronchodilators was kept throughout the study, many individuals in the experimental group reported using their prescription medication for EIA less if not at all. Interestingly, there were no such reports from those subjects within the placebo control group. Further research should include this variable

in the methods to lend further support to the notion that resistive respiratory training can serve as training aid in general.

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Appendix 1-aa
Institutional Review Board Application IRB# E0468 Approval

Oklahoma State University
Institutional Review Board

Protocol Expires: 1/14/2005

Date: Thursday, January 15, 2004 IRB Application No ED0468

Proposal Title: Resistive Respiratory Training and it Effects on Pulmonary Mechanics in Individuals Diagnosed with Exercise Induced Asthma

Principal Investigator(s):

Matthew O'Brien
418 Willard
Stillwater, OK 74078

Michael Davis
264 McElroy Hall
Stillwater, OK 74078

Reviewed and Processed as: Full Board

Approval Status Recommended by Reviewer(s): Approved

Dear PI :

Your IRB application referenced above has been approved for one calendar year. Please make note of the expiration date indicated above. It is the judgment of the reviewers that the rights and welfare of individuals who may be asked to participate in this study will be respected, and that the research will be conducted in a manner consistent with the IRB requirements as outlined in section 45 CFR 46.

As Principal Investigator, it is your responsibility to do the following:

1. Conduct this study exactly as it has been approved. Any modifications to the research protocol must be submitted with the appropriate signatures for IRB approval.
2. Submit a request for continuation if the study extends beyond the approval period of one calendar year. This continuation must receive IRB review and approval before the research can continue.
3. Report any adverse events to the IRB Chair promptly. Adverse events are those which are unanticipated and impact the subjects during the course of this research; and
4. Notify the IRB office in writing when your research project is complete.

Please note that approved projects are subject to monitoring by the IRB. If you have questions about the IRB procedures or need any assistance from the Board, please contact me in 415 Whitehurst (phone: 405-744-5700, colson@okstate.edu).

Sincerely,

Carol Olson, Chair
Institutional Review Board

Appendix 1-bb
Institutional Review Board Application IRB# E0468 Adjoiner

Oklahoma State University
Institutional Review Board

Protocol Expires: 1/14/2005

Date : Thursday, April 22, 2004 IRB Application No ED0468

Proposal Title: Resistive Respiratory Training and it Effects on Pulmonary Mechanics in Individuals Diagnosed with Exercise Induced Asthma

Principal Investigator(s) :

Matthew O'Brien
418 Willard
Stillwater, OK 74078

Michael Davis
264 McElroy Hall
Stillwater, OK 74078

Reviewed and Processed as: Full Board

Approval Status Recommended by Reviewer(s) : Approved **Modification**

Please note that the protocol expires on the following date which is one year from the date of the approval of the original protocol:

Protocol Expires: 1/14/2005

Signature : Carol Olson

Carol Olson, Director of University Research Compliance

Thursday, April 22, 2004
Date

Approvals are valid for one calendar year, after which time a request for continuation must be submitted. Any modifications to the research project approved by the IRB must be submitted for approval with the advisor's signature. The IRB office MUST be notified in writing when a project is complete. Approved projects are subject to monitoring by the IRB. Expedited and exempt projects may be reviewed by the full Institutional Review Board.

OKLAHOMA STATE UNIVERSITY RESEARCH

Resistive respiratory training and its effects on the pulmonary mechanics within individuals diagnosed with exercise induced asthma

IRB# E0468

Investigation Procedure Explanation

According to the original IRB approved methodology, two groups of subjects were used to ensure a true experimental design consisting of a control and experimental group. Upon initial testing of pulmonary function prior to the five (5) week training period, subjects were randomly assigned into an experimental group and issued an intact resistive training device while the control group received the modified device offering a uniform minimal resistance throughout the same training period. This approved methodology was formulated so that the potential for measuring a treatment effect could be established. At the conclusion of the training period all subjects' pulmonary function were tested again and subjects within the control group were debriefed regarding their participation within the study. These subjects were informed of the modified resistive training device and then issued a fully intact device as explained in the original methods and informed consent form. Each was instructed again in the proper use of the device and advised that this newly issued resistive respiratory training device will provide for a greater resistance when used in the same fashion as their previously modified devices. All other procedures as presented in the original methods and informed consent letter remained unchanged.

_____

Matthew S. O'Brien, M.Ed., ATC/L
Primary Investigator

Appendix 1-A
Letter of Informed Consent

OKLAHOMA STATE UNIVERSITY RESEARCH
LETTER OF CONSENT FORM

Resistive respiratory training and its effects on the pulmonary mechanics within individuals diagnosed with exercise induced asthma

AUTHORIZATION

I,_____ (Please clearly print name) , hereby authorize Matthew S. O'Brien, M.Ed., ATC, Dr. Michael Davis, DVM, PhD, or Ken Smith, DO to perform the following procedures to assess my respiratory function.

DESCRIPTION OF RESEARCH AND ITS ASSOCIATED RISKS AND BENEFITS

The Research Project. You are invited to participate in a research study investigating the effects of resistive respiratory training on pulmonary function within individuals diagnosed with exercise induced asthma. The Principal Investigators are Matthew S. O'Brien, Dr. Ken Smith, DO, Oklahoma State University Health Services and Dr. Michael Davis, DVM, PhD, Physiological Sciences, Oklahoma State University, Stillwater OK. This invitation has been extended because you have expressed interest in participating in a study of your exercise induced asthma.

Description of project: This six week study has been created to determine the effectiveness of a resistive training device on the pulmonary function of a person diagnosed with exercise induced asthma. It is theorized that by limiting the amount of air one can inhale and exhale, the muscles that control the breathing process will undergo some training effect much like resistive weight training for the musculoskeletal system. It is hoped that the results from this study will introduce a new method of controlling the severity of symptoms associated with exercise induced asthma and improve lung function in athletes.

You were invited as a participant in this study because of your diagnosis with exercise induced asthma. If you choose to participate in this study, you will be asked to report two (2) different times, once at the beginning of the study and once at the end of the five week training period, to a testing site in the Seretean Wellness Center, West Farm Road, located on campus. On days of testing, it is important that you do not engage in any vigorous exercise or use any rescue inhalers for your exercise induced asthma prior to the testing as this will mask potential results. However, it is important that you bring any rescue inhalers prescribed to you (for exercise induced asthma) from your physician in case you might need them during the testing procedures. On both occasions, you will partake in a respiratory measurement that will require you to breath into a device that will assess several factors regarding your respiratory function. You will then be asked to engage in a seven (7) minute sub-maximal (about 85% of your maximal heart rate) run on a standard treadmill. If at any time during the testing procedures you or the investigator feels you might pose risk to your health, all testing will be stopped and your participation in the study will be excused. At the end of a uncomplicated seven minute run, you will perform the breathing measures one more time. At the conclusion of these tests, the primary investigator (Matthew O'Brien) will give you a resistive respiratory training device and educate you on its proper use. To assure that you do not suffer from any secondary effects of the testing procedures (i.e. severe asthmatic attack) we will monitor you while at the testing facility for approximately one hour after the treadmill test. You will be asked to follow the protocol requiring you to breath into this

Subjects Initials _____

device for approximately 2 minutes, two times per day for a period of 5 weeks. It is very important that you adhere to this protocol as the study's success depends on your compliance.

IMPORTANT: The resistive respiratory training device used in this study will **not** substitute for a fast acting bronchodilator such as Albuterol®, or Ventolin®. Use your rescue inhaler as directed by your physician throughout the course of the five-week training period.

Duration of the study: This investigation will last approximately six weeks including one for testing before and after a five (5) week training period. You may, however, withdraw your participation from the study at anytime.

Risks: This study's procedures do not pose any greater risk than you would encounter within your participation in collegiate athletics. The seven minute sub-maximal run will resemble a moderate running drill within your conditioning program

In case of injury or illness resulting from participation in the study, the Oklahoma State University is not financially responsible for any subsequent medical care. No funds have been set aside to compensate subjects medical treatment due to participation. During testing at the Seretean Wellness center, a member of the investigation team (Matthew O'Brien) will be present to offer any assistance such as cardio-pulmonary resuscitation (CPR) including automatic external defibrillation and/or first aid. There will also be a licensed physician on call (Dr. Ken Smith) and readily accessible if needed.

Benefits: You will not be paid for your participation in this study. However, upon successful completion of the project, you will be allowed to keep the resistive respiratory training device. Also, you will learn several factors regarding your respiratory function such as flow and lung volumes.

Confidentiality: At no time will your name or identity be disclosed in any publication or presentation of the results of this study. Only Matthew S. O'Brien, Dr. Ken Smith, DO, OSU Health Services and Dr. Michael Davis, DVM, PhD will view any information associated with your name obtained through this study regarding your participation. Information kept in hard copy or computer form will be secured through locked file cabinets and password protected computer software in Matthew O'Brien's locked office. At the conclusion of the study, all paper materials and records indicating personal information such as your name will be shredded while all computer data will be deleted using a computer program that prevents electronic recovery. Although it is impossible to guarantee complete confidentiality throughout the course of this study and the time proceeding its conclusion, every reasonable effort will be made to ensure your privacy. The federal funding agency (National Institutes of Health), regulatory agencies, and the OSU Institutional Review Board have the right to review research protocols.

Cost: There will be no cost to participate in this investigation. All materials and equipment use have been donated.

Other information: If any significant new findings are developed during the course of the research that may relate to your willingness to continue participation, we will provide them to you. Number of subjects: There will be approximately 24 subjects involved in this study.

If you have any questions regarding this research study or your participation in it, please feel free to contact Matthew S. O'Brien at (405) 744-9439 or matthso@okstate.edu. This research project

Subjects Initials _____

has been reviewed by the Institutional Review Board of the Oklahoma State University. You may request information from this Board about your rights as a research subject or research-related injuries. For information on subjects' rights, contact Dr. Carol Olson, Institutional Review Board Chair, 415 Whitehurst, 405-744-1676.

IMPORTANT: The resistive respiratory training device used in this study will **not** substitute for a fast acting bronchodilator such as Albuterol®, or Ventolin®. Use your rescue inhaler as directed by your physician throughout the course of the five-week training period.

VOLUNTARY PARTICIPATION

Your participation in this study is entirely voluntary. If you decide not to participate, you will not be penalized, and you will not lose any benefits or services to which you are otherwise entitled. Your decision whether or not to participate will not prejudice your future relations with Oklahoma State University. If you decide to participate, you are free to withdraw your consent and to stop participating at any time without penalty or loss of benefits to which you are otherwise entitled.

Terminating your participation: If you choose to be part of the study, you will be asked to report for the first set of respiratory measurements and then adhere to the training program outlined for you. If you decide that you do not wish to participate at any time before the study's conclusion, we ask that you inform Matthew S. O'Brien and return the resistive respiratory training device. Other circumstances may arise that may impact your participation within this study without regard to your consent. If your compliance with the research protocol becomes sporadic or if subject population parameters change, your participation may be discontinued.

If you desire to stop participating, simply inform Matthew S. O'Brien at (405) 744-9439 or matthso@okstate.edu. There will be no adverse consequences of your decision to withdraw from the project.

CONSENT DOCUMENTATION

Participant: I have read and fully understand the consent form. I sign it freely and voluntarily. A copy has been given to me.

_____ _____

Printed Name of Participant Signature of Participant

Date_____

Investigator: I certify that I have personally explained all elements of this form to the participant before requesting him/ her to sign it.

_____

Matthew S. O'Brien
Primary Investigator

Appendix 1-B
Exercise-induced asthma and Allergy Questionnaire

Exercise-Induced Asthma Screening Questionnaire
Asthma and Allergy History

1. Do you have, or have you ever had, asthma? Y N

2. Have you ever had wheezing? Y N

3. Do you have, or have you ever had, allergies to dust, pollen, or animal dander? Y N

4. Have you ever had a chronic cough? Y N

5. Do you have nasal polyps, allergic rhinitis, or eczema? Y N

6. Have you ever had shortness of breath, difficulty breathing, cough, wheezing, chest pain, or chest tightness during or **after** exercise? Y N

7. If you were to run a kilometer (about half of a mile), would you have to stop to catch your breath or have trouble breathing **afterward**? Y N

8. Do you smoke? Y N

Medical History

9. Are you currently taking any medication? Y N

If yes, what medication?_____

10. Have you had a recent respiratory tract infection? Y N

11. Have you recently received the influenza vaccine? (Can exacerbate asthma.) Y N

12. Do you have a history of heart disease, abnormal heart rhythm, or high blood pressure? Y N

13. Do you have my other medical problem that could be worsened with strenuous activity? Y N

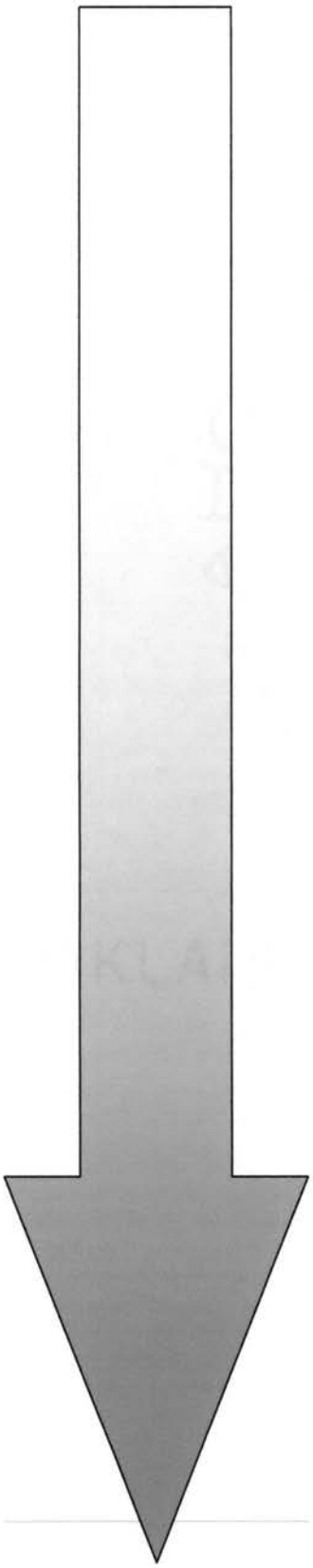
If yes, what condition(s)?_____

14. Does anyone in your family have asthma? Y N

Adapted from Feinstein RA, LaRussa J, Wang-Dohlman A, et al: Screening adolescent athletes for exercise-induced asthma. Clin J Sport Med 1996;6(2):119-123

Appendix 1-C
Borg Rating of Perceived Exertion (RPE) scale

BORG'S RATINGS OF PERCEIVED EXERTION SCALE



6	No Exertion at all
7	Very, very light
8	
9	Very Light
10	
11	Fairly Light
12	
13	Somewhat hard
14	
15	Hard, Heavy
16	
17	Very Hard
18	
19	Extremely Hard
20	MAXIMAL EXERTION

Appendix 1-D
Respiratory Training Instructions

RESPIRATORY TRAINING

Resistance INHALE EXHALE	**Sunday**	**Monday**	**Tuesday**	**Wednesday**	**Thursday**	**Friday**	**Saturday**
Week 1 **I= 1 E= 1**	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM
Week 2 **I= 2 E= 2**	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM
Week 3 **I= 3 E= 3**	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM
Week 4 **I= 4 E= 4**	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM
Week 5 **I= 4 E= 5**	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM
If necess. I= 4 E= 5	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM	AM / PM

Training Tips

1. Use lips, NOT teeth to grip the SportsBreather®.
2. INHALE for 3-4 seconds, EXHALE for 5 seconds.
3. If you feel faint or begin to cough during training, stop, let the symptoms subside and continue.
4. THIS DEVICE SHOULD NOT BE USED IN PLACE OF A FAST ACTING BRONCHODILATOR.
5. Use ONLY the device you were assigned.

I will call every Friday of the training period to make sure you are experiencing no problems with the device or training.

Appendix 2-A
Study Factors and Categories

Study Factors and Categories

PRE-EXERCISE STRESS TESTING (BASELINE)			
Experimental		Control	
Pre-Training	Post- Training	Pre- Training	Post- Training

POST-EXERCISE STRESS TESTING (RESPONSE)			
Experimental		Control	
Pre- Training	Post- Training	Pre- Training	Post- Training

Appendix 2-B
t-tests for Randomization

t-tests for Randomization

Independent Samples Test

	Levene's Test for Equality of Variances		t-test for Equality of Means						
								95% Confidence Interval of the Difference	
	F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
A_Z_5HZ	1.455	.255	.353	10	.731	.4250	1.20244	-2.25421	3.10421
A_R_5HZ	.962	.350	.351	10	.733	.3962	1.12962	-2.12069	2.91319
A_R_20HZ	.742	.409	.348	10	.735	.2575	.73965	-1.39054	1.90554
A_X_5HZ	5.384	.043	-.320	10	.756	-.1525	.47697	-1.21526	.91026
A_FVC	3.766	.081	2.279	10	.046	1.2325	.54070	.02774	2.43726
A_FEV_1	.842	.380	1.499	10	.165	.8987	.59953	-.43708	2.23458
AFEV1FVC	1.568	.239	-.390	10	.705	-2.4438	6.26291	-16.39839	11.51089
A_PEF	.360	.562	1.019	10	.332	1.9063	1.87142	-2.26354	6.07604
AFEF2575	.499	.496	.305	10	.767	.2938	.96423	-1.85469	2.44219

Appendix 2-C
Hypothesis 1: Baseline Spirometry Group & Training Measures

Hypothesis 1: Baseline Spirometry Group & Training Measures

FVC

Group	Pre-training	Post- training
0	3.90	3.69
0	6.17	6.98
0	4.87	4.78
0	6.61	6.32
Mean	5.39	5.44
SD	1.24	1.49

FVC

Group	Pre- training	Post- training
1	2.88	3.26
1	4.07	3.77
1	4.75	4.39
1	4.86	5.24
1	4.17	4.18
1	4.16	3.48
1	4.90	5.04
1	3.83	3.88
Mean	4.20	4.16
SD	.67	.71

FEV1

Group	Pre- training	Post- training
0	2.44	.90
0	5.00	4.84
0	4.16	3.96
0	5.34	5.33
Mean	4.24	3.76
SD	1.30	1.99

FEV1

Group	Pre- training	Post- training
1	2.18	.72
1	2.38	3.20
1	3.77	1.29
1	4.60	4.80
1	3.87	2.15
1	3.64	3.44
1	3.28	3.85
1	2.97	3.38
Mean	3.34	2.85
SD	.80	1.36

FEV1/ FVC

Group	Pre- training	Post- training
0	66.35	76.44
0	82.08	71.39
0	82.88	82.97
0	80.77	83.33
Mean	78.02	78.53
SD	7.83	5.72

FEV/FVC

Group	Pre- training	Post- training
1	71.15	66.51
1	65.58	83.39
1	86.89	81.93
1	92.55	91.30
1	94.53	53.67
1	85.29	85.15
1	68.47	77.87
1	79.25	87.18
Mean	80.46	78.38
SD	11.10	12.42

PEF

Group	Pre- training	Post- training
0	2.97	1.54
0	7.95	7.82
0	7.99	6.40
0	13.16	6.71
Mean	8.02	5.62
SD	4.16	2.79

PEF

Group	Pre- training	Post- training
1	5.09	1.79
1	2.94	5.39
1	7.47	3.49
1	10.85	10.64
1	5.95	3.24
1	7.36	7.19
1	4.61	5.74
1	4.62	6.41
Mean	6.11	5.49
SD	2.43	2.75

FEF25/75

Group	Pre-treatment	Post-treatment
0	1.71	.65
0	4.70	3.56
0	3.87	3.90
0	4.80	5.76
Mean	3.77	3.47
SD	1.44	2.11

FEF25/75

Group	Pre-treatment	Post-treatment
1	1.46	.41
1	1.63	3.46
1	4.11	1.13
1	5.91	6.08
1	5.28	1.14
1	4.04	3.98
1	2.75	3.45
1	2.63	3.67
Mean	3.48	2.92
SD	1.63	1.89

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
FVC	Sphericity Assumed	1.853	1	1.853	2.498	.145
FVC * GROUP	Sphericity Assumed	1.505E-02	1	1.505E-02	.020	.890
Error(FVC)	Sphericity Assumed	7.416	10	.742		

Tests of Within-Subjects Effects

Measure: TIME

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
FEV1	Sphericity Assumed	1.229	1	1.229	2.023	.185
FEV1 * GROUP	Sphericity Assumed	3.333E-05	1	3.333E-05	.000	.994
Error(FEV1)	Sphericity Assumed	6.074	10	.607		

Tests of Within-Subjects Effects

Measure: TIME

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
FEV1_FVC	Sphericity Assumed	3.313	1	3.313	.028	.871
FEV1_FVC * GROU	Sphericity Assumed	9.022	1	9.022	.076	.788
Error(FEV1_FVC)	Sphericity Assumed	1186.193	10	118.619		

Tests of Within-Subjects Effects

Measure: TIME

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
PEF	Sphericity Assumed	12.201	1	12.201	3.774	.081
PEF * GROUP	Sphericity Assumed	4.201	1	4.201	1.299	.281
Error(PEF)	Sphericity Assumed	32.329	10	3.233		

Tests of Within-Subjects Effects

Measure: TIME

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
FEV25_75	Sphericity Assumed	.995	1	.995	.611	.452
FEV25_75 * GROUI	Sphericity Assumed	8.927E-02	1	8.927E-02	.055	.820
Error(FEV25_75)	Sphericity Assumed	16.268	10	1.627		

Appendix 2-D
Hypothesis 2: Response Spirometry Group & Training Measures

Hypothesis 2: Response Spirometry Group & Training Measures

FVC Response (% of pre-exercise)

Group	Pre-training	Post- training	Difference
0	105.98	312.71	206.73
0	101.31	102.95	1.64
0	97.21	106.94	9.73
0	100.00	98.90	-1.1
Mean	101.12	155.38	54.26
SD	3.66	104.94	101.28

FVC Response (% of pre-exercise)

Group	Pre- training	Post- training	Difference
1	94.12	299.08	204.96
1	112.12	98.18	-13.94
1	109.45	277.85	168.4
1	97.79	99.81	2.02
1	101.71	104.24	2.53
1	97.42	86.14	-11.28
1	102.08	102.02	-0.06
1	102.13	100.00	-2.13
Mean	102.10	145.91	43.81
SD	6.08	88.33	82.25

FEV1 Response (% of pre-exercise)

Group	Pre- training	Post- training	Difference
0	127.46	303.33	175.87
0	98.60	100.00	1.4
0	91.11	96.97	5.86
0	103.56	102.06	-1.5
Mean	105.18	150.59	45.41
SD	15.71	101.85	86.14

FEV1 Response (% of pre-exercise)

Group	Pre- training	Post- training	Difference
1	89.45	386.11	296.66
1	100.84	100.63	-0.21
1	98.94	296.90	197.96
1	68.91	97.92	29.01
1	103.10	126.98	23.88
1	93.96	88.08	-5.88
1	118.60	104.94	-13.66
1	106.06	100.89	-5.17
Mean	97.48	162.80	65.32
SD	14.42998	113.37	98.94002

FEV1/ FVC Response (% of pre-exercise)

Group	Pre- training	Post- training	Difference
0	120.24	96.85	-23.39
0	99.00	97.10	-1.9
0	94.35	96.81	2.46
0	106.81	103.28	-3.53
Mean	105.10	98.51	-6.59
SD	11.33	3.18	-8.15

FEV/FVC Response (% of pre-exercise)

Group	Pre- training	Post- training	Difference
1	95.17	127.95	32.78
1	90.01	102.28	12.27
1	100.68	106.53	5.85
1	70.45	99.32	28.87
1	101.20	127.59	26.39
1	98.79	102.33	3.54
1	116.53	102.90	-13.63
1	105.03	100.88	-4.15
Mean	97.23	108.72	11.49
SD	13.29	11.93	-1.36

PEF Response (% of pre-exercise)

Group	Pre- training	Post- training	Difference
0	184.18	306.49	122.31
0	97.48	112.66	15.18
0	74.47	120.31	45.84
0	89.51	130.85	41.34
Mean	111.41	167.58	56.17
SD	49.44	92.91	43.47

PEF Response (% of pre-exercise)

Group	Pre- training	Post- training	Difference
1	90.18	401.12	310.94
1	105.78	93.88	-11.9
1	91.70	197.99	106.29
1	53.27	108.18	54.91
1	119.16	109.88	-9.28
1	95.52	97.08	1.56
1	134.92	115.68	-19.24
1	144.37	104.84	-39.53
Mean	104.36	153.58	49.22
SD	28.80	105.42	76.62

FEF 25/ 75Response (% of baseline)

Group	Pre- training	Post- training	Difference
0	153.80	332.31	178.51
0	100.21	83.71	-16.5
0	88.11	85.64	-2.47
0	119.79	105.56	-14.23
Mean	115.48	151.80	36.32
SD	28.69	120.74	92.05

FEF 25/ 75 Response (% of baseline)

Group	Pre- training	Post- training	Difference
1	78.77	700.00	621.23
1	130.06	106.36	-23.7
1	102.92	364.60	261.68
1	37.56	98.85	61.29
1	93.75	176.32	82.57
1	89.85	79.90	-9.95
1	129.45	99.71	-29.74
1	109.51	107.90	-1.61
Mean	96.48	216.70	120.22
SD	29.91	216.26	186.35

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
FVC	Sphericity Assumed	12821.898	1	12821.898	2.968	.116
FVC * GROUF	Sphericity Assumed	145.285	1	145.285	.034	.858
Error(FVC)	Sphericity Assumed	43207.424	10	4320.742		

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
FEV1	Sphericity Assumed	16349.057	1	16349.057	2.784	.126
FEV1 * GROUF	Sphericity Assumed	528.597	1	528.597	.090	.770
Error(FEV1)	Sphericity Assumed	58720.810	10	5872.081		

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
FEV1FVC	Sphericity Assumed	31.973	1	31.973	.273	.613
FEV1FVC * GROL	Sphericity Assumed	436.062	1	436.062	3.719	.083
Error(FEV1FVC)	Sphericity Assumed	1172.413	10	117.241		

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
PEF	Sphericity Assumed	14808.075	1	14808.075	2.957	.116
PEF * GROUP	Sphericity Assumed	64.437	1	64.437	.013	.912
Error(PEF)	Sphericity Assumed	50082.659	10	5008.266		

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
FEF2575	Sphericity Assumed	32674.515	1	32674.515	1.729	.218
FEF2575 * GROUI	Sphericity Assumed	9384.974	1	9384.974	.497	.497
Error(FEF2575)	Sphericity Assumed	188986.879	10	18898.688		

Appendix 2-E
Hypothesis 3: RPE, Group & Training

Hypothesis 3: RPE, Group & Training

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
RPE	Sphericity Assumed	49.527	1	49.527	2.294	.161
RPE * GROUP	Sphericity Assumed	19.527	1	19.527	.904	.364
Error(RPE)	Sphericity Assumed	215.920	10	21.592		
TIME	Sphericity Assumed	375.869	6	62.645	46.692	.000
TIME * GROUP	Sphericity Assumed	24.583	6	4.097	3.054	.011
Error(TIME)	Sphericity Assumed	80.500	60	1.342		
RPE * TIME	Sphericity Assumed	2.536	6	.423	.711	.642
RPE * TIME * GROI	Sphericity Assumed	6.869	6	1.145	1.927	.091
Error(RPE*TIME)	Sphericity Assumed	35.643	60	.594		

Appendix 2-F
Change in RPE, Group & Training

Change in RPE, Group & Training

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
RPE	Sphericity Assumed	5.071	6	.845	.711	.642
RPE * GROUP	Sphericity Assumed	13.738	6	2.290	1.927	.091
Error(RPE)	Sphericity Assumed	71.286	60	1.188		

Appendix 2-G
Hypothesis 4: Response Oscillometry Group & Training

Hypothesis 4: Response Oscillometry Group & Training

Z at 5 Hz Response (% of pre-exercise)

Group	Pre-treatment	Post-treatment	Difference
0	87.70	175.96	88.26
0	123.51	137.54	14.03
0	108.01	129.63	21.62
0	111.95	92.71	-19.24
Mean	107.79	133.96	26.17
SD	14.92	34.14	19.22

Z at 5 Hz Response (% of pre-exercise)

Group	Pre-treatment	Post-treatment	Difference
1	106.44	129.11	22.67
1	118.93	110.23	-8.7
1	121.87	141.21	19.34
1	103.52	110.61	7.09
1	124.07	106.32	-17.75
1	104.71	115.90	11.19
1	152.09	102.06	-50.03
1	88.64	93.65	5.01
Mean	115.03	113.63	-1.4
SD	19.00	15.18	-3.82

R at 5 Hz Response (% of pre-exercise)

Group	Pre-treatment	Post-treatment	Difference
0	89.95	300.80	210.85
0	122.36	141.59	19.23
0	107.46	134.64	27.18
0	117.37	95.71	-21.66
Mean	109.29	168.19	58.9
SD	14.30	90.69	76.39

R at 5 Hz Response (% of pre-exercise)

Group	Pre-treatment	Post-treatment	Difference
1	151.40	132.85	-18.55
1	124.07	109.07	-15
1	127.76	143.75	15.99
1	112.95	120.56	7.61
1	122.14	109.67	-12.47
1	107.46	118.40	10.94
1	150.82	102.93	-47.89
1	87.53	97.83	10.3
Mean	123.02	116.88	-6.14
SD	21.40	15.42	-5.98

R at 20 Hz Response (% of pre-exercise)

Group	Pre-treatment	Post-treatment	Difference
0	90.32	105.98	15.66
0	125.39	136.67	11.28
0	105.70	115.53	9.83
0	115.61	96.62	-18.99
Mean	109.26	113.70	4.44
SD	14.96	17.15	2.19

R at 20 Hz Response (% of pre-exercise)

Group	Pre-treatment	Post-treatment	Difference
1	100.76	148.16	47.4
1	118.77	118.55	-0.22
1	124.11	142.99	18.88
1	102.48	111.44	8.96
1	117.18	113.20	-3.98
1	110.71	120.25	9.54
1	158.11	112.82	-45.29
1	95.59	108.21	12.62
Mean	115.96	121.95	5.99
SD	19.67	15.13	-4.54

X at 5 Hz Response (% of pre-exercise)

Group	Pre-treatment	Post-treatment	Difference
0	73.83	129.28	55.45
0	150.98	119.59	-31.39
0	117.58	87.83	-29.75
0	84.71	82.14	-2.57
Mean	106.78	104.71	-2.07
SD	34.85	23.23	-11.62

X at 5 Hz Response (% of pre-exercise)

Group	Pre-treatment	Post-treatment	Difference
1	369.55	124.64	-244.91
1	97.39	129.17	31.78
1	59.84	113.33	53.49
1	83.06	80.67	-2.39
1	134.48	119.08	-15.4
1	91.87	93.97	2.1
1	181.97	88.24	-93.73
1	132.95	93.97	-38.98
Mean	143.89	105.38	-38.51
SD	98.71	18.33	-80.38

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Z5HZ	Sphericity Assumed	818.073	1	818.073	1.628	.231
Z5HZ * GROUF	Sphericity Assumed	1013.271	1	1013.271	2.016	.186
Error(Z5HZ)	Sphericity Assumed	5026.206	10	502.621		

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
R5HZ	Sphericity Assumed	3712.492	1	3712.492	2.096	.178
R5HZ * GROUF	Sphericity Assumed	5639.106	1	5639.106	3.184	.105
Error(R5HZ)	Sphericity Assumed	17709.006	10	1770.901		

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
R20HZ	Sphericity Assumed	145.169	1	145.169	.531	.483
R20HZ * GROUF	Sphericity Assumed	3.183	1	3.183	.012	.916
Error(R20HZ)	Sphericity Assumed	2732.711	10	273.271		

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
X5HZ	Sphericity Assumed	2194.935	1	2194.935	.651	.439
X5HZ * GROUF	Sphericity Assumed	1770.726	1	1770.726	.525	.485
Error(X5HZ)	Sphericity Assumed	33738.133	10	3373.813		

Appendix 2-H
Hypothesis 5: Response Spirometry, Experimental Group & Training

Hypothesis 5: Response Spirometry, Experimental Group & Training

FVC Response (% of pre-exercise)

Group	Pre-training	Post- training	Difference
1	94.12	299.08	204.96
1	112.12	98.18	-13.94
1	109.45	277.85	168.4
1	97.79	99.81	2.02
1	101.71	104.24	2.53
1	97.42	86.14	-11.28
1	102.08	102.02	-0.06
1	102.13	100.00	-2.13
Mean	102.10	145.91	43.81
SD	6.08	88.33	82.25

FEV1 Response (% of pre-exercise)

Group	Pre- training	Post- training	Difference
1	89.45	386.11	296.66
1	100.84	100.63	-0.21
1	98.94	296.90	197.96
1	68.91	97.92	29.01
1	103.10	126.98	23.88
1	93.96	88.08	-5.88
1	118.60	104.94	-13.66
1	106.06	100.89	-5.17
Mean	97.48	162.80	65.32
SD	14.42998	113.37	98.94002

FEV/FVC Response (% of pre-exercise)

Group	Pre- training	Post- training	Difference
1	95.17	127.95	32.78
1	90.01	102.28	12.27
1	100.68	106.53	5.85
1	70.45	99.32	28.87
1	101.20	127.59	26.39
1	98.79	102.33	3.54
1	116.53	102.90	-13.63
1	105.03	100.88	-4.15
Mean	97.23	108.72	11.49
SD	13.29	11.93	-1.36

PEF Response (% of pre-exercise)

Group	Pre- training	Post- training	Difference
1	90.18	401.12	310.94
1	105.78	93.88	-11.9
1	91.70	197.99	106.29
1	53.27	108.18	54.91
1	119.16	109.88	-9.28
1	95.52	97.08	1.56
1	134.92	115.68	-19.24
1	144.37	104.84	-39.53
Mean	104.36	153.58	49.22
SD	28.80	105.42	76.62

FEF 25/ 75 Response (% of baseline)

Group	Pre- training	Post- training	Difference
1	78.77	700.00	621.23
1	130.06	106.36	-23.7
1	102.92	364.60	261.68
1	37.56	98.85	61.29
1	93.75	176.32	82.57
1	89.85	79.90	-9.95
1	129.45	99.71	-29.74
1	109.51	107.90	-1.61
Mean	96.48	216.70	120.22
SD	29.91	216.26	186.35

Paired Samples Test: Response Spirometry, Experimental Group & Training

	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1 %FEV1 - %FEV_1	-65.3220	116.32461	41.12696	162.5718	31.9278	-1.588	7	.156
Pair 2 %FVC - %FVC	-43.8124	88.92228	31.43877	118.1533	30.5285	-1.394	7	.206
Pair 3 %FEV1FVC - %FEVFV	-11.4907	16.68767	5.89998	-25.4419	2.4606	-1.948	7	.092
Pair 4 %PEF - %PEF	-49.2167	115.74756	40.92294	145.9841	47.5507	-1.203	7	.268
Pair 5 %FEF2575 - %FEF25/	120.2204	223.89586	79.15914	307.4020	66.9613	-1.519	7	.173

#2

VITA

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Candidate for the Degree of

Doctor of Philosophy

Thesis: RESISTIVE RESPIRATORY TRAINING AND ITS EFFECTS ON PULMONARY MECHANICS IN INDIVIDUALS DIAGNOSED WITH EXERCISE INDUCED ASTHMA

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